Right heart failure

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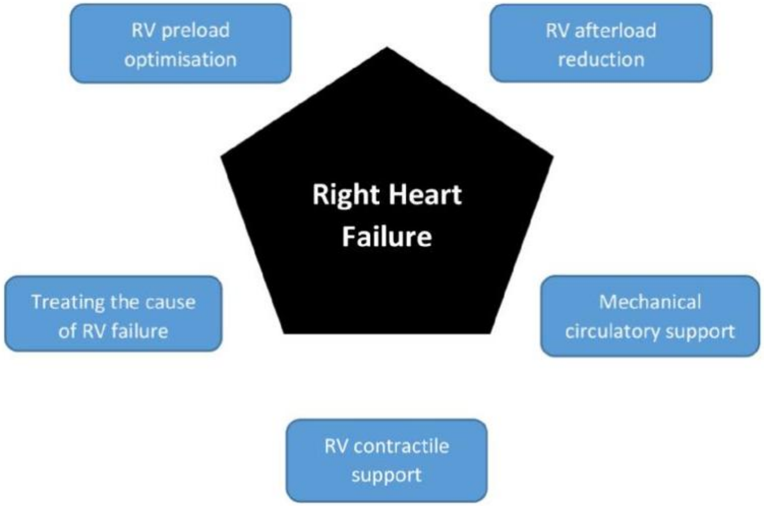
I. INTERNÍ KLINIKA
KARDIOLOGIE
FAKULTNÍ NEMOCNICE OLOMOUC

INTRODUCTION

RHF syndrome is **characterised** by the inability of the RV to generate enough stroke volume, thereby resulting in systemic venous congestion, underfilling of the left ventricle and, in the most advanced cases, CS shock. RHF portends a **poor prognosis** in almost every clinical scenario.

Although the **aetiologies** of RVF are diverse, treatment often involves simultaneous and timely execution of **multiple strategies** aimed at optimising RV preload, afterload, and contractility. Amelioration of the primary driver of RVF when feasible are desirable.

Timely institution of **MCS** can offer a bridge to RV recovery or to definitive management of the underlying cause.



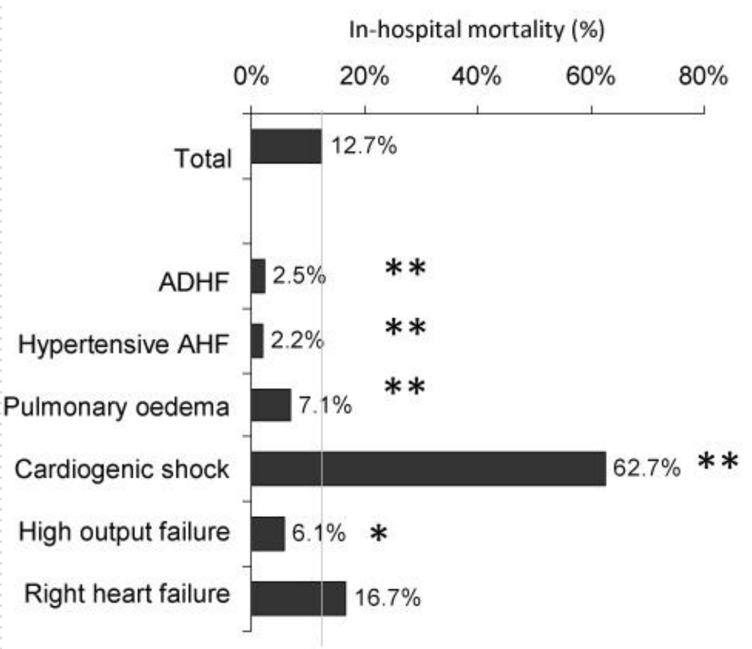
Congestive Heart Disease

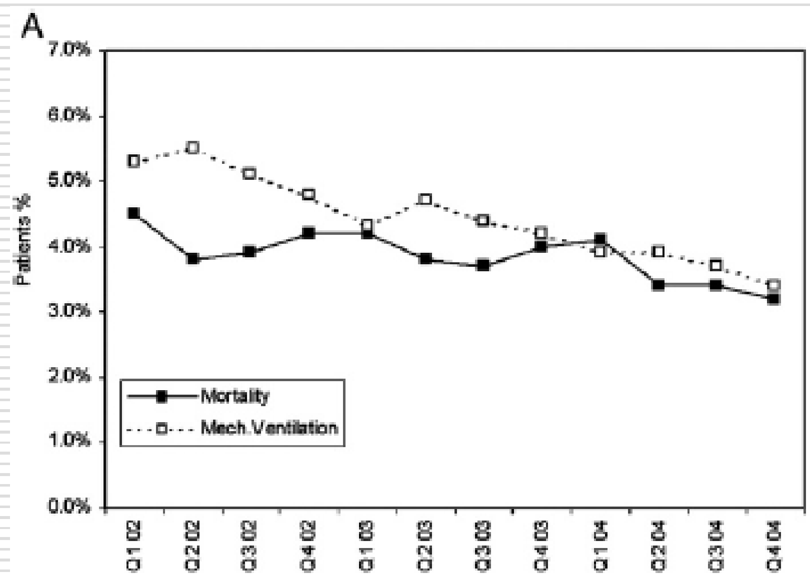
Temporal trends in clinical characteristics, treatments, and outcomes for heart failure hospitalizations, 2002 to 2004: findings from Acute Decompensated Heart Failure National Registry (ADHERE)

Gregg C. Fonarow, MD,[a] J. Thomas Heywood, MD,[b] Paul A. Heidenreich, MD, MS,[c] Margarita Lopatin, MS,[d] and Clyde W. Yancy, MD,[e] for the ADHERE Scientific Advisory Committee and Investigators *Los Angeles, La Jolla, Palo Alto, and Fremont, CA; and Dallas, TX*

EPIDEMIOLOGY

Right heart failure as the primary presentation of acute decompensated HF and cause of hospitalisation accounted for 2.2% of HF admissions in the CHARITEM registry; however, it was present as secondary to acute LV failure in more than one fifth of the cases.

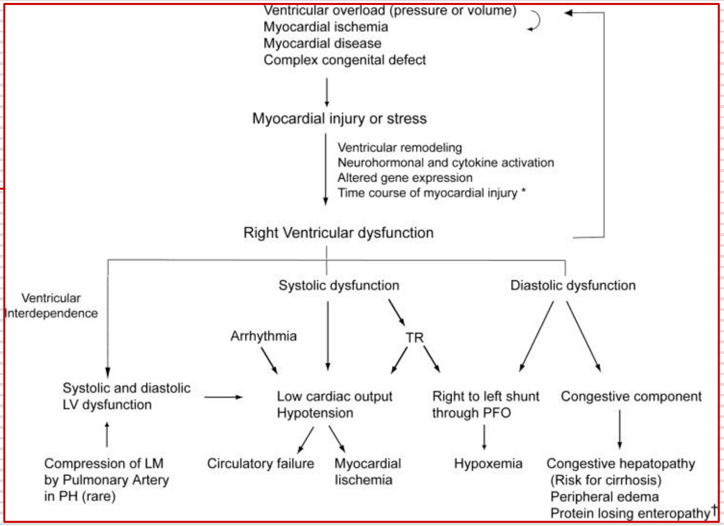


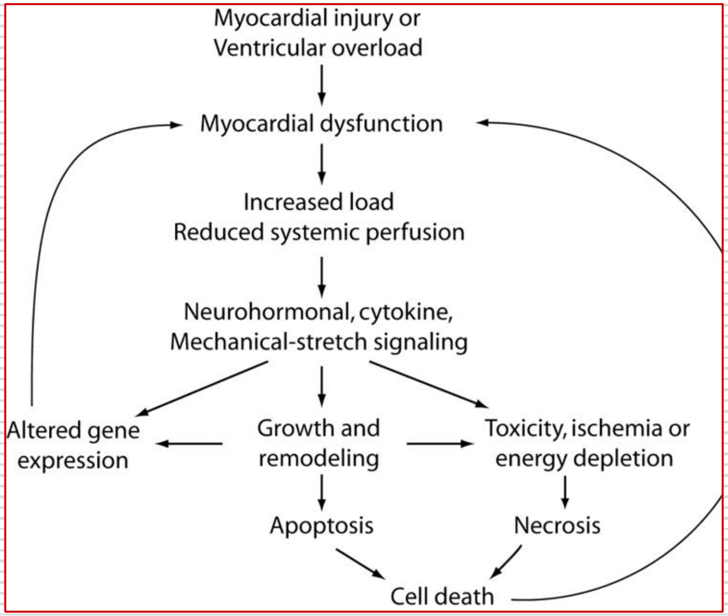


Spinar *et al. Critical Care* 2011 **15**:R291 doi:10.1186/cc10584

PATHOPHYSIOLOGY

Failure to adapt **acutely** results in rapid RV dilatation and dysfunction which is clinically manifest as hypotension and cardiogenic shock. On the other hand, when pulmonary arterial pressure (PAP) rises **more gradually**, the RV dilates using Starling's law to preserve flow output.
Usually, **RV function is maintained** until late stages of the disease.
Eventually, the RV fails, becomes more spherical, tricuspid regurgitation ensues causing more right heart failure and a spiral process develops ending in venous system congestion.





Right Ventricular Function in Cardiovascular Disease, Part II, Volume: 117, Issue: 13, Pages: 1717-1731, DOI: (10.1161/CIRCULATIONAHA.107.653584)

I. INTERNÍ KLINIKA
KARDIOLOGIE
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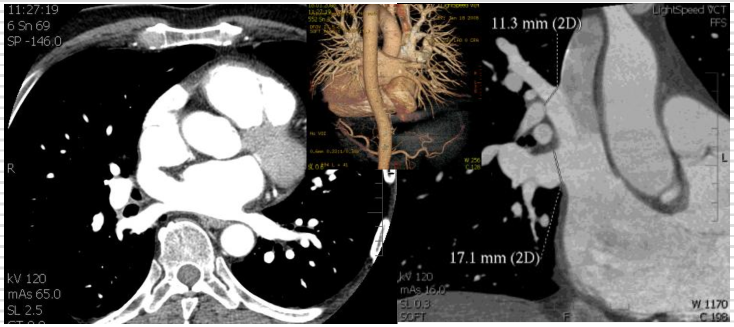
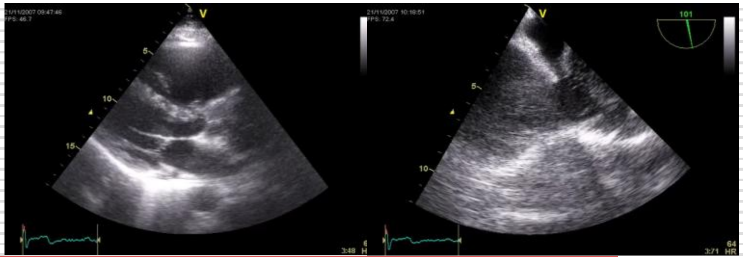
Many a time the right ventricle (RV) is regarded as the **"younger brother"** of the left ventricle (LV) and is treated as a less important member of the contractile apparatus.
This view stemmed from the concept that the RV functions rather as a *passive conduit* and its importance is not great as *it pumps blood to only one organ*, the lungs.

However, the circulatory system is a closed one and both ventricles are interdependent, working together in an orchestrated complex pattern in health and disease.
The failure of one ventricle deleteriously affects the performance of the other.

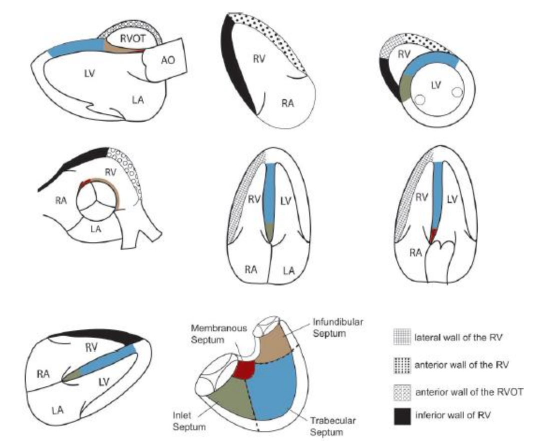


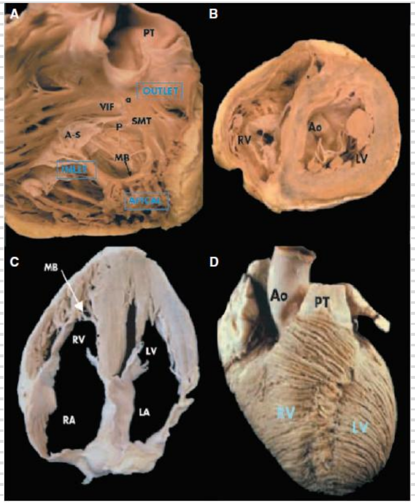


Sanz J, Sánchez-Quintana D, Bossone E, Bogaard HJ, Naeije R. Anatomy, Function, and Dysfunction of the Right Ventricle: JACC State-of-the-Art Review. J Am Coll Cardiol 2019;73:1463-1482.

I. INTERNÍ KLINIKA
KARDIOLOGIE
FAKULTNÍ NEMOCNICE OLOMOUC

- ✓ RV has historically received **less attention** than its counterpart of the left side of the heart, yet there is a substantial body of evidence showing that RV size and function are perhaps equally important in predicting adverse outcomes in CV disease.
- ✓ RV dysfunction is associated with **excess morbidity and mortality** in patients with chronic left-sided HF, AMI (with or without RV involvement), PE, PAH, CHD.
- ✓ RV has a **unique crescent shape**, which adds complexity to the quantification of its size and function. This chamber plays an important role in the morbidity/mortality of patients presenting with signs and symptoms of cardiopulmonary disease.
- ✓ Advances in **noninvasive imaging of the RV** have yielded insights into the pathophysiology of this complex and once elusive chamber.

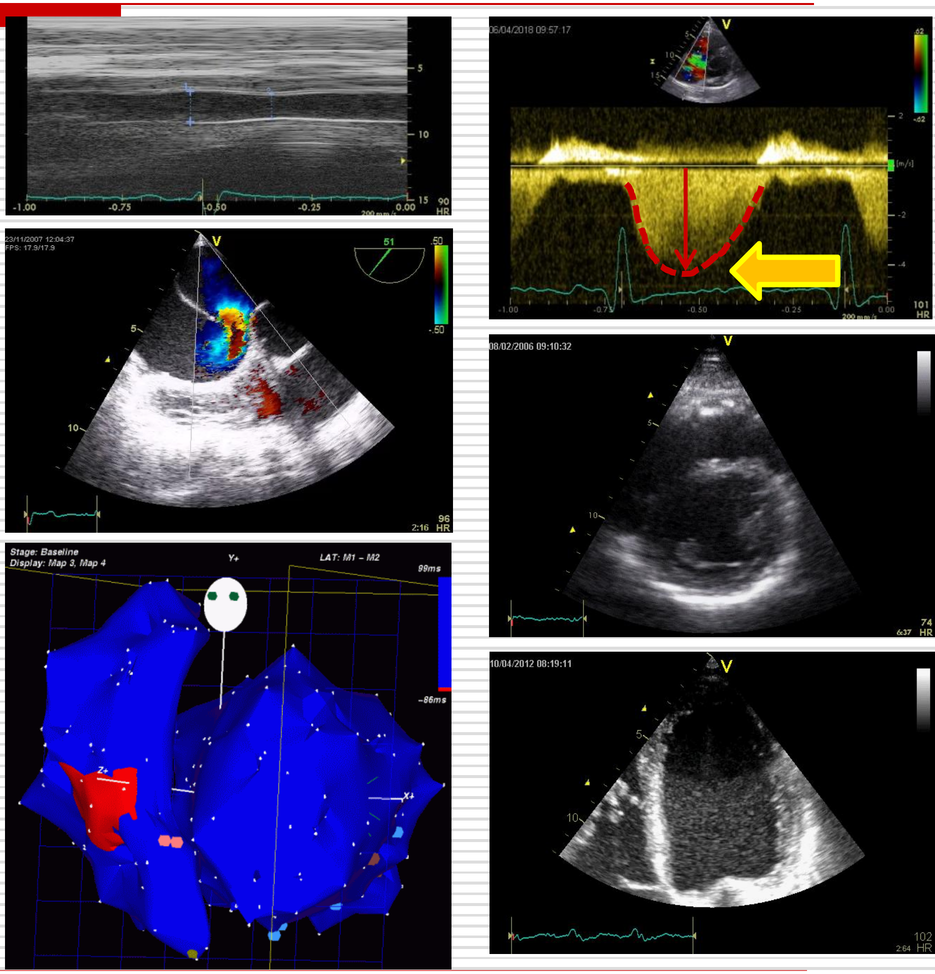




Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1.
Haddad F, Hunt SA, Rosenthal DN., et al. Right Ventricular Function in Cardiovascular. Disease, Part I. Anatomy, Physiology, Aging, and Functional Assessment of the Right Ventricle. *Circulation*. 2008;117:1436-1448.

