

# **The right heart: the Cinderella of heart failure**

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# Disclosure

none

# Look into the Heart Failure ESC guidelines



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## ESC GUIDELINES

### ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012

**The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC**

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# **Right heart (RV) in the HF ESC Guidelines**

- 1. RV & LV interaction – cause of limited cardiac reserve**
- 2. RVF – other cause of elevated BNP / NT-proBNP (diagnosis)**
- 3. Echocardiography – information about RV function / RA pressure**
- 4. AVRC – imaging techniques**
- 5. RV failure as a potential cause of renal dysfunction**
- 6. RV infarction – cause of acute deterioration / AHF**
- 7. Adult congenital heart disease**
- 8. MCS – need for RV function assessment; deteriorating RV function as an indication for VAD implantation**
- 9. Isolated RV failure**

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# Right heart (RV) in the HF ESC Guidelines

## 12.7.2 Isolated right ventricular failure

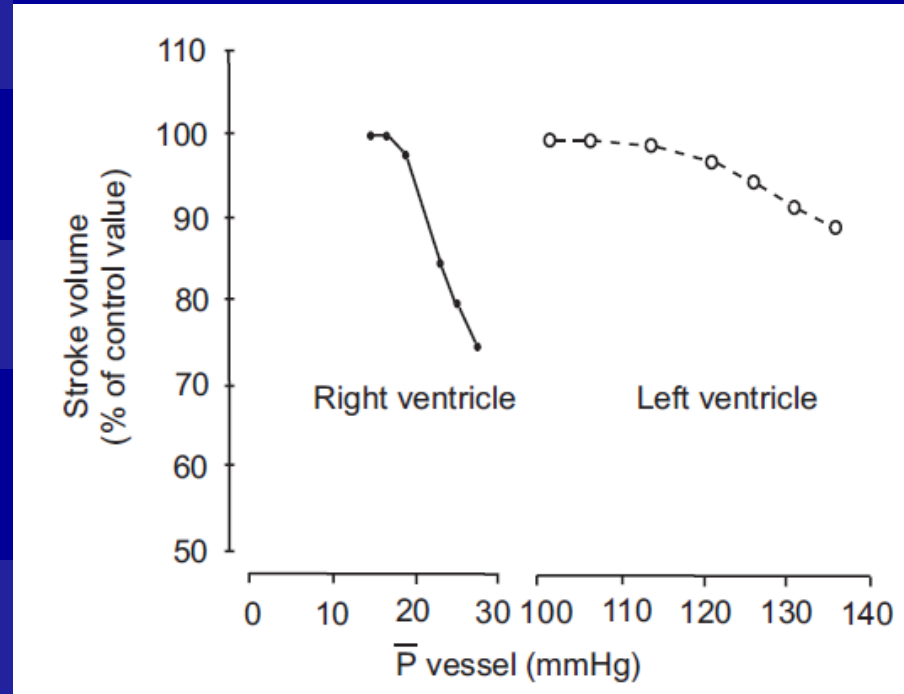
New-onset isolated right ventricular failure may occur secondarily to an acute coronary syndrome (and is managed as described above) and following massive pulmonary embolism (see pulmonary embolism guidelines<sup>239</sup>). In both situations, diuretics and vasodilators should be used cautiously or avoided so as not to reduce right ventricular filling.

Progressive isolated right ventricular failure may occur in patients with pulmonary hypertension. Type V phosphodiesterase inhibitors, endothelin antagonists, and prostacyclin analogues may help by decreasing pulmonary arterial resistance (see guidelines<sup>240</sup>).

## 9. Isolated RV failure

# The normal right ventricle

Characteristic	RV	LV
End Diastolic Volume (ml)	75±13 (49-101)	66±12 (44-89)
Mass (g/m <sup>2</sup> )	26±5 (17-34)	87±12 (64-109)
Wall thickness (mm)	2 – 5	7 – 11
Ejection Fraction (%)	61±7 (47-76) NI: >40-45	67±5 (57-78) NI>50
Ventricular pressure (mmHg)	25/4 [(15-30)/(1-7)]	130/8 [(90-140)/(5-12)]
Vascular Resistance (dyne·s·cm <sup>-5</sup> )	70 (20-130)	1100 (700-1600)



**The response of the RV and LV to experimental increase in afterload**

**Comparison of normal LV and RV structure and function**

Haddad F et al. *Circulation* 2008;117:1436-48.