RV ANATOMY AND PHYSIOLOGY

1. The right atrium transmits and pumps blood across the TV into the right ventricle (RV), which then ejects the stroke volume through the pulmonic valve and into the main pulmonary artery.
2. In the absence of shunt, forward stroke volume of the right heart is obligately equal to that of the left.
3. Anatomic and physiologic features, coupled with the less accessible retrosternal position of the RV, have resulted in many challenges in the noninvasive evaluation of RV size and function by echocardiography.



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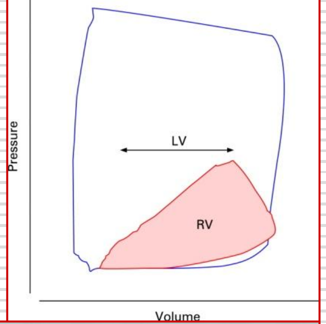
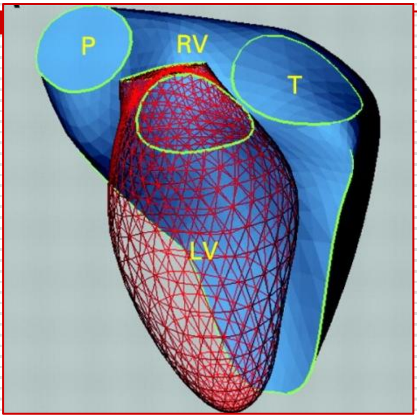
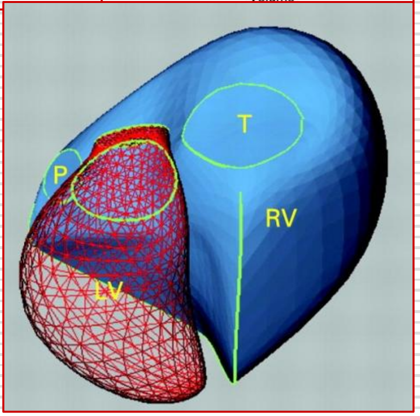
The RV differs from the LV in terms of anatomy and physiology



1. The RV loosely resembles a pyramid and is composed of **three portions**: the inlet, the body, and the outflow tract. Contraction is generated by a deep layer of longitudinal fibers that result in longitudinal (base to apex) **shortening**, and a superficial layer of circumferential fibers that result in inward **thickening**.
2. The RV **lacks a third layer of spiral fibers** as seen in the left ventricle.
3. The **RV end-diastolic volume** is slightly larger than that of the left ventricle, and as a result has a slightly lower ejection fraction.
4. RV ejection is accomplished with a mass that is approximately **one-fifth that of the left ventricle**. Accordingly, the RV is well **suited as a volume pump**, but is **prone to failure** when faced with an acute pressure challenge.





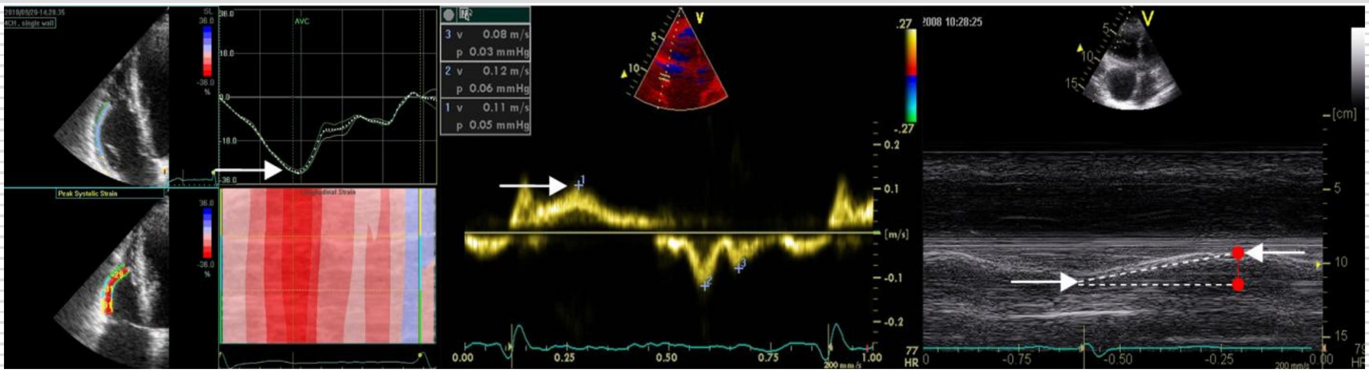
Characteristics	RV	LV
Structure	Inflow region, trabeculated myocardium, infundibulum	Inflow region and myocardium, no infundibulum
Shape	From the side: triangular[9] cross section: crescentic	Elliptic[9]
End-diastolic volume, mL/m^2	75±13 (49–101)[8]	66±12 (44–89)[8]
Mass, g/m^2	26±5 (17–34)[8]	87±12 (64–109)[8]
Thickness of ventricular wall, mm	2 to 5[5,6]	7 to 11[6]
Ventricular pressures, mm Hg	25/4 [(15–30)/(1–7)][11]	130/8 [(90–140)/(5–12)][11]
RVEF, %	61±7 (47–76)[8] >40–45*	67±5 (57–78)[8] >50*
Ventricular elastance (Emax), mm Hg/mL	1.30±0.84[20]	5.48±1.23[18]
Compliance at end diastole, $mm\ Hg^{-1}$	Higher compliance than LV[26]†	$5.0 \pm 0.52 \times 10^{-2}$[27]
Filling profiles	Starts earlier and finishes later ↑ lower filling velocities[6]	Starts later and finishes[6] earlier higher filling velocities
PVR vs SVR, $dyne \cdot s \cdot cm^{-5}$	70 (20–130)[11]	1100 (700–1600)[11]
Stroke work index, g/m^2 per beat	8±2 (1/6 of LV stroke work)[9]	50±20[11]
Exercise reserve	↑ RVEF ≥5%[9]	↑ LVEF ≥5%[37]
Resistance to ischemia	Greater resistance to ischemia[9]	More susceptible to ischemia[9]
Adaptation to disease state	Better adaptation to volume overload states[9]	Better adaptation to pressure overload states[9]

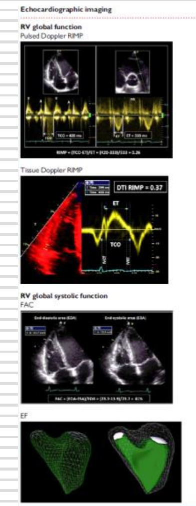
Florence Sheehan, and Andrew Redington Heart 2008;94:1510-1515
Haddad F, Hunt SA, Rosenthal DN., et al. Right Ventricular Function in Cardiovascular. Disease, Part I. Anatomy, Physiology, Aging, and Functional Assessment of the Right Ventricle. *Circulation*. 2008;117:1436-1448.

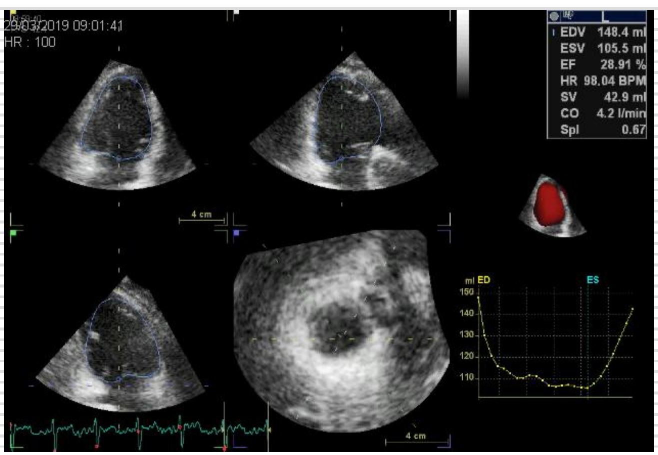
European Heart Journal – Cardiovascular Imaging (2015) 16, 233–271
doi:10.1093/ehjci/jev014
POSITION PAPER

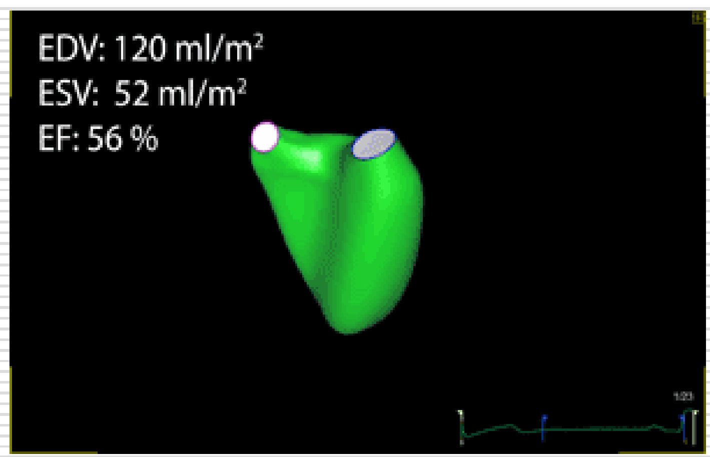
Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging

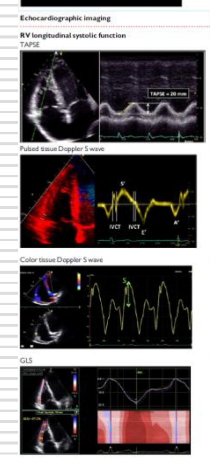
RV MORPHOLOGY/FUNCTION











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I. INTERNÍ KLINIKA KARDIOLOGIE
FAKULTNÍ NEMOCNICE OLOMOUC

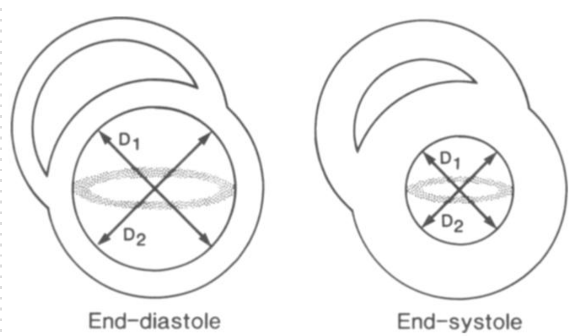
918
JACC Vol. 5, No. 4
April 1985:918–24

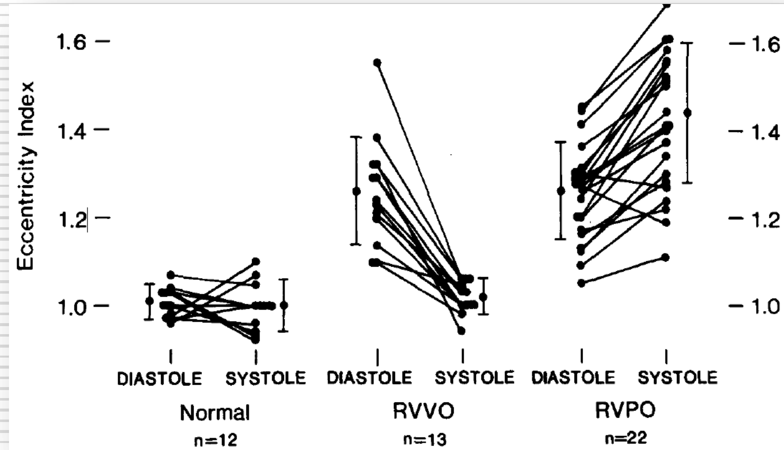
An Echocardiographic Index for Separation of Right Ventricular Volume and Pressure Overload

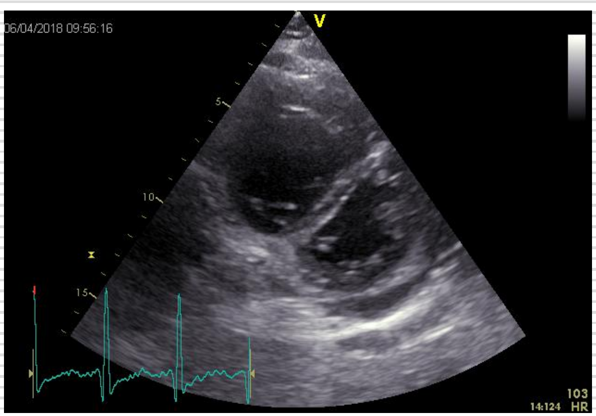
THOMAS RYAN, MD, OLIVERA PETROVIC, MD, JAMES C. DILLON, MD, FACC,
HARVEY FEIGENBAUM, MD, FACC, MARY JO CONLEY, WILLIAM F. ARMSTRONG, MD, FACC

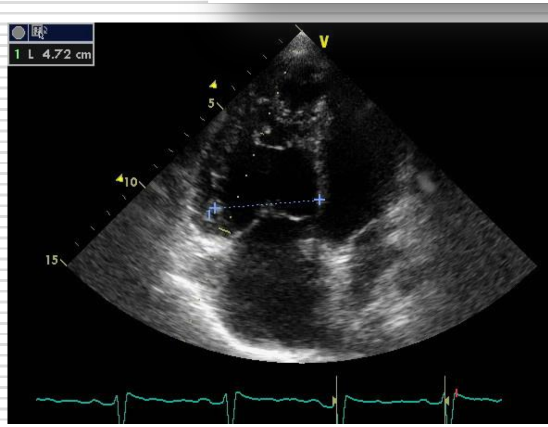
D-shaped LV cavity in systole suggests RV pressure overload, in diastole suggests RV volume overload.