# **RV: “the take home message”**

**the main aim of the RV is to**

**keep RVEDP or RAP**

**as low as possible**

**to optimize the venous return**

**to provide sustained low-pressure perfusion  
through the lungs**

# Right ventricle

- generates **flow**
- pulmonary vessels:
  - low resistances, transpulmonary gradient = 5mmHg
  - great distensibility
  - no changes in compliance throughout the lungs
  - no reflection

## **Special Report**

### **Right Ventricular Function and Failure**

#### **Report of a National Heart, Lung, and Blood Institute Working Group on Cellular and Molecular Mechanisms of Right Heart Failure**

Norbert F. Voelkel, MD; Robert A. Quaife, MD; Leslie A. Leinwand, PhD; Robyn J. Barst, MD; Michael D. McGoon, MD; Daniel R. Meldrum, MD; Jocelyn Dupuis, MD, PhD; Carlin S. Long, MD; Lewis J. Rubin, MD; Frank W. Smart, MD; Yuichiro J. Suzuki, PhD; Mark Gladwin, MD; Elizabeth M. Denholm, PhD; Dorothy B. Gail, PhD

*Circulation 2006;114:1883-91*

## **Contemporary Reviews in Cardiovascular Medicine**

### **Right Ventricular Function in Cardiovascular Disease, Part II**

#### **Pathophysiology, Clinical Importance, and Management of Right Ventricular Failure**

François Haddad, MD; Ramona Doyle, MD; Daniel J. Murphy, MD; Sharon A. Hunt, MD

*Circulation 2008;117:1436-48.*



# Right ventricular failure

RV failure is a complex clinical syndrome that can result from any structural or functional cardiovascular disorder that impairs the ability of the RV to fill or to eject blood.

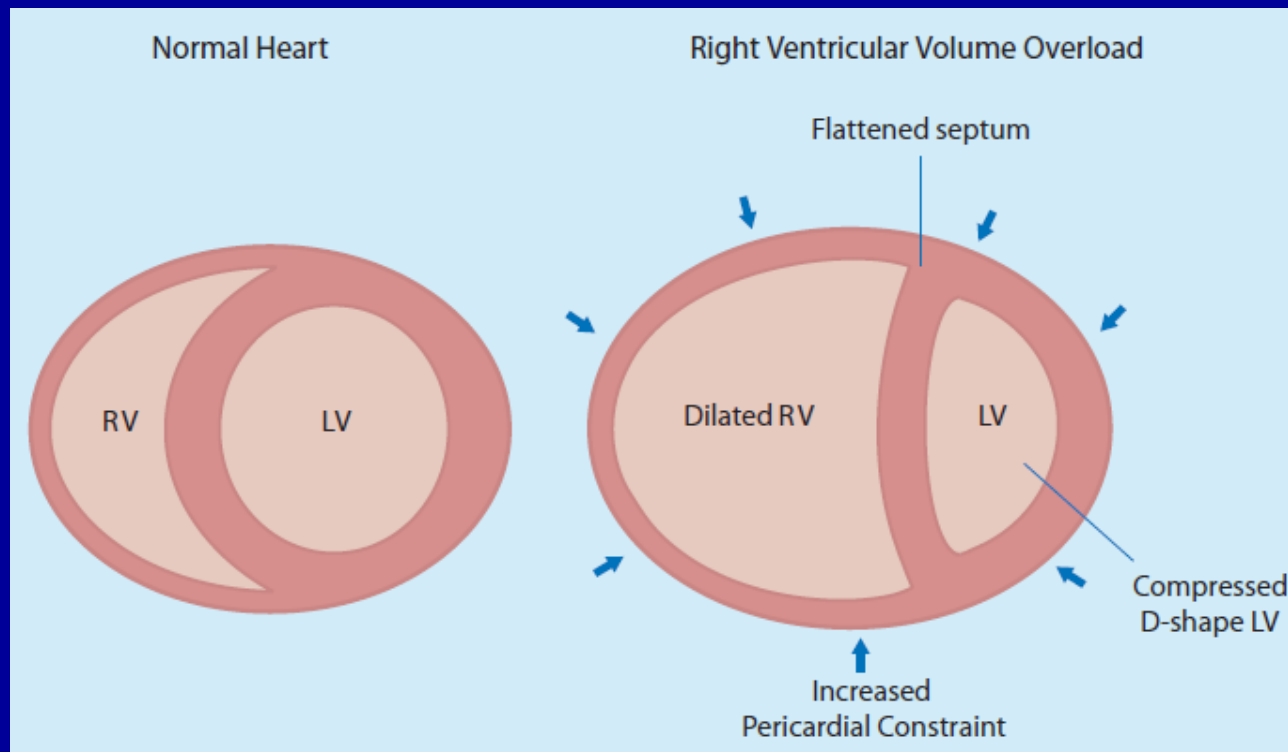
The cardinal clinical manifestations of RV failure are:

- (1) **fluid retention**, which may lead to peripheral edema, ascites, and anasarca;
- (2) **decreased systolic reserve or low cardiac output**, which may lead to exercise intolerance and fatigue;
- (3) **atrial or ventricular arrhythmias**

# RV and LV are interlinked:

- by a shared wall (the septum)
- by mutually encircling epicardial fibers
- by attachment of the RV free wall to the anterior and posterior septum
- by sharing the pericardial space

*Voelkel NF et al. Circulation 2006;114:1883-91*



*Haddad F et al. Circulation 2008;117:1436-48.*

# Right ventricular failure

## Mechanisms & Specific Causes

### 1. Pressure overload

left-sided HF (most common cause)

other causes of pulmonary hypertension

...

### 2. Volume overload

### 3. Ischaemia and infarction

### 4. Intrinsic myocardial process

cardiomyopathy and heart failure

### 5. Inflow limitation

### 6. Complex congenital defects

### 7. Pericardial disease

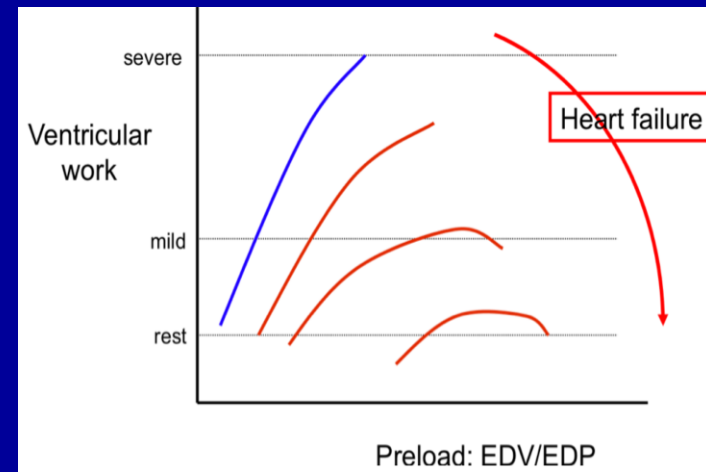
# STARLINGS LAW

- RV is subject to the same haemodynamic mechanisms as the left: as the right heart fails, so a higher filling pressure is required to maintain right heart output. In health, the CVP is around zero.

- RV **-very sensitive to changes in afterload,** more compliant than the LV.