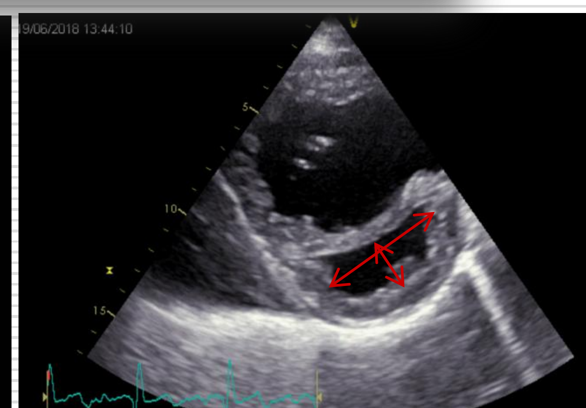
LV Eccentricity Index = D2/D1











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I. INTERNÍ KLINIKA
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STATE-OF-THE-ART PAPER

Medical and Surgical Treatment of Acute Right Ventricular Failure

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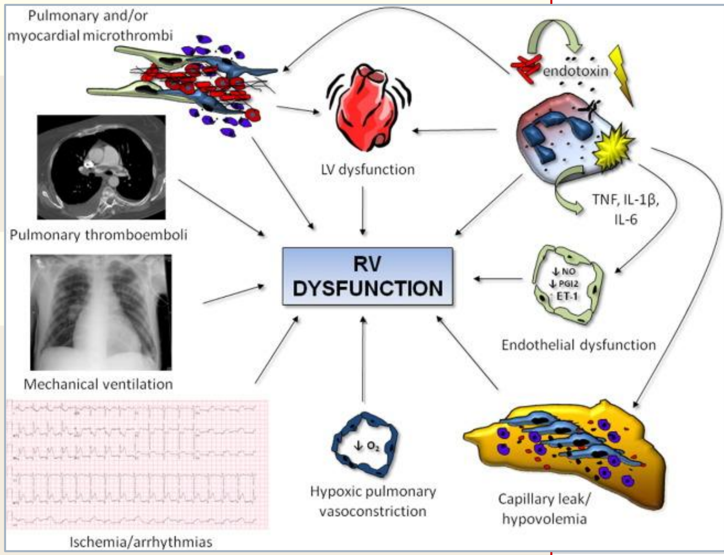
Indianapolis, Indiana

ETIOLOGY

Left ventricular dysfunction	Most common cause of right heart failure RV co-involvement in structural or ischemic heart disease or indirect RV dysfunction due to ventricular interdependence, pulmonary venous congestion, and/or arrhythmias
RV ischemia (via negative effects on inotropy and/or relaxation or via arrhythmias)	RV infarction Relative RV ischemia secondary to RV pressure or volume overload
Afterload increase (endothelial dysfunction, vasoconstriction, and/or mechanical obstruction)	Pulmonary arterial hypertension and secondary forms of PH Hypoxic pulmonary vasoconstriction Post-cardiothoracic surgery (CABG, corrective surgery for CHD, heart/lung transplantation, pneumonectomy) Pulmonary embolus Pulmonary microthrombi (sepsis and acute lung injury) Pulmonary stenosis/RV outflow tract obstruction Acute chest syndrome in sickle cell disease Mechanical ventilation
Pre-load decrease (via effects on RV fiber length and contractility)	Hypovolemia/capillary leak Superior vena cava syndrome Tricuspid stenosis Cardiac tamponade (inhibition of diastolic filling) Mechanical ventilation
Intrinsic myocardial disease	Cardiomyopathies Arrhythmogenic RV dysplasia Sepsis (cytokine-induced myocardial depression)
Congenital and valvular heart disease	Ebstein's anomaly Tetralogy of Fallot Transposition of the great arteries Atrial septum defect Anomalous pulmonary venous return Tricuspid regurgitation Pulmonary regurgitation Mitral valve disease
Pericardial disease (via negative effects on diastolic filling)	Constrictive pericarditis



I. INTERNÍ KLINIKA
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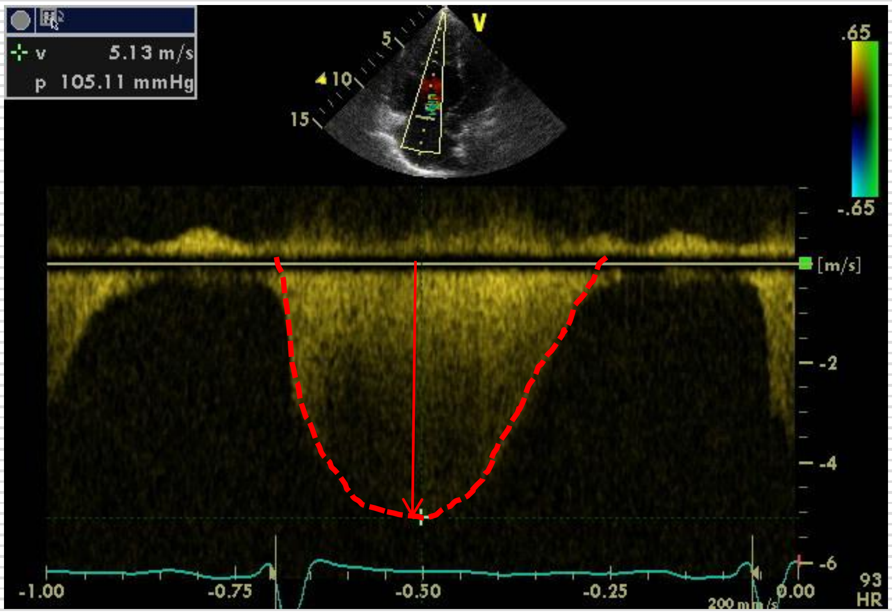
European Heart Journal
doi:10.1093/eurheartj/ehv317

ESC/ERS GUIDELINES

2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension

The Joint Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS)

PAH/CTEPH



Peak tricuspid regurgitation velocity (m/s)	Presence of other echo 'PH signs'[a]	Echocardiographic probability of pulmonary hypertension
≤2.8 or not measurable	No	Low
≤2.8 or not measurable	Yes	Intermediate
2.9–3.4	No	
2.9–3.4	Yes	High
>3.4	Not required	

A: The ventricles[a]	B: Pulmonary artery[a]	C: Inferior vena cava and right atrium[a]
Right ventricle/ left ventricle basal diameter ratio >1.0	Right ventricular outflow Doppler acceleration time <105 msec and/or midsystolic notching	Inferior cava diameter >21 mm with decreased inspiratory collapse (<50 % with a sniff or <20 % with quiet inspiration)
Flattening of the interventricular septum (left ventricular eccentricity index >1.1 in systole and/or diastole)	Early diastolic pulmonary regurgitation velocity >2.2 m/sec	Right atrial area (end-systole) >18 cm^2
	PA diameter >25 mm.	

I. INTERNÍ KLINIKA
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European Heart Journal (2014) 35, 3033–3080
doi:10.1093/eurheartj/ehu283

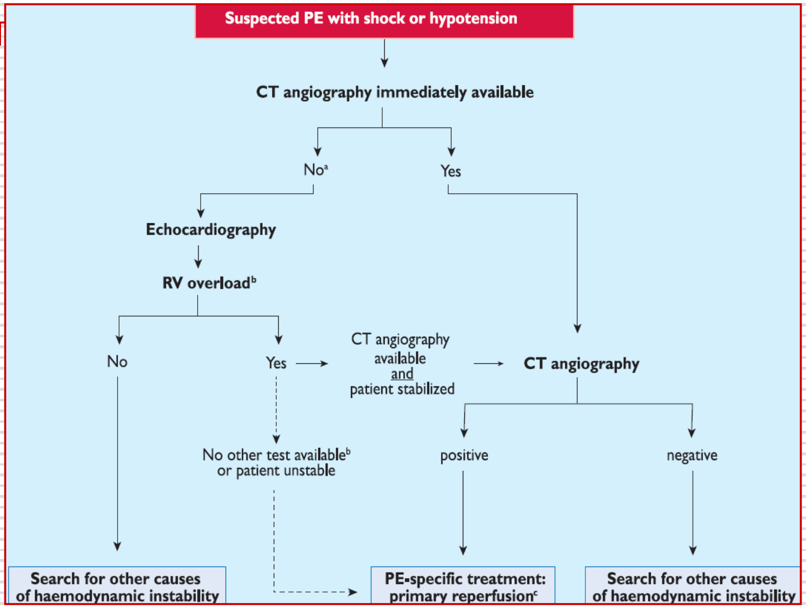
ESC GUIDELINES

2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism

The Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC)

Pulmonary embolism

Echocardiographic criteria of RV dysfunction include RV dilation and/or an increased end-diastolic RV–LV diameter ratio (in most studies, the reported threshold value was 0.9 or 1.0); hypokinesia of the free RV wall; **increased velocity of the tricuspid regurgitation jet**; or combinations of the above.



Early mortality risk		Risk parameters and scores			
		Shock or hypotension	PESI class III-V or sPESI ≥I[a]	Signs of RV dysfunction on an imaging test[b]	Cardiac laboratory biomarkers[c]
High		+	(+)[d]	+	(+)[d]
Intermediate	Intermediate–high	–	+	Both positive	
	Intermediate–low	–	+	Either one (or none) positive[e]	
Low		–	–	Assessment optional; if assessed, both negative[e]	

I. INTERNÍ KLINIKA
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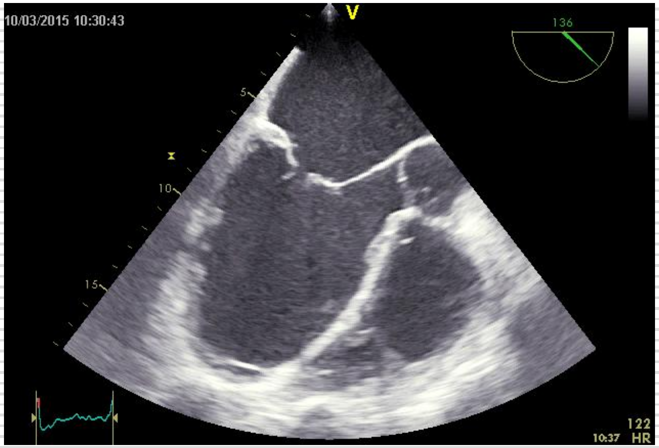
European Heart Journal (2016) 37, 2129–2200
doi:10.1093/eurheartj/ehw128

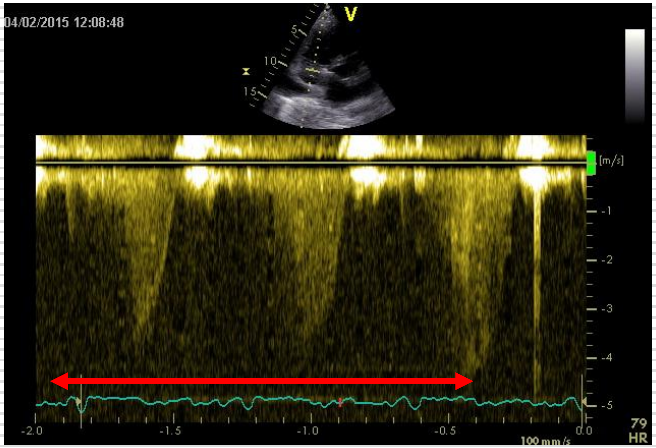
ESC GUIDELINES

2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)

Heart failure





Patients to consider	End-stage HF with severe symptoms, a poor prognosis, and no remaining alternative treatment options. Motivated, well informed, and emotionally stable. Capable of complying with the intensive treatment required postoperatively.
Contra-indications	Active infection. Severe peripheral arterial or cerebrovascular disease. Pharmacologically irreversible pulmonary hypertension (LVAD should be considered with a subsequent re-evaluation to establish candidacy).

Patients with >2 months of severe symptoms despite optimal medical and device therapy and more than one of the following:
LVEF <25% and, if measured, peak VO_2 <12 mL/kg/min.
≥3 HF hospitalizations in previous 12 months without an obvious precipitating cause.
Dependence on i.v. inotropic therapy.
Progressive end-organ dysfunction (worsening renal and/or hepatic function) due to reduced perfusion and not to inadequate ventricular filling pressure (PCWP ≥20 mmHg and SBP ≤80–90 mmHg or CI ≤2 L/min/m^2).
Absence of severe right ventricular dysfunction together with severe tricuspid regurgitation.

Study	Population	NYHA Class (%)	n	RV Dysfunction Criteria	Main Findings (Significant Findings)
Polak et al,[42] 1983	CAD	II–IV	34	RVEF <35%	23% survival (RVD) vs 71% survival at 2 y
Di Salvo et al,[39] 1995	CAD, IDC	III–IV	67	RVEF <35%	RVD and % Vo_2-independent predictors of survival at 2 y
De Groote et al,[38] 1998	CAD, IDC	II–III	205	RVEF <35%	RVD, maximal Vo_2, NYHA-independent predictors of survival at 2 y
Ghio et al,[41] 2001	CAD, IDC	III–IV (70)	377	RVEF <35%	Incremental value of PAP and RV function in predicting event-free survival
Sun et al,[44] 1997	IDC	III–IV (74)	100	RV area/LV area >0.5	RV enlargement independent predictor of survival
Meluzin et al,[43] 2005	CAD, IDC	II–IV	140	RVMPI >1.20, IVA <2.52 cm/s, TAV <10.8 cm/s	RVMPI and TDI indexes were predictive of mortality or event-free survival

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ESC European Society of Cardiology
European Heart Journal (2017) 38, 2739–2791
doi:10.1093/eurheartj/ehx391
ESC/EACTS GUIDELINES

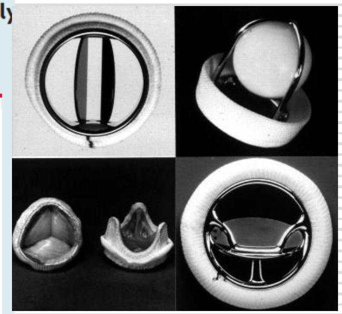
Valvular heart disease

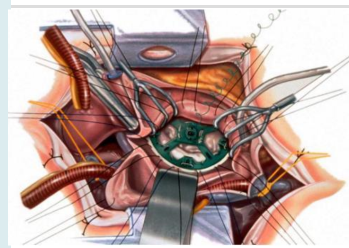
2017 ESC/EACTS Guidelines for the management of valvular heart disease

The Task Force for the Management of Valvular Heart Disease of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS)

Indications for PMC and mitral valve surgery in clinically significant (moderate or severe) mitral stenosis (valve area ≤1.5 cm²)

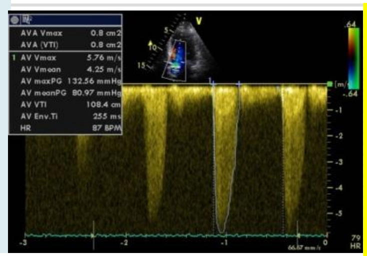
Recommendations	Class[a]	Level[b]
PMC is indicated in symptomatic patients without unfavourable characteristics[c] for PMC.[144,146,148]	I	B
PMC is indicated in any symptomatic patients with a contraindication or a high risk for surgery.	I	C
Mitral valve surgery is indicated in symptomatic patients who are not suitable for PMC.	I	C
PMC should be considered as initial treatment in symptomatic patients with suboptimal anatomy but no unfavourable clinical characteristics for PMC.[c]	IIa	C
PMC should be considered in asymptomatic patients without unfavourable clinical and anatomical characteristics[c] for PMC and: • high thromboembolic risk (history of systemic embolism, dense spontaneous contrast in the LA, new-onset or paroxysmal atrial fibrillation), and/or • high risk of haemodynamic decompensation (systolic pulmonary pressure >50 mmHg at rest, need for major non-cardiac surgery, desire for pregnancy).	IIa	C





Indications for intervention in severe primary mitral regurgitation

Recommendations	Class[a]	Level[b]
Mitral valve repair should be the preferred technique when the results are expected to be durable.	I	C
Surgery is indicated in symptomatic patients with LVEF >30%.[121,131,132]	I	B
Surgery is indicated in asymptomatic patients with LV dysfunction (LVESD ≥45 mm[c] and/or LVEF ≤60%).[122,131]	I	B
Surgery should be considered in asymptomatic patients with preserved LV function (LVESD <45 mm and LVEF >60%) and atrial fibrillation secondary to mitral regurgitation or pulmonary hypertension[d] (systolic pulmonary pressure at rest >50 mmHg).[123,124]	IIa	B



Indications for surgery in asymptomatic aortic stenosis

New IIa C recommendation:
Severe pulmonary hypertension (systolic pulmonary artery pressure at rest >60 mmHg confirmed by invasive measurement) without other explanation.

Indications for intervention in asymptomatic severe primary mitral regurgitation

New additional statement:
If pulmonary hypertension (SPAP >50 mmHg at rest) is the only indication for surgery, the value should be confirmed by invasive measurement.