**accommodates volume overload well,**  
with only small increases in systemic  
venous pressure

**tolerates pressure overload poorly**  
(hence R heart failure in L heart failure  
or pulmonary vascular pathology)



# Auto-aggravation of CO decrease in ARVF

Right Ventricular Failure



Reduction in CO + RV dilatation + reduction in LV preload



← **tricuspid regurgitation**



**hypotension**



**decrease in RCPP**

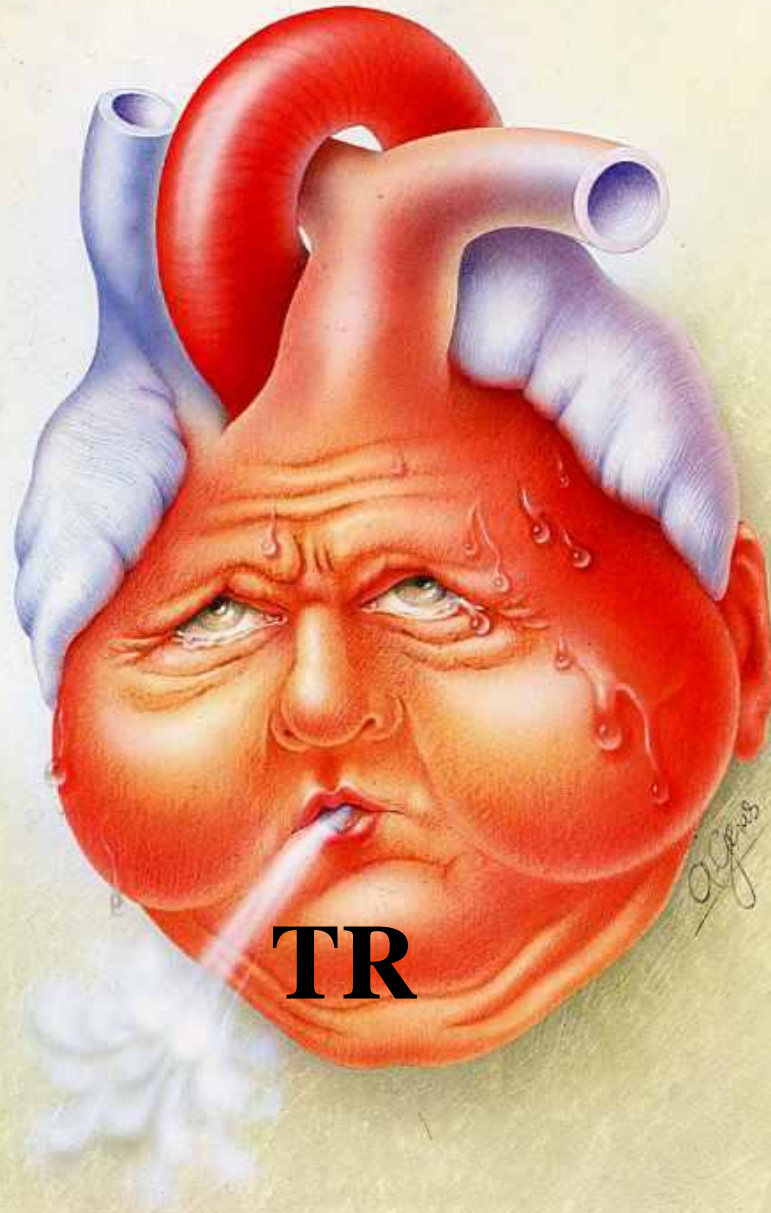


**organ's hypoperfusion  
+ congestion (acidosis, ...)**



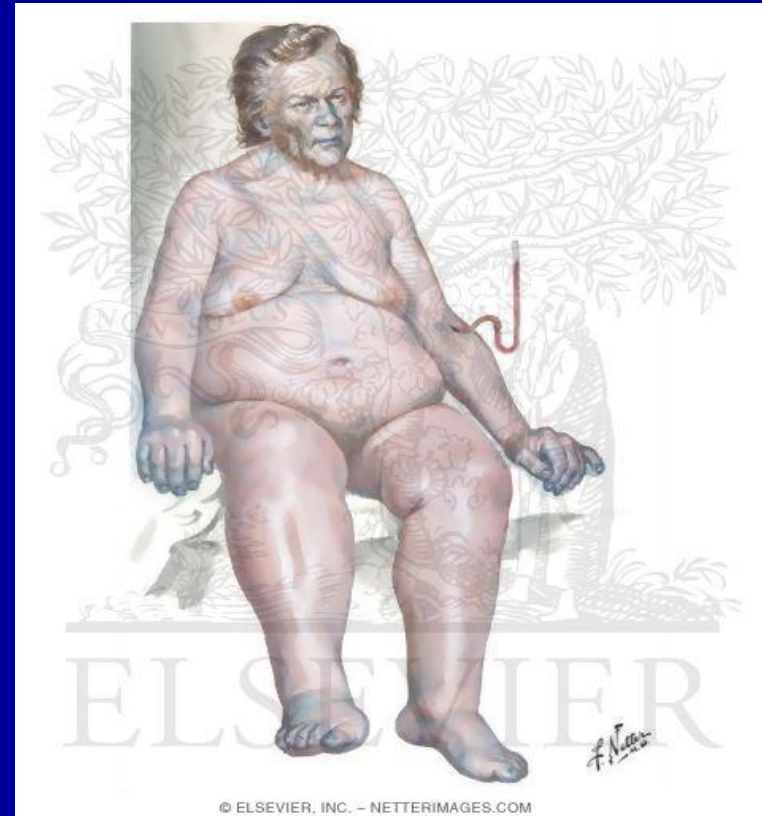
**greater reduction in CO**

# Dilated Right Ventricle !



# The Syndrome of “Right Heart Failure”

- Characterised by Peripheral Oedema
  - Ankle, sacral oedema
  - Ascites
- Anasarca-ανασαρα
- Genuine Fluid overload
  - At least 5 litres for ankle oedema
  - Often , 20 litres or more
- Gradual
  - Key stage, neurohormonal response to poor renal perfusion
  - Increase venous hydrostatic pressure
  - Starling forces
  - Fluid loss from vessels into tissues