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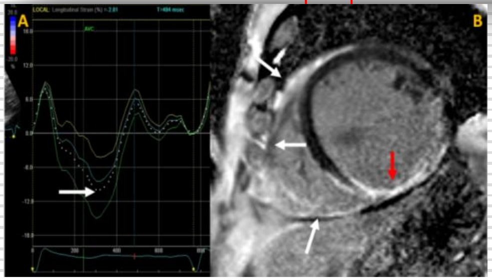
Impact of Right Ventricular Involvement on Mortality and Morbidity in Patients With Inferior Myocardial Infarction

Shamir R. Mehta, MD, FACC,*† John W. Eikelboom, MBBS, FRACP,†

RV myocardial infarction

- Right ventricular (RV) ischemia complicates up to 50% of inferior myocardial infarctions (MI).
- RV myocardial involvement extent and RV function provide strong prognostic information in patients treated with primary percutaneous coronary intervention for AMI.
- The early recognition of RVMI in a patient with acute MI is of prime importance, not only for prognostication purposes, but also because it can guide specific therapy

	Anterior MI (%) (n = 971)	Inferior MI (%) RVMI (n = 491)	Inferior MI (%) No RVMI (n = 638)	Odds Ratio (95% CI) RVMI vs. No RVMI	Odds Ratio (95% CI) RVMI vs. Anterior MI	Odds Ratio (95% CI) No RVMI vs. Anterior MI
Mortality						
In-hospital	9.7	7.1	5.5	1.3 (0.8–2.1)	0.7 (0.5–1.1)	0.5* (0.4–0.8)
At 35 days	10.6	7.5	5.6	1.4 (0.8–2.2)	0.7 (0.5–1.0)	0.5† (0.3–0.8)
At 6 months	13.2	8.9	6.9	1.3 (0.9–2.0)	0.6* (0.4–0.9)	0.5‡ (0.3–0.7)
Pump failure or mechanical complications						
Left heart failure	17.7	11.7	11.5	1.0 (0.7–1.5)	0.6* (0.4–0.8)	0.6† (0.4–0.8)
Cardiogenic shock	7.8	6.9	5.5	1.3 (0.8–2.1)	0.9 (0.6–1.3)	0.7 (0.4–1.0)
Cardiac rupture	1.5	0.8	0.3	2.6 (0.5–14.4)	0.5 (0.2–1.6)	0.2* (0.04–0.9)
Hypotension§	16.1	29.0	19.3	1.7‡ (1.3–2.3)	2.1‡ (1.6–2.8)	1.2 (1.0–1.6)
Electrical complications						
Atrial fibrillation	8.3	12.5	2.2	1.6 (1.1–2.4)	1.6 (1.1–2.3)	1.0 (0.9–1.4)
Ventricular fibrillation	5.0	8.4	2.7	3.3‡ (1.9–6.0)	1.7 (1.1–2.7)	0.5‖ (0.3–0.9)
Sustained VT¶	4.4	6.8	2.7	2.6† (1.4–4.8)	1.6 (1.0–2.5)	0.6 (0.3–1.0)
2° or 3° AV block	3.1	21.0	9.1	2.7‡ (1.9–3.7)	8.4 (5.5–12.8)	3.2‡ (2.0–5.0)



Kakouros N , Cokkinos DV. Right ventricular myocardial infarction: pathophysiology, diagnosis, and management. Postgrad Med J doi:10.1136/pgmj.2010.103887
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Lloyd EA, Gersh BJ, Kennelly BM. Hemodynamic spectrum of "dominant" right ventricular infarction in 19 patients. Am J Cardiol 1981;48:1016–22.
Goldstein JA. Right heart ischemia: pathophysiology, natural history, and clinical management. Prog Cardiovasc Dis 1998;40:325–41.
Andersen HR. Right ventricular infarction: frequency, size and topography in coronary heart disease: a prospective study comprising 107 consecutive autopsies from a coronary care unit. JACC 1987;10:1223–32.

Doporučení pro… | Guidelines

Souhrn Doporučených postupů ESC pro diagnostiku a léčbu akutního a chronického srdečního selhání z roku 2016.

Připraven Českou kardiologickou společností

(Summary of the 2016 ESC Guidelines on the diagnosis and treatment of acute and chronic heart failure. Prepared by the Czech Society of Cardiology)

EUROPEAN SOCIETY OF CARDIOLOGY®

ČESKÁ KARDIOLOGICKÁ SPOLEČNOST
THE CZECH SOCIETY OF CARDIOLOGY

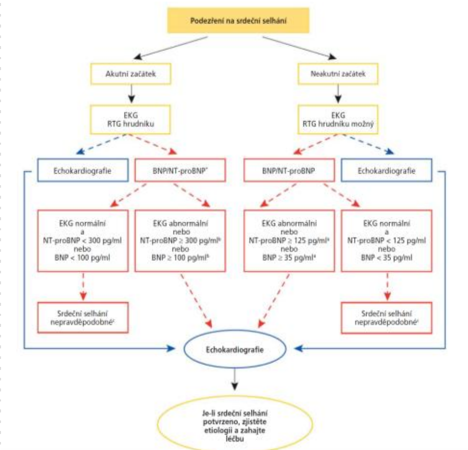
European Heart Journal (2016) 37, 2129–2200
doi:10.1093/eurheartj/ehw128

ESC GUIDELINES

2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)

DIAGNOSIS



Symptoms	Signs
Typical	**More specific**
Breathlessness Orthopnoea Paroxysmal nocturnal dyspnoea Reduced exercise tolerance Fatigue, tiredness, increased time to recover after exercise Ankle swelling	Elevated jugular venous pressure Hepatojugular reflux Third heart sound (gallop rhythm) Laterally displaced apical impulse
Less typical	**Less specific**
Nocturnal cough Wheezing Bloated feeling Loss of appetite Confusion (especially in the elderly) Depression Palpitations Dizziness Syncope Bendopnea[53]	Weight gain (>2 kg/week) Weight loss (in advanced HF) Tissue wasting (cachexia) Cardiac murmur Peripheral oedema (ankle, sacral, scrotal) Pulmonary crepitations Reduced air entry and dullness to percussion at lung bases (pleural effusion) Tachycardia Irregular pulse Tachypnoea Cheyne Stokes respiration Hepatomegaly Ascites Cold extremities Oliguria Narrow pulse pressure

I. INTERNÍ KLINIKA
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STATE-OF-THE-ART PAPER

Medical and Surgical Treatment of Acute Right Ventricular Failure

Tim Lahm, MD,‡§ Charles A. McCaslin, MD,‡|| Thomas C. Wozniak, MD,* Waqas Ghumman, MD,‡ Yazid Y. Fadl, MD, MPH,¶ Omar S. Obeidat, MD,¶ Katie Schwab, PA,* Daniel R. Meldrum, MD*†#**
Indianapolis, Indiana

Diagnosis of RVF in the ICU

BNP, NT-proBNP, troponin	Increase in LV dysfunction, renal failure, sepsis, but significant RV dysfunction less likely if values normal BNP predicts survival in acute RVF in PAH; increased levels (1,415 pg/ml vs. 628 pg/ml) associated with increased mortality (14) BNP >168 pg/ml identifies RV dysfunction in CTEPH patients with 88% sensitivity, 86% specificity (15) Risk stratification in patients with subtle RV dysfunction during acute, nonmassive PE (16,17)
Sodium	≤136 mmol/l predicts RVF and increased risk of death in PAH patients (18) Predicts survival in PAH patients with acute RVF; decreased levels associated with increased mortality (14)
Creatinine	Predicts survival in PAH patients with acute RVF; increased levels (1.5 mg/dl vs. 1.25 mg/dl) suggest increased mortality (14)
C-reactive protein	Predicts survival in PAH patients with acute RVF; increased levels (4 mg/dl vs. 1.2 mg/dl) associated with increased mortality (14)
Transaminases	Increase reflects hepatic congestion and/or hypoperfusion due to compromised LV function and forward failure Prognostic value not established
Growth differentiation factor-15	Stress responsive, transforming growth factor-beta–related myocardial cytokine Independent predictor of long-term mortality in acute PE; increased value of established prognostic markers (19) Risk stratification in PAH patients; increased levels associated with increase in markers of RV dysfunction (20)
Right atrial pressure, cardiac index	Strongest hemodynamic prognosticators in PAH (22); more accurate reflection of RV function than PAP Right atrial pressure ≥15 mm Hg, cardiac index ≤2 $l/min/m^2$ indication for transplantation referral in PAH (22)
PVR	Differentiates whether increased afterload is due to PAH, secondary PH, or hyperdynamic states (23) PVR >1,000–1,200 $dynes·s·cm^{-5}$: contraindication for atrial septal defect closure (24), balloon atrial septostomy in severe PAH (22), pulmonary endarterectomy in CTEPH (22)
Right ventricular stroke work index	Prognosticates RVF after LVAD placement and transplantation-free survival in dilated cardiomyopathy (25,26) Easily obtained via PAC; may allow for further prognostication in acute RVF, but further studies needed
Pulmonary artery impedance	Evaluates and integrates PVR and pulmonary artery elastance, flow, pulsatile pressure, and wave reflection (27) Superior and more complete method of RV afterload assessment than PVR alone (27)
RVEF, RA and RV volume, tricuspid regurgitation, ventricular septal shift, pericardial effusion	Established and readily available markers of RV dysfunction (3) Limited by marked pre-load dependence (3)
Right ventricular systolic pressure	Calculated from tricuspid regurgitant jet and RAP; cannot be obtained if no regurgitant jet identified Off by >10 mm Hg in almost 50% of measurements in PAH patients (32)
TAPSE, tissue Doppler, Tei index	More specific and less pre-load–dependent than traditional echocardiographic markers (29–31) Established prognostic value of TAPSE in PAH patients; significantly decreased survival if TAPSE <1.8 cm (29)

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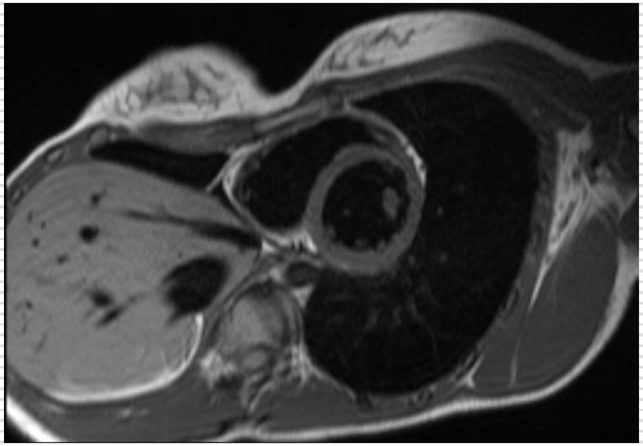
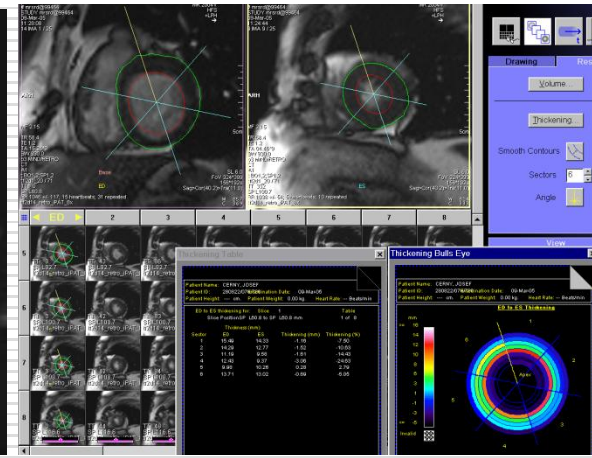
CMR imaging is recommended to evaluate cardiac structure and function, to measure LVEF, and to characterize cardiac tissue, especially in subjects with inadequate echocardiographic images or where the echocardiographic findings are inconclusive or incomplete (but taking account of cautions/contraindications to CMR).	I	C





First
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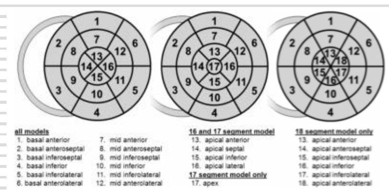
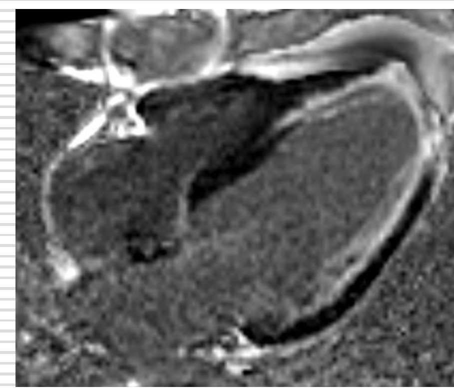


Delayed
enhancement



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Pericardial Disease

Constrictive Pericarditis in the Modern Era

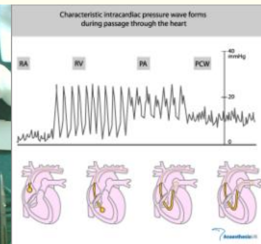
Novel Criteria for Diagnosis in the Cardiac Catheterization Laboratory

Deepak R. Talreja, MD, FACC, Rick A. Nishimura, MD, FACC, Jae K. Oh, MD, FACC, David R. Holmes, MD, FACC

Rochester, Minnesota

Table 2 **Catheterization Criterion**

Criterion	Sensitivity (%)	Specificity (%)	Positive Predictive Accuracy (%)	Negative Predictive Accuracy (%)
LVEDP − RVEDP ≤5 mm Hg	46	54	58	40
PASP <55 mm Hg	90	29	73	66
RVEDP/RVSP >1/3	93	46	71	79
LVRFW >7 mm Hg	45	44	62	42
Inspiratory decrease in RAP <5 mm Hg	71	37	62	39
Systolic area index >1.1	97	100	100	95



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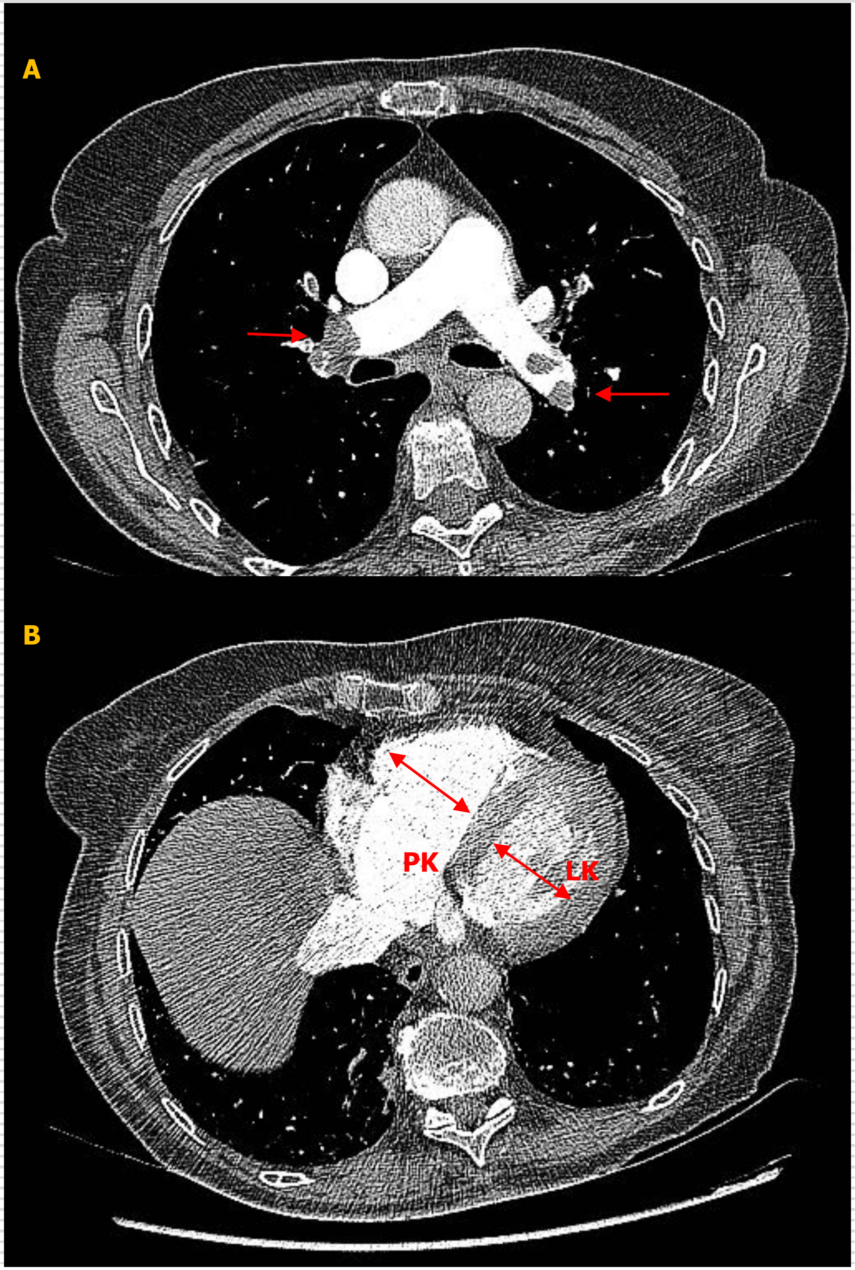

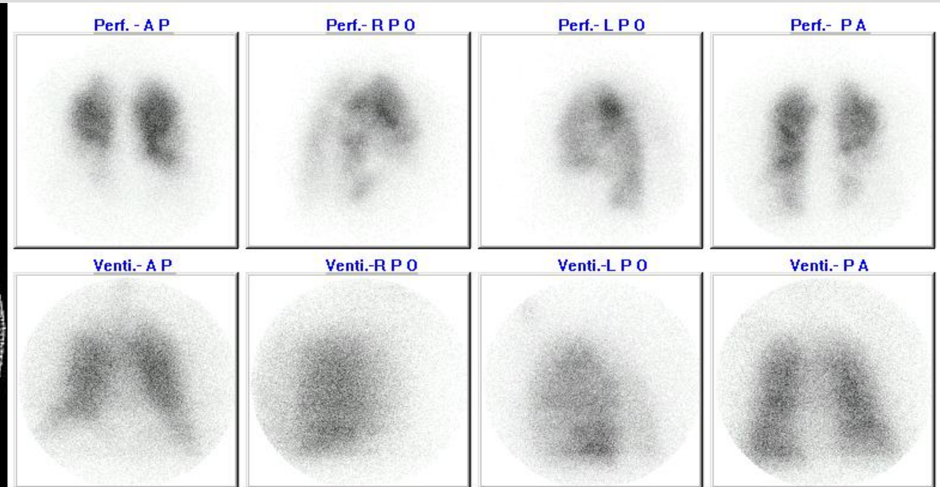

Test or biomarker	Cut-off value	Sensitivity, % (95% CI)	Specificity, % (95% CI)	NPV, % (95% CI)	PPV, % (95% CI)	OR or HR (95% CI)	No. patients	Study design (reference)	Remarks
Echocardiography	Various criteria of RV dysfunction	74 (61–84)	54 (51–56)	98 (96–99)	8 (6–10)	2.4 (1.3–4.3)	1249	Meta-analysis[226]	RV dysfunction on echocardiography or CT was one of the inclusion criteria in two randomized trials investigating thrombolysis in normotensive patients with PE.[252,253]
CT angiography	RV/LV ≥1.0	46 (27–66)	59 (54–64)	93 (89–96)	8 (5–14)	1.5 (0.7–3.4)	383	Meta-analysis[226]	
	RV/LV ≥0.9	84 (65–94)	35 (30–39)	97 (94–99)	7 (5–10)	2.8 (0.9–8.2)	457	Prospective cohort[228]	
BNP	75–100 pg/mL	85 (64–95)	56 (50–62)	98 (94–99)	14 (9–21)	6.5 (2.0–21)	261	Meta-analysis[232]	The optimal cut-off value for PE has not been defined.
NT-proBNP	600 pg/mL	86 (69–95)	50 (46–54)	99 (97–100)	7 (5–19)	6.3 (2.2–18.3)	688	Prospective cohort[234e]	NT-proBNP <500 pg/mL was one of the inclusion criteria in a single-armed management trial investigating home treatment of PE.[237]
Troponin I	Different assays/ cut-off values[c]	NR	NR	NR	NR	4.0 (2.2–7.2)	1303	Meta-analysis[239]	A positive cardiac troponin test was one of the inclusion criteria in a randomized trial investigating thrombolysis in normotensive patients with PE.[253]
Troponin T	Different assays/cut-off values[c]	NR	NR	NR	NR		682	Meta-analysis[239]	
	14 pg/mL[d]	87 (71–95)	42 (38–47)	98 (95–99)	9 (6–12)	5.0 (1.7–14.4)	526	Prospective cohort[76e]	
H-FABP	6 ng/mL	89 (52–99)	82 (74–89)	99 (94–99)	28 (13–47)	36.6 (4.3–304)	126	Prospective cohort[244e]	

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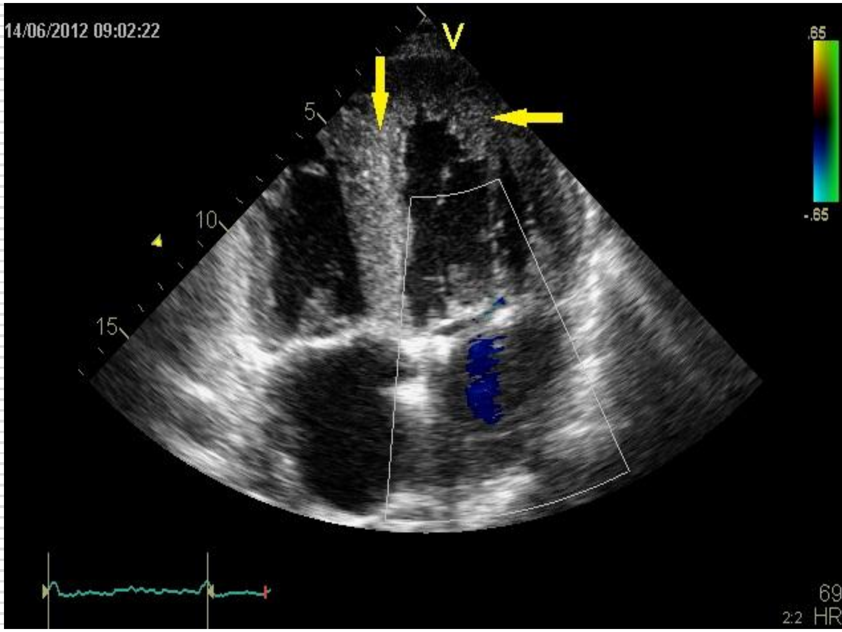
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V
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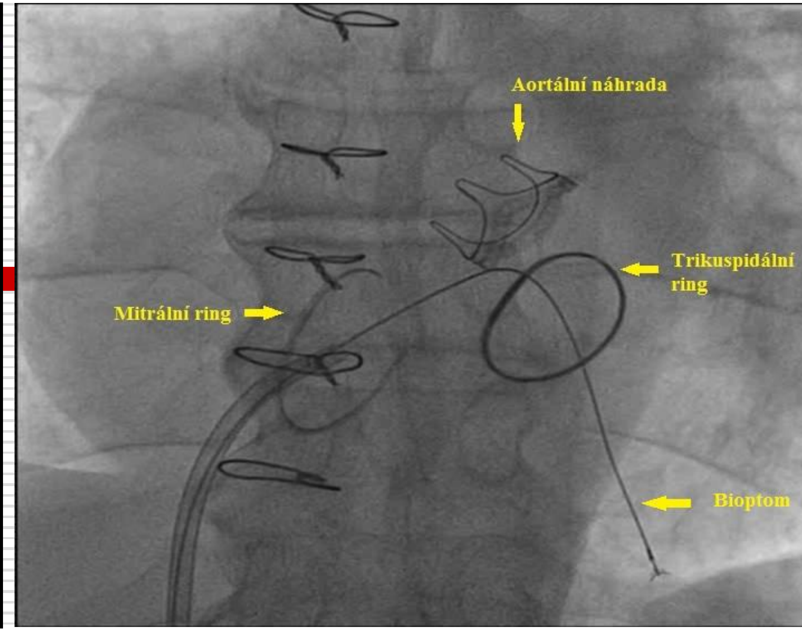
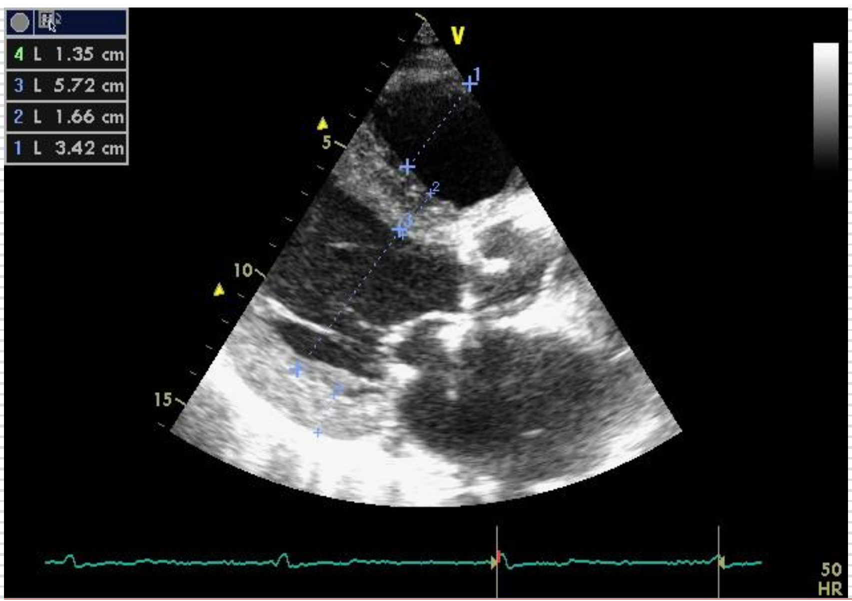

Aortální náhrada
Trikuspidální ring
Mitrální ring
Bioptom


4 L 1.35 cm
3 L 5.72 cm
2 L 1.66 cm
1 L 3.42 cm
V
5
10
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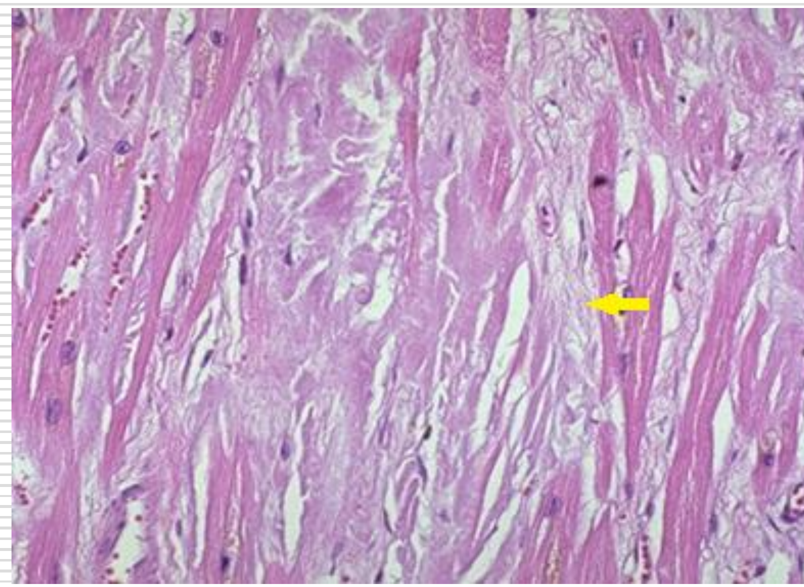
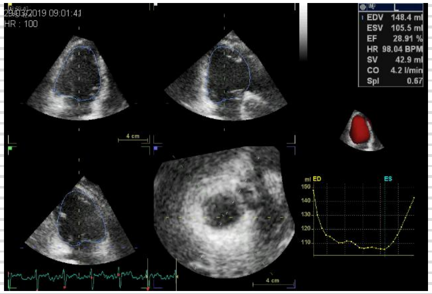
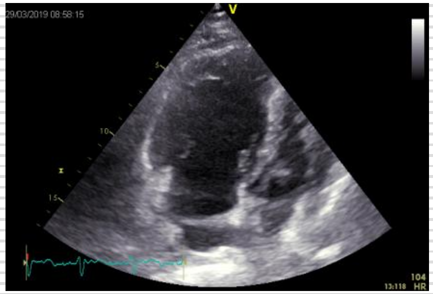
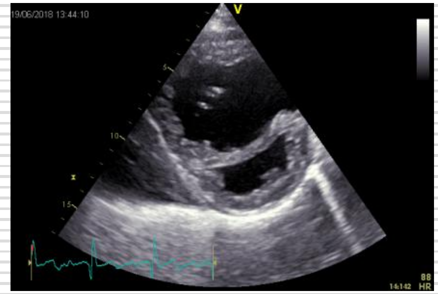
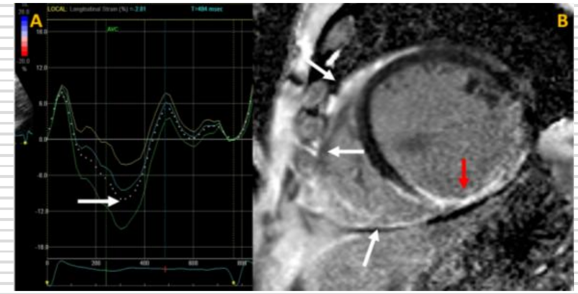
50 HR



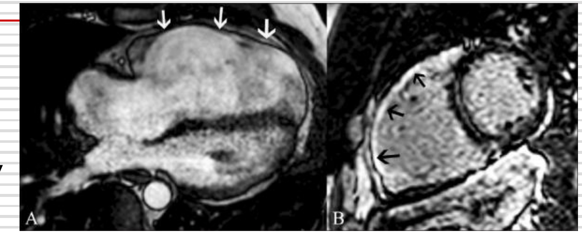








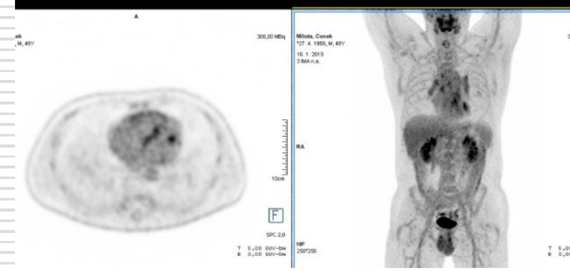
Up to 50% of patients with **acute myocardial infarction** at postmortem show RV involvement. RV injury is more common in inferior infarcts, but also seen in anterior infarcts. After the acute ischemic event, the RV function tends to recover.



In **arrhythmogenic RV cardiomyopathy**, newer studies show preferential involvement of the RV basal inferior and anterior segments in early disease with the LV basal inferolateral segment. Microstructural abnormalities precede the electrical phase of the disease, challenging the conventional notion of electrical disease preceding structural disease.

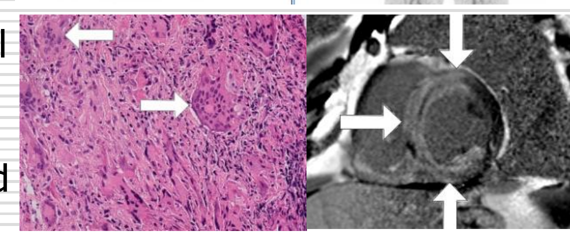
In other **nonischemic cardiomyopathies**, RV dysfunction (EF ≤45%) is present in 35-40% of patients. RV scarring is usually absent.

In **hypertrophic cardiomyopathy**, RV myocardial disarray and hypertrophy are seen in up to 30% of patients.



In **cardiac amyloidosis**, increased RV wall thickness and late enhancement are common. RV dysfunction is related to RV amyloid deposition and LV involvement.

In **acute myocarditis**, approximately 20% of patients have RV free wall involvement and RV involvement signals worse outcomes.



In patients with proven extracardiac **sarcoidosis**, 15-20% show RV free wall or interventricular septum involvement. RV involvement is associated with a higher risk for mortality from ventricular tachyarrhythmias.

Sanz J, Sánchez-Quintana D, Bossone E, Bogaard HJ, Naeije R. Anatomy, Function, and Dysfunction of the Right Ventricle: JACC State-of-the-Art Review. J Am Coll Cardiol 2019;73:1463-1482.