# The Syndrome of “Right Heart Failure”

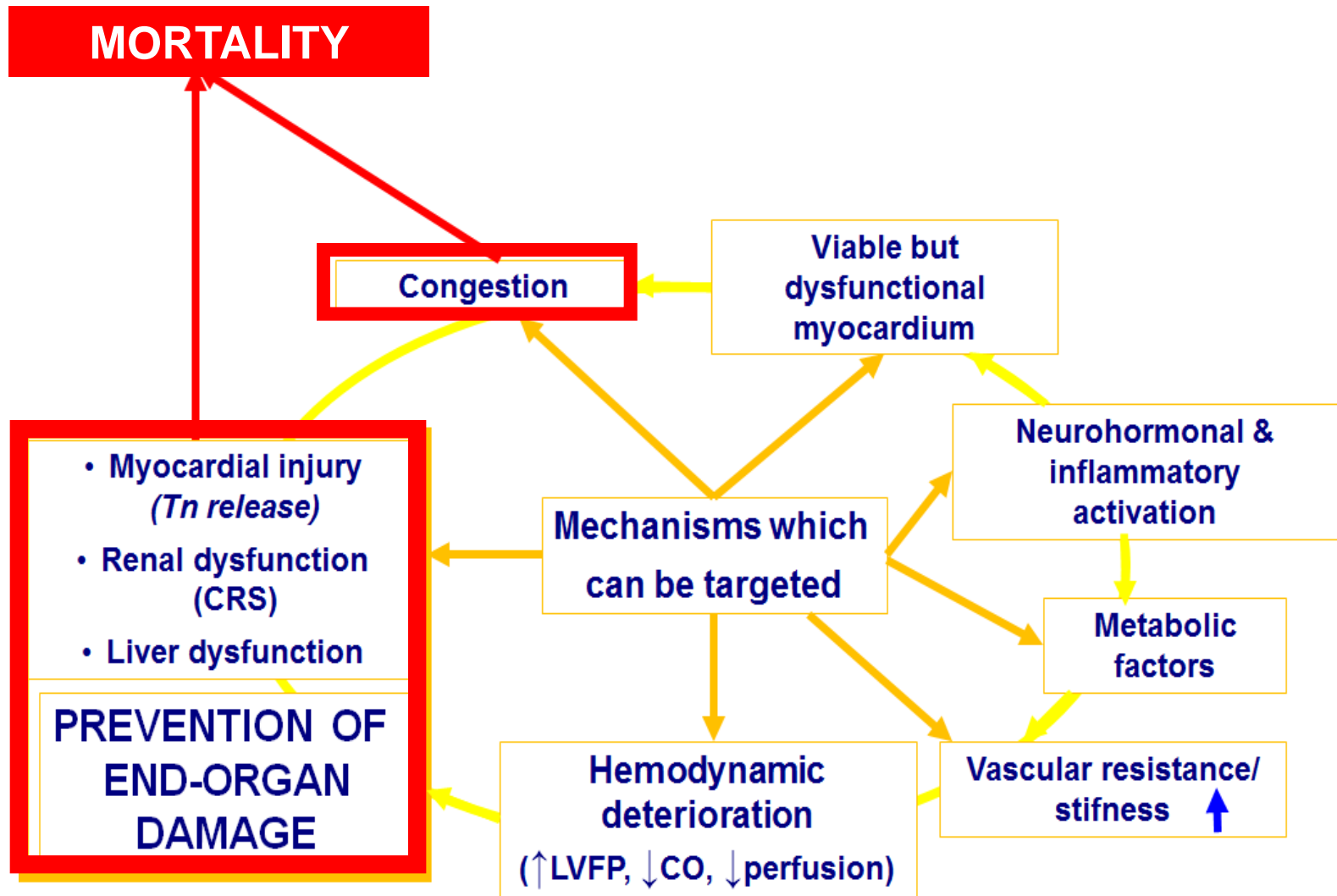
## Clinical signs

- Ankle oedema
  - Legs
  - Abdominal wall
- Pleural effusions
- Ascites
- Pericardial effusions
- Sinus tachycardia or AF,
- low SBP,  $\uparrow$ JVP, TR, cardiomegaly, S3
  - **Clear lungs or some pulmonary oedema**

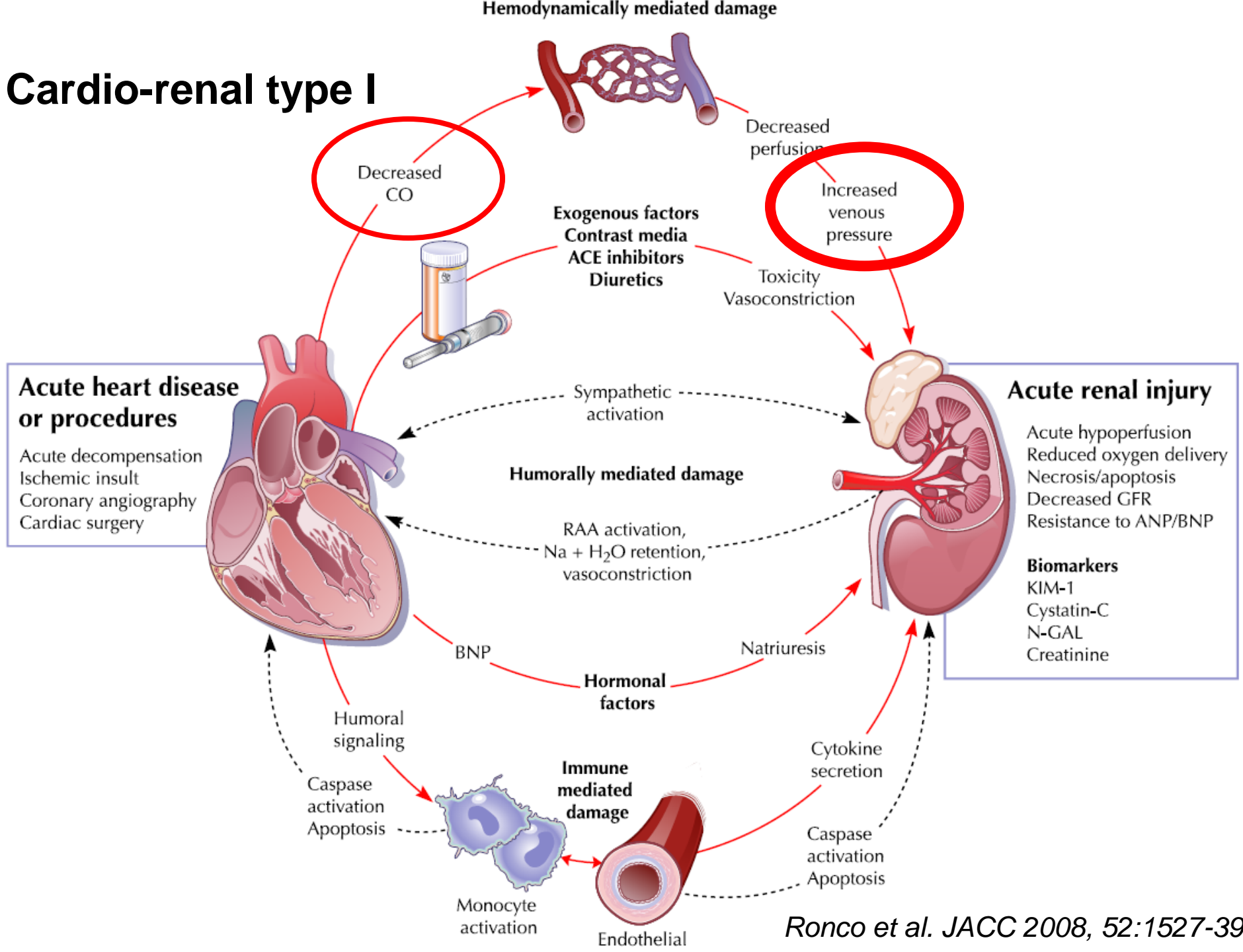




# Better understanding of Acute Heart Failure pathophysiology

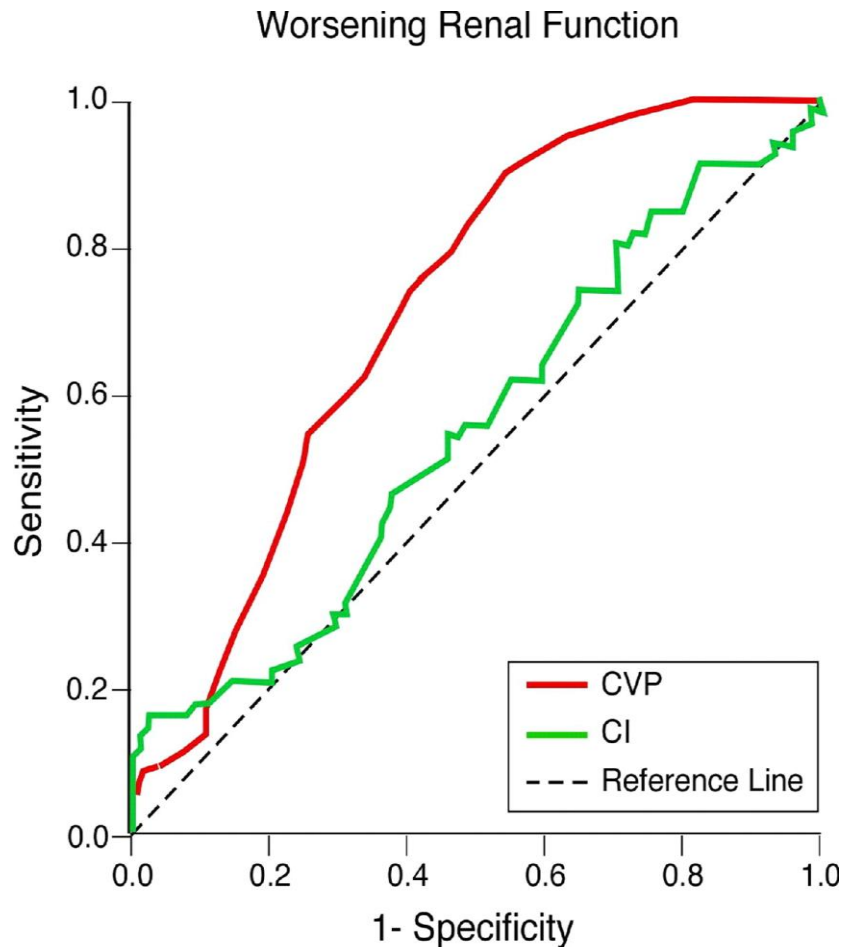


# Cardio-renal type I

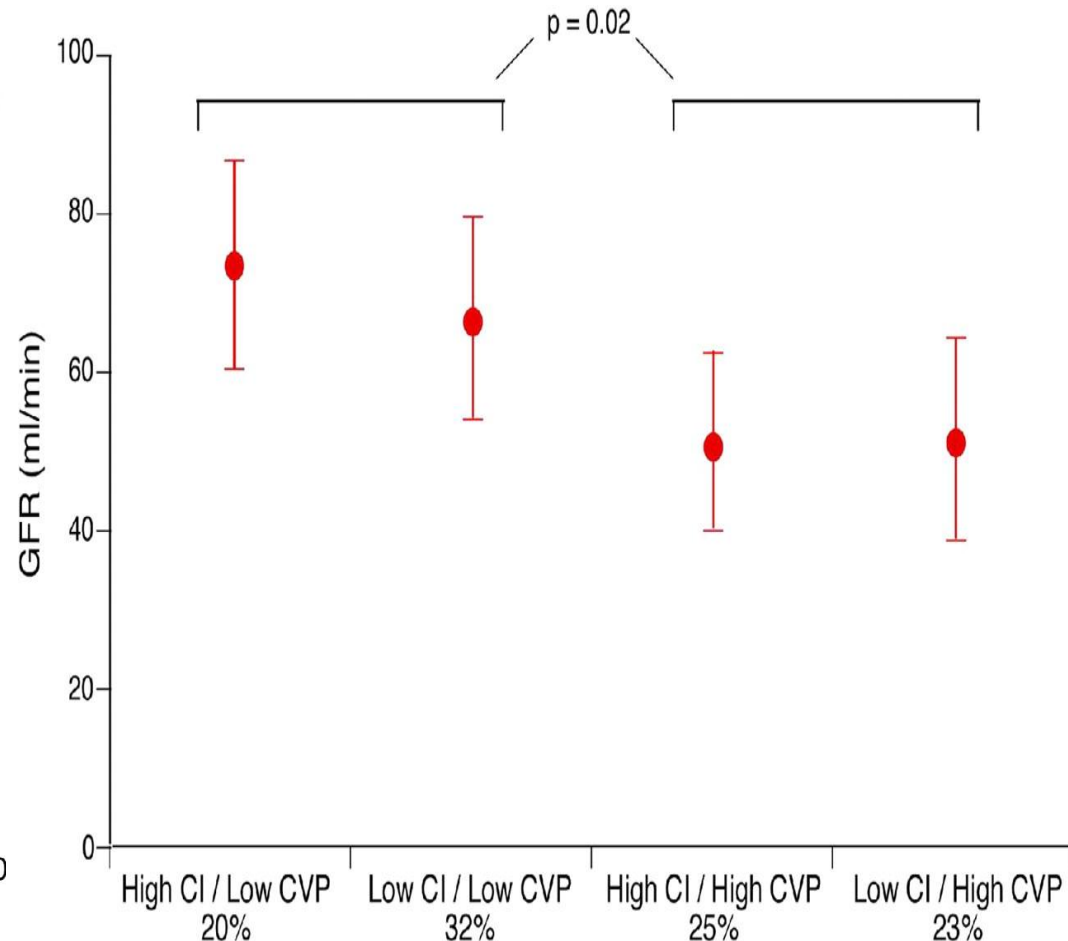


# Venous congestion as relevant hemodynamic factor underlying WRF in acute heart failure

**ROC for CVP and CI on Admission for the Development of WRF**



**Relative Contributions of CVP and CI to GFR at Time of PAC Removal**



# Clinical characteristics of pts with liver dysfunction

	Alk Phosphatase			Transaminases		
	normal	abnormal		normal	abnormal	
N	944	261 (22%)		740	427 (37%)	
<i>Clinical signs at baseline</i>						
SBP (mmHg)	117	114	0.013	117	114	0.012
DBP (mmHg)	71	69	0.073	70	71	NS
HR (bpm)	83	83	NS	81	87	<0.001
Peripheral edema (%)	65.8	79.3	<0.001	70.0	63.7	0.023
Ascites (%)	16.9	31.0	<0.001	22.0	17.1	0.049
Cold extremities (%)	20.8	26.1	0.076	19.6	25.5	0.022
<i>Biological parameters at baseline</i>						
BNP (pg/mL)	1465.1	2250.9	<0.001	1464	1918	<0.001
<i>Initial hospitalization characteristics (%)</i>						
Acute MI	19.0	10.7	0.002	11.1	30.1	<0.001
LVEF	24.0	23.3	0.071	24.1	23.5	0.048
Tricuspid regurgitation	45.8	52.9	0.04	51.6	40.8	<0.001
<i>All-cause mortality (%)</i>						
at 31 d	11.1	14.6	NS	8.4	17.6	<0.001
at 180 d	23.5	34.9	0.001	22.4	31.6	<0.001

# Cardio-hepatic syndromes

- *Abnormal transaminases:*
  - systemic and peripheral **hypoperfusion**
  - « Forward » HF
- *Abnormal Alkaline Phosphatase:*
  - clinical and biological signs of marked systemic - mostly right-sided- **congestion**
  - Varies with BNP changes
  - « backward » HF, marker of « congested liver ».

# Markers of RV Dysfunction Associated With Clinical Status and Prognosis

Right ventricular ejection fraction (echocardiography, radionuclide angiography or thermodilution) <sup>28,29,31,33,35-38,77</sup>

Right ventricular ejection fraction response to pulmonary vasodilation <sup>65</sup>

Right ventricular dilation <sup>78</sup