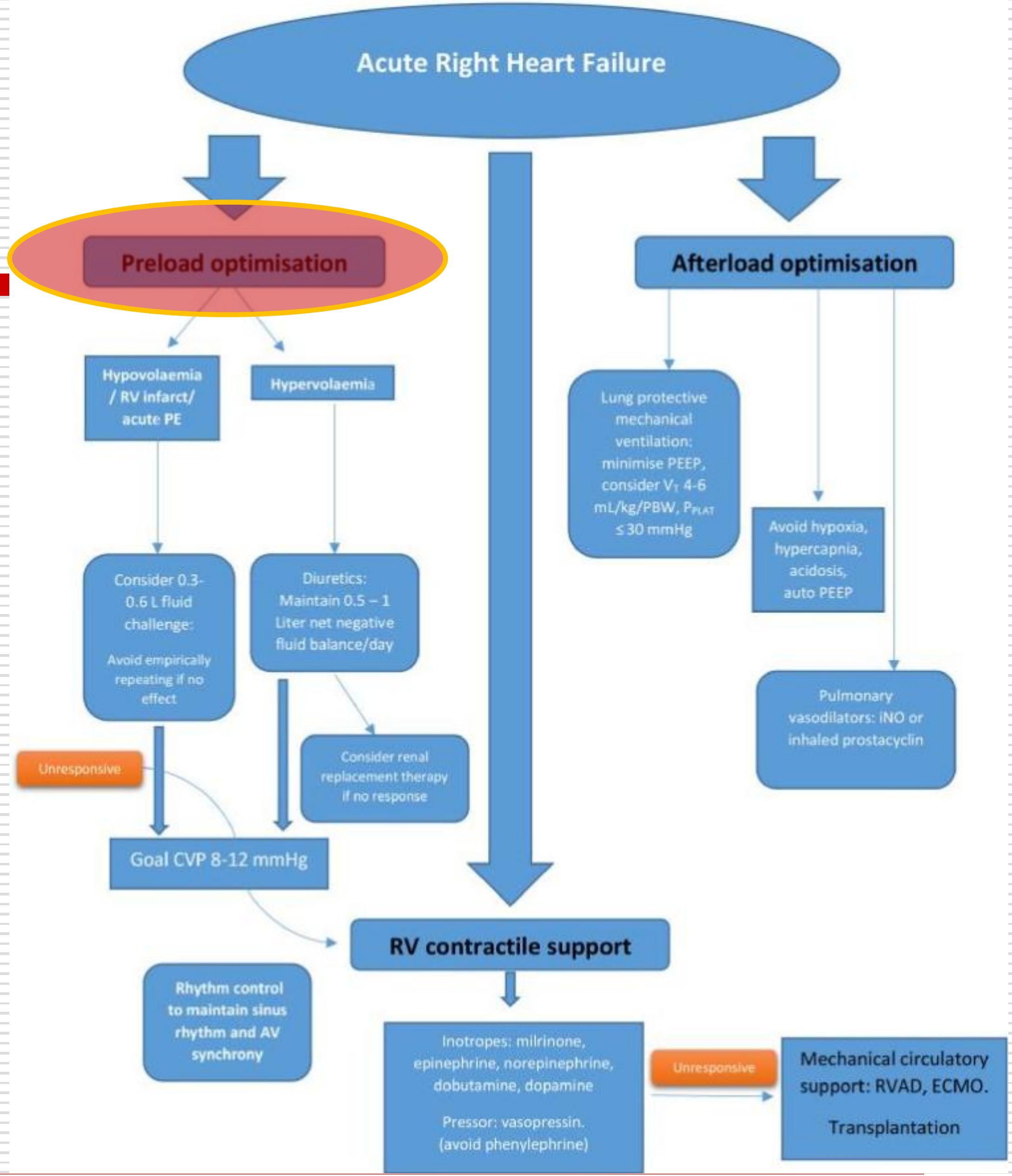
1. Assessment of the right heart is a critical component of every echocardiographic study. Measurement of chamber dimensions, evaluation of RV systolic function, and estimation of hemodynamic parameters (RAP, PASP) should be performed.
2. Following core measurements should be reported:

- **RV basal diameter** from the RV-focused apical four-chamber view (normal ≤4.1 cm), or, if feasible, RV volume from a 3D acquisition.
- **RA volume** from the apical four-chamber view using the single-plane Simpson's method.
- **RA pressure** from the inferior vena cava size and collapse (3/8/15 mmHg).
- **PASP** from the tricuspid regurgitation velocity and estimated RA pressure.
- **RV systolic function** using at least one quantitative parameter: tricuspid annular plane systolic excursion (TAPSE; normal ≥1.7 cm), tricuspid annular velocity (S') (normal ≥9.5 cm/s), fractional area change (FAC; normal ≥35 percent), myocardial performance index (MPI; normal ≤0.43 by pulsed Doppler or ≤0.55 by tissue Doppler). In addition, 3D-derived RV ejection fraction is recommended when suitable technology/expertise is available.

Feature	Criteria (Reference)	Interpretation
Dilatation	Volume >101 mL/m^2[8]	Volume overload
	RV max SAX >43 mm[6]	Pressure overload
	RVEDA/LVEDA >2/3[6]	Intrinsic myocardial disease
D-shaped LV	Eccentricity index >1[49]*	RV pressure or volume overload
		Diastolic D-shape LV suggests volume overload
		Systolic D-shape LV suggests pressure overload
Hypertrophy	Mass >35 g/m^2[8]	Pressure-overloaded RV
	RV inferior wall >5 mm[6]	Hypertrophic cardiomyopathy, infiltrative disease; exclude double-chambered RV
Aneurysm	Localized RV dilatation[6]	ARVD; RVMI; localized absence of pericardium
TV septal insertion	Septal insertion >1 cm or 8 mm/m^2[50]	Consider Ebstein's anomaly
Delayed enhancement	Area of delayed contrast uptake and washout in MRI	Suggests myocardial fibrosis
Fatty infiltration	High-intensity signal on MRI	Consider ARVD

Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1.
Haddad F, Hunt SA, Rosenthal DN., et al. Right Ventricular Function in Cardiovascular. Disease, Part I. Anatomy, Physiology, Aging, and Functional Assessment of the Right Ventricle. *Circulation*. 2008;117:1436-1448.

THERAPY


Acute Right Heart Failure
Preload optimisation
Hypovolaemia / RV infarct/ acute PE
Hypervolaemia
Consider 0.3-0.6 L fluid challenge:
Avoid empirically repeating if no effect
Diuretics: Maintain 0.5 – 1 Liter net negative fluid balance/day
Consider renal replacement therapy if no response
Unresponsive
Goal CVP 8-12 mmHg
Afterload optimisation
Lung protective mechanical ventilation: minimise PEEP, consider V_T 4-6 mL/kg/PBW, P_{PLAT} ≤30 mmHg
Avoid hypoxia, hypercapnia, acidosis, auto PEEP
Pulmonary vasodilators: iNO or inhaled prostacyclin
RV contractile support
Rhythm control to maintain sinus rhythm and AV synchrony
Inotropes: milrinone, epinephrine, norepinephrine, dobutamine, dopamine
Pressor: vasopressin. (avoid phenylephrine)
Unresponsive
Mechanical circulatory support: RVAD, ECMO.
Transplantation


The NEW ENGLAND JOURNAL of MEDICINE

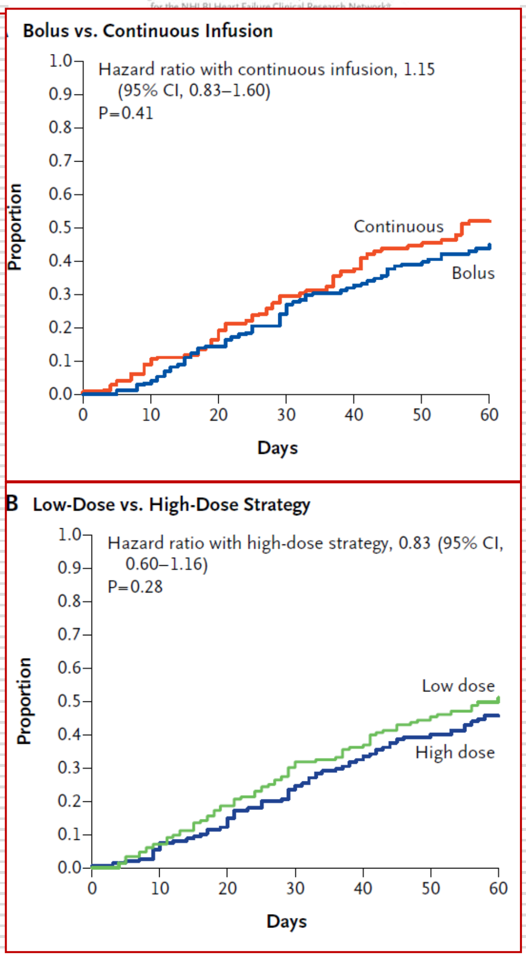
ESTABLISHED IN 1812 MARCH 3, 2011 VOL. 364 NO. 9

Diuretic Strategies in Patients with Acute Decompensated Heart Failure

G. Michael Felker, M.D., M.H.S., Kerry L. Lee, Ph.D., David A. Bull, M.D., Margaret M. Redfield, M.D., Lynne W. Stevenson, M.D., Steven R. Goldsmith, M.D., Martin M. LeWinter, M.D., Anita Deswal, M.D., M.P.H., Jean L. Rouleau, M.D., Elizabeth O. Ofili, M.D., M.P.H., Kevin J. Anstrom, Ph.D., Adrian F. Hernandez, M.D., Steven E. McNulty, M.S., Eric J. Velazquez, M.D., Abdallah G. Kfoury, M.D., Horng H. Chen, M.B., B.Ch., Michael M. Givertz, M.D., Marc J. Semigran, M.D., Bradley A. Bart, M.D., Alice M. Mascette, M.D., Eugene Braunwald, M.D., and Christopher M. O'Connor, M.D.,

Critically ill patients may have reduced right heart preload due to volume loss, reduced venous tone from medications, sepsis or vasoplegia, and positive pressure ventilation.

However, the majority of conditions leading to RHF are characterised by high RV afterload. In these scenarios, reducing excessive RV preload with diuretics or haemofiltration is key to reducing RV dilatation and free wall tension, thereby minimising RV ischaemia and optimising contractility. It is generally agreed that maintaining a moderately high RV diastolic filling pressure of 8-12 mmHg is optimal in RHF.



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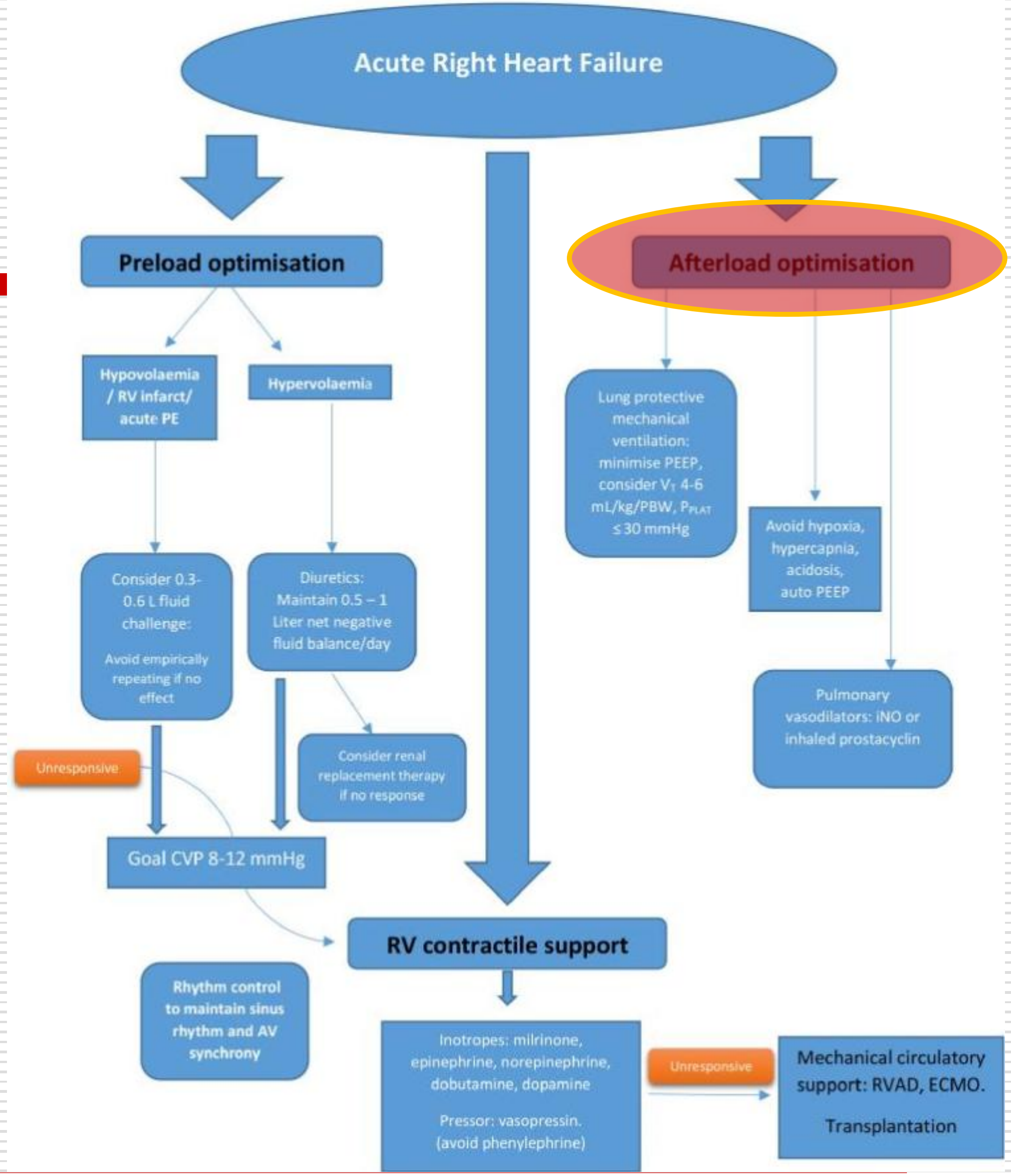
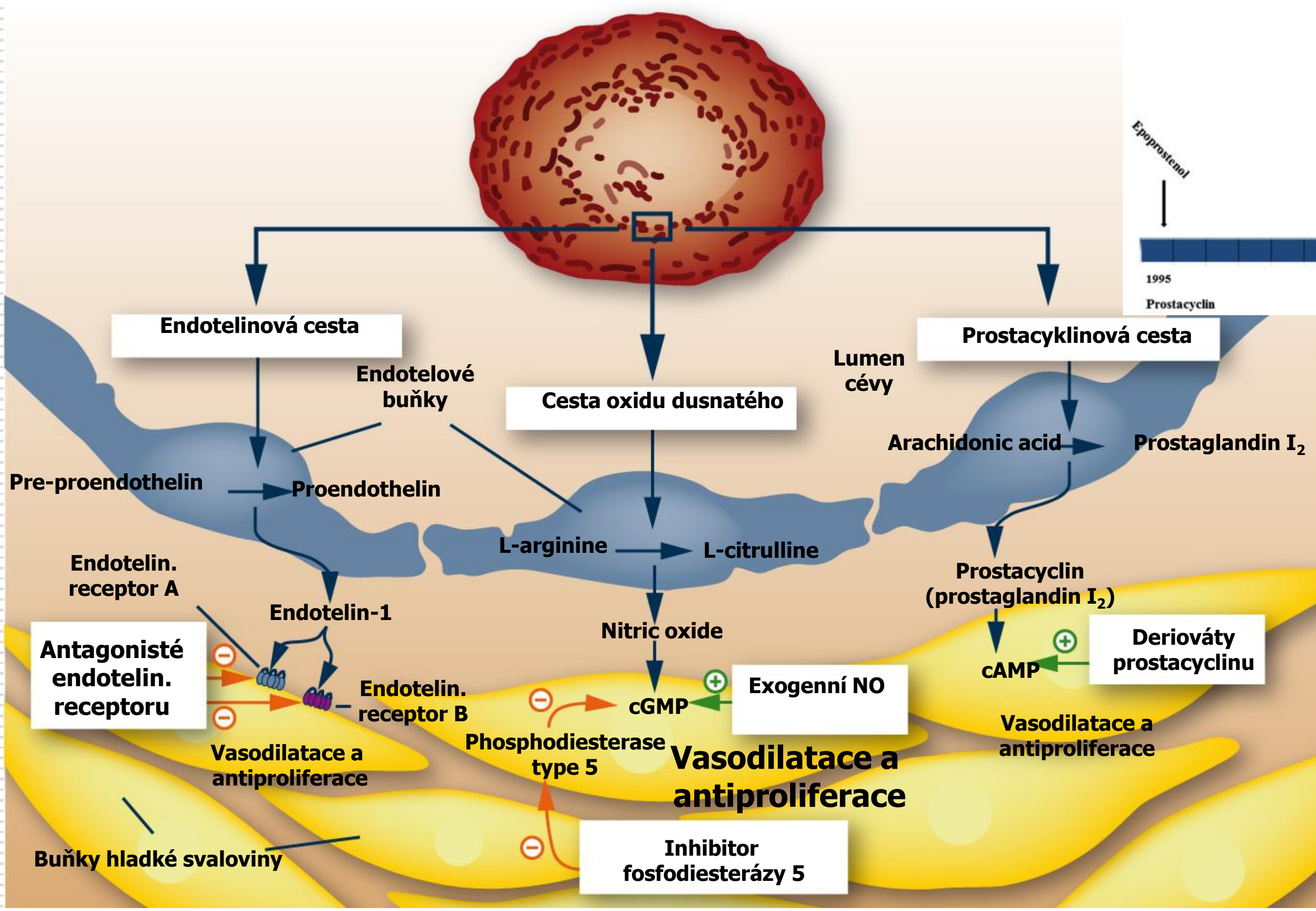
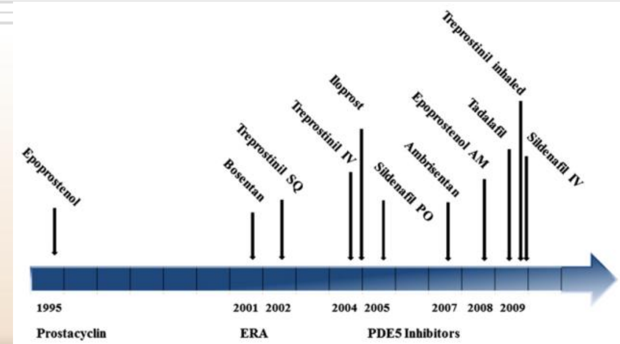
THERAPY

General measures are aimed at correcting conditions that can increase PVR in critically ill patients. These conditions include acidosis, hypoxia (which causes pulmonary vasoconstriction), and hypercapnia.
Lung protective ventilation, using the lowest effective plateau pressure, tidal volume, and positive end-expiratory pressure while avoiding hypoxaemia and hypercarbia, assists with optimising both RV preload and afterload.

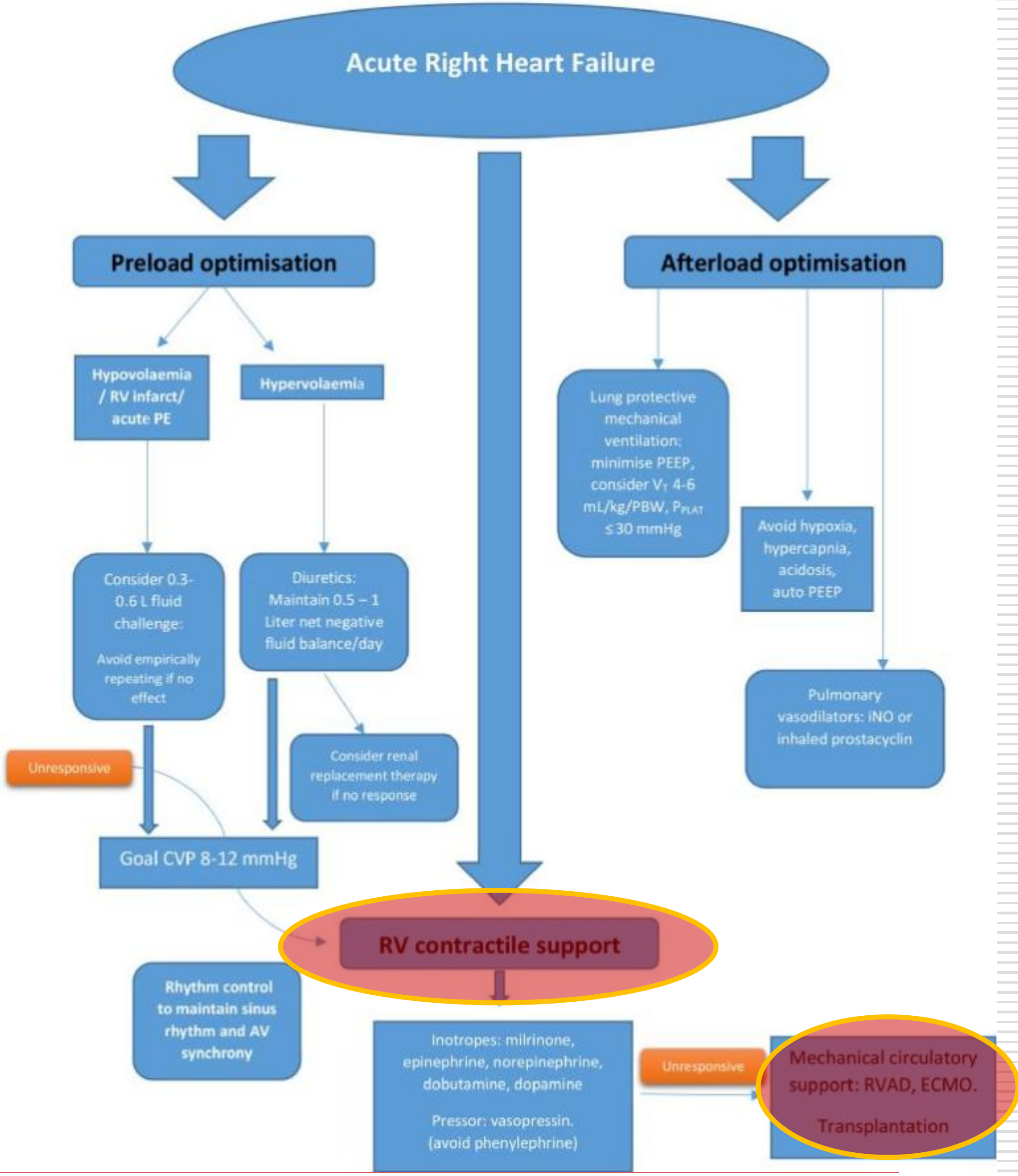




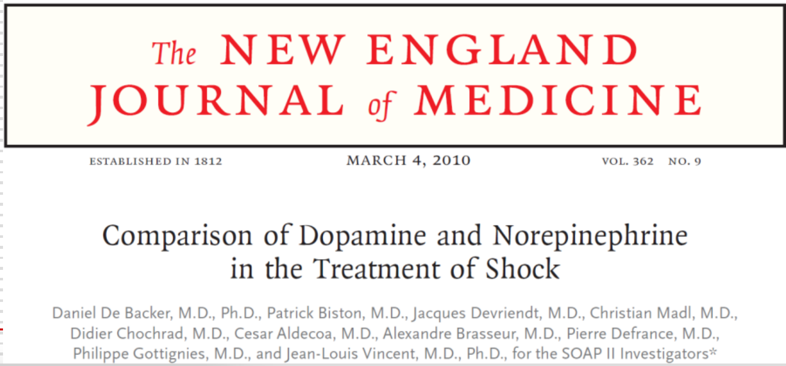


Humbert; NEJM (2004)

THERAPY



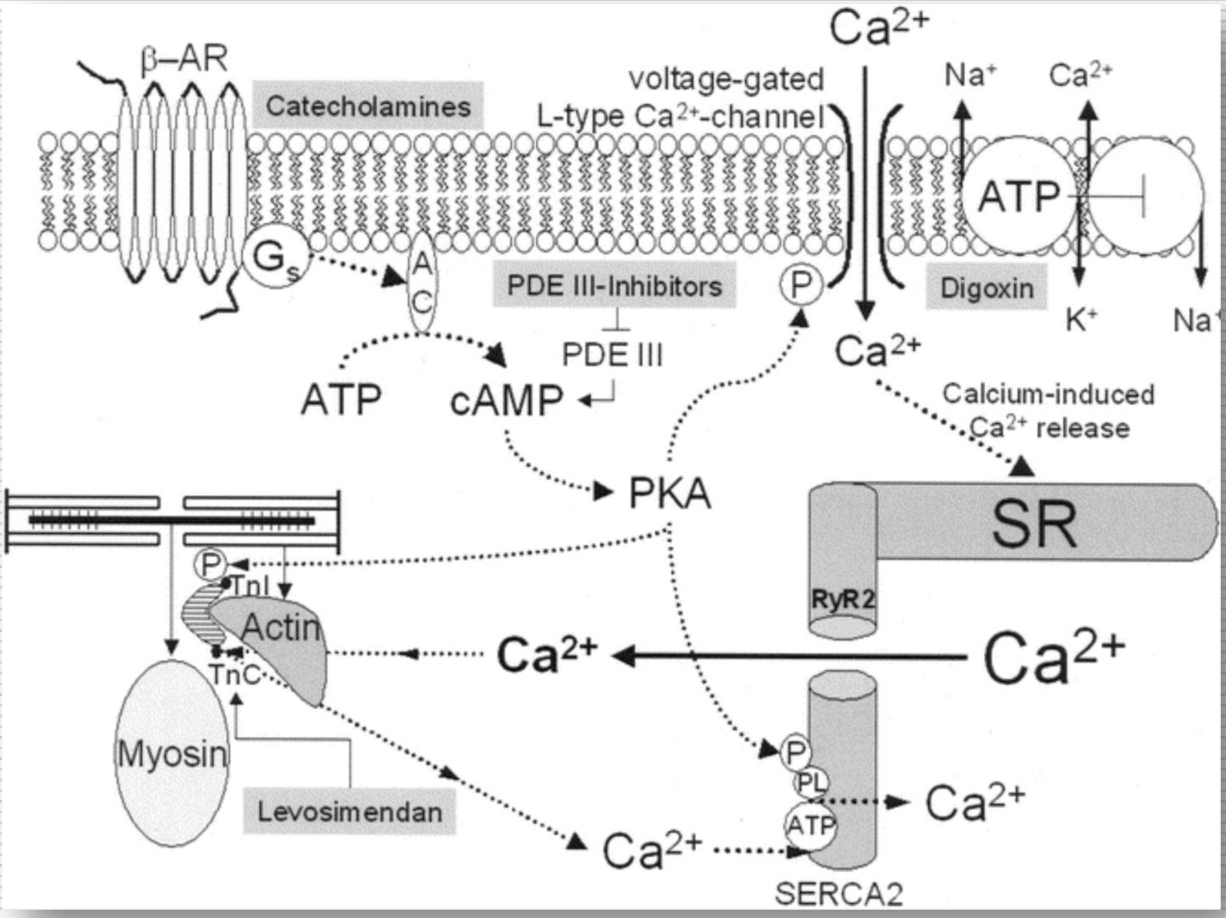
Acute Right Heart Failure
Preload optimisation
Afterload optimisation
Hypovolaemia / RV infarct/ acute PE
Hypervolaemia
Lung protective mechanical ventilation: minimise PEEP, consider V_T 4-6 mL/kg/PBW, P_{PLAT} ≤ 30 mmHg
Avoid hypoxia, hypercapnia, acidosis, auto PEEP
Consider 0.3-0.6 L fluid challenge:
Avoid empirically repeating if no effect
Diuretics: Maintain 0.5 – 1 Liter net negative fluid balance/day
Pulmonary vasodilators: iNO or inhaled prostacyclin
Unresponsive
Consider renal replacement therapy if no response
Goal CVP 8-12 mmHg
RV contractile support
Rhythm control to maintain sinus rhythm and AV synchrony
Inotropes: milrinone, epinephrine, norepinephrine, dobutamine, dopamine
Pressor: vasopressin. (avoid phenylephrine)
Unresponsive
Mechanical circulatory support: RVAD, ECMO.
Transplantation


The NEW ENGLAND JOURNAL of MEDICINE

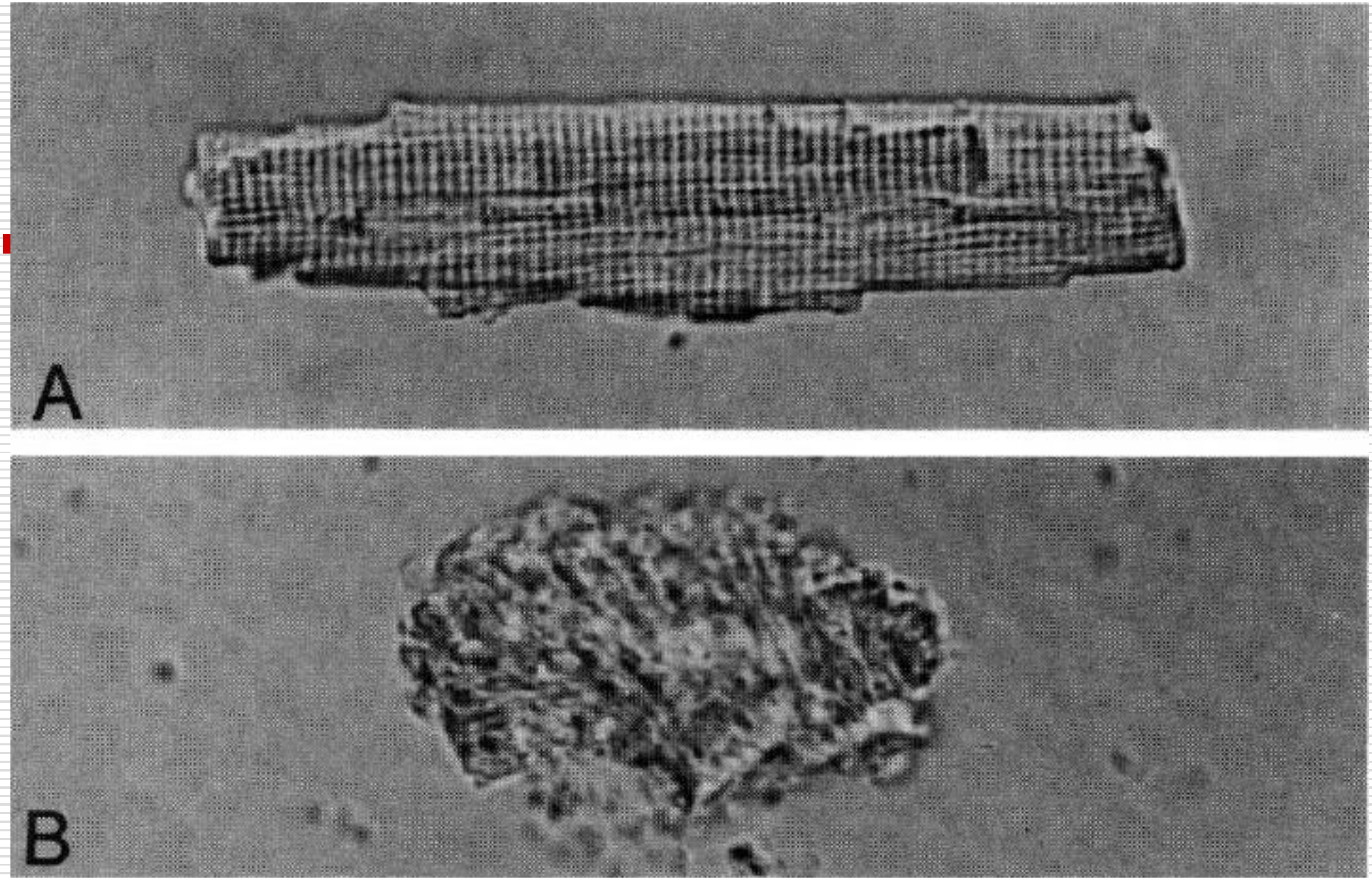
ESTABLISHED IN 1812 — MARCH 4, 2010 — VOL. 362 NO. 9

Comparison of Dopamine and Norepinephrine in the Treatment of Shock

Daniel De Backer, M.D., Ph.D., Patrick Biston, M.D., Jacques Devriendt, M.D., Christian Madl, M.D., Didier Chochrad, M.D., Cesar Aldecoa, M.D., Alexandre Brasseur, M.D., Pierre Defrance, M.D., Philippe Gottignies, M.D., and Jean-Louis Vincent, M.D., Ph.D., for the SOAP II Investigators*



Levosimendan, a New Inotropic and Vasodilator Agent Anesthes. 2006;104(3):556-569.



Because catecholamines increase myocardial oxygen consumption and vasoconstrictors may impair microcirculation as well as tissue perfusion, their use should be restricted to the shortest possible duration and the lowest possible dose.

De Backer D. et al. Comparison of dopamine and norepinephrine in the treatment of shock. *N Engl J Med* 2010;362:779–789
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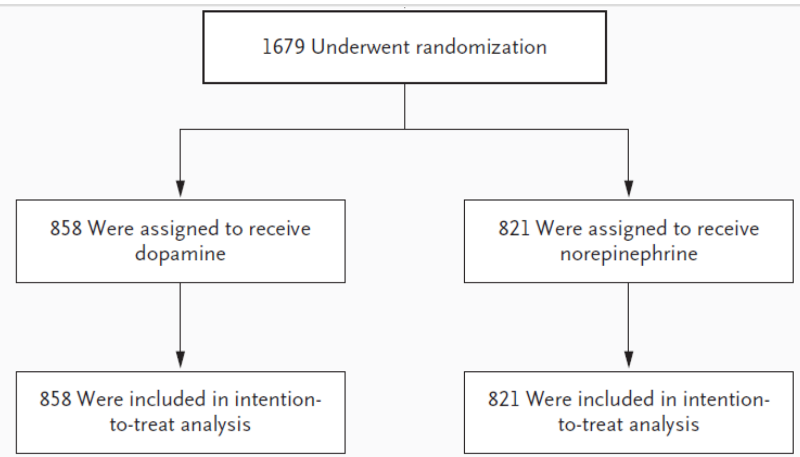

Variable	Dopamine (N = 858)	Norepinephrine (N = 821)
Age — yr		
Median	68	67
Interquartile range	55–76	56–76
Male sex — no. (%)	507 (59.1)	449 (54.7)
APACHE II score†		
Median	20	20
Interquartile range	15–28	14–27
SOFA score‡		
Median	9	9
Interquartile range	7–12	6–12
Reason for admission — no. (%)		
Medical	565 (65.9)	532 (64.8)
Scheduled surgery	168 (19.6)	161 (19.6)
Emergency surgery	125 (14.6)	128 (15.6)
Cause of shock — no. (%)		
Sepsis	542 (63.2)	502 (61.1)
Lungs	278 (32.4)	246 (30.0)
Abdomen	138 (16.1)	135 (16.4)
Urine	51 (5.9)	42 (5.1)
Catheter	14 (1.6)	10 (1.2)
Endocardium	9 (1.0)	11 (1.3)
Mediastinum	10 (1.2)	15 (1.8)
Soft tissues	11 (1.3)	13 (1.6)
Other	15 (1.7)	20 (2.4)
Cardiogenic source	135 (15.7)	145 (17.6)
Myocardial infarction	75 (8.7)	86 (10.5)
Dilated cardiomyopathy	25 (2.9)	19 (2.3)
Tamponade	2 (0.2)	7 (0.9)
Pulmonary embolism	10 (1.2)	8 (1.0)
Valvular disease	4 (0.5)	5 (0.6)
After cardiopulmonary bypass	19 (2.2)	20 (2.4)
Other		
Hypovolemia	138 (16.1)	125 (15.2)
Hemorrhage	130 (15.2)	116 (14.1)
Trauma	17 (2.0)	23 (2.8)
Gastrointestinal bleeding	31 (3.6)	22 (2.7)
Bleeding at surgical site	64 (7.5)	57 (6.9)
Other	18 (2.1)	14 (1.7)
Dehydration	8 (0.9)	9 (1.1)

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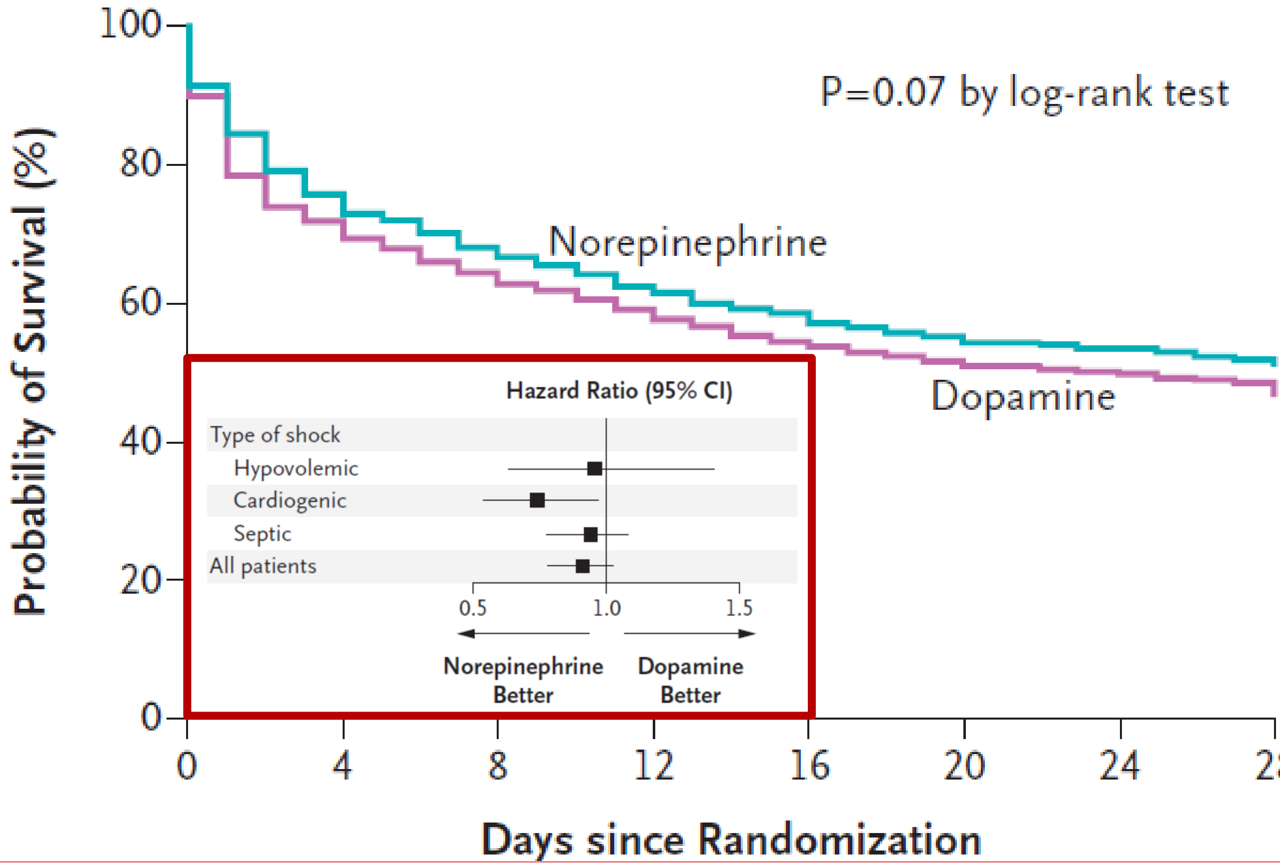
Time Period	Dopamine	Norepinephrine	Odds Ratio (95% CI)†	P Value
	percent mortality			
During stay in intensive care unit	50.2	45.9	1.19 (0.98–1.44)	0.07
During hospital stay	59.4	56.6	1.12 (0.92–1.37)	0.24
At 28 days	52.5	48.5	1.17 (0.97–1.42)	0.10
At 6 mo	63.8	62.9	1.06 (0.86–1.31)	0.71
At 12 mo	65.9	63.0	1.15 (0.91–1.46)	0.34



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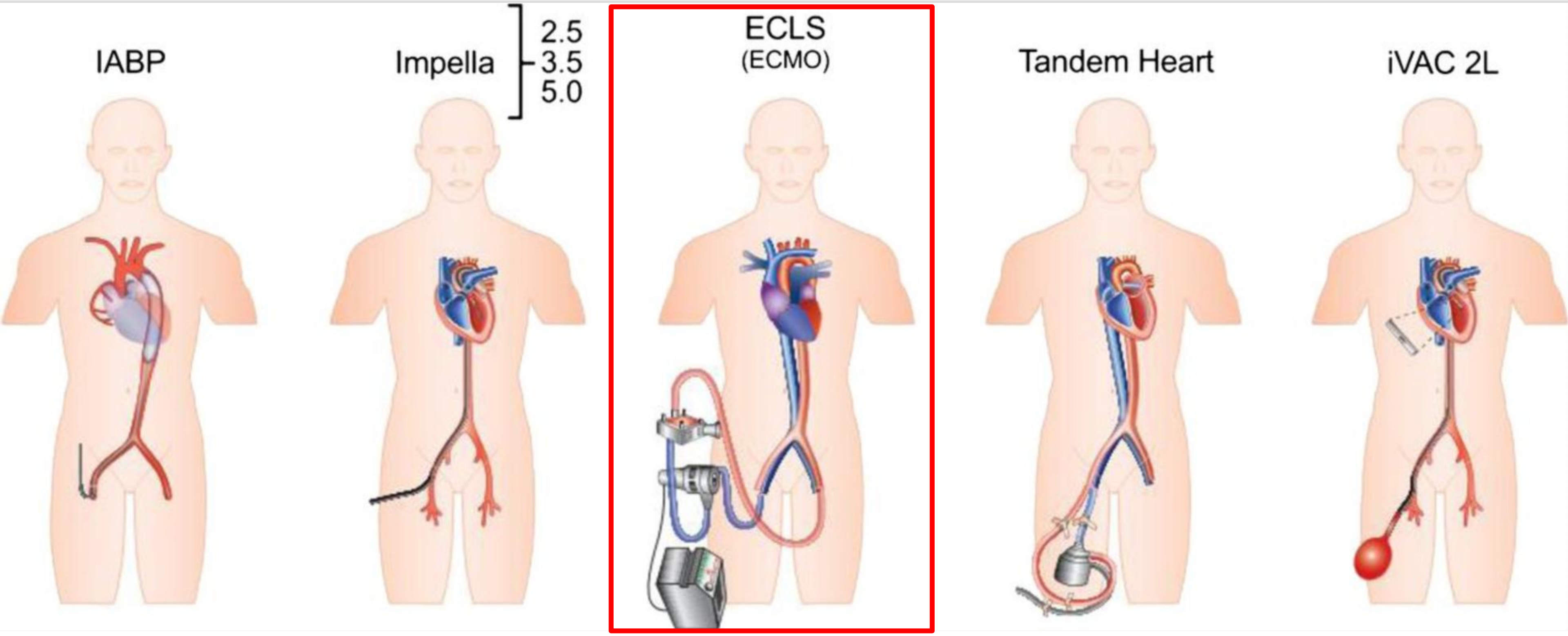
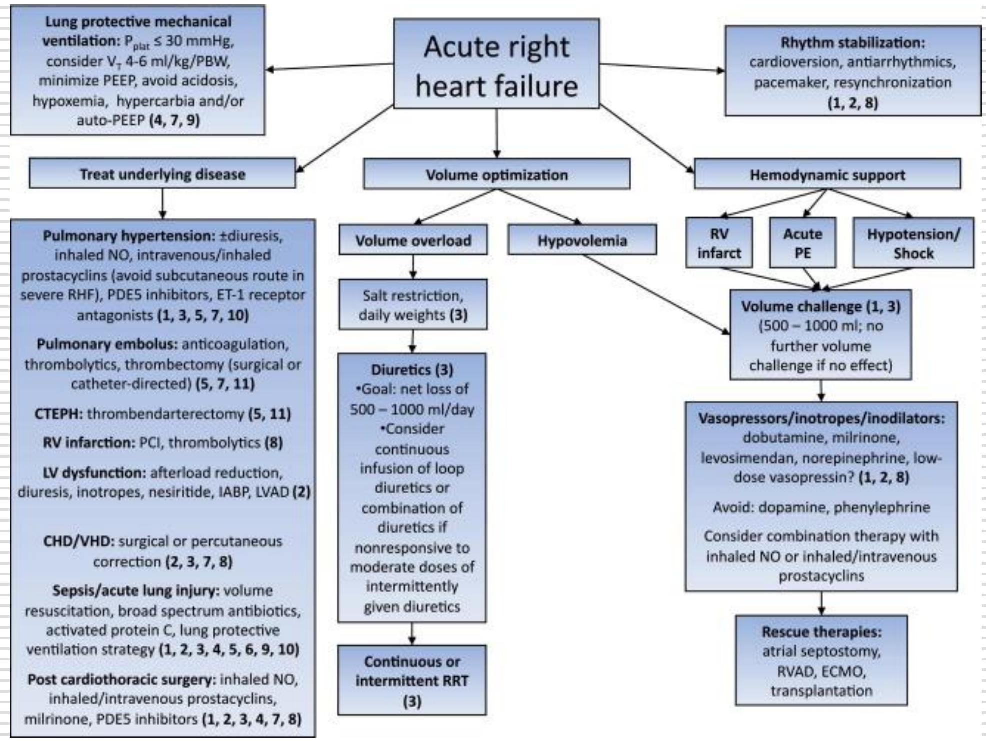
Mechanical circulatory support devices and treating the underlying cause of right heart failure

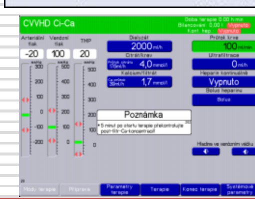


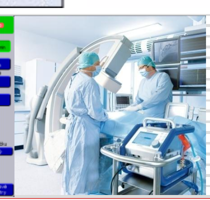
Thiele H. et al. Management of cardiogenic shock. Eur Heart J. 2015;36(20):1223-1230. doi:10.1093/eurheartj/ehv051

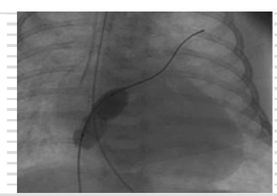
SUMMARY AND RECOMMENDATIONS

The management of isolated acute right heart failure remains **more of an art than a science** in the absence of robust randomised data.
In addition to **(1) treating the specific cause**, **(2) RV preload optimisation,** the use of **(3) selective pulmonary vasodilators, (4) RV inotropic support** and **(5) temporary mechanical circulatory device** therapy form integral components of a comprehensive strategy to support the failing right heart.









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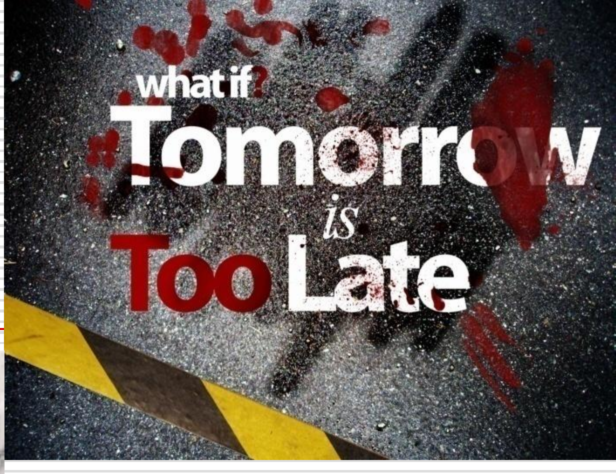

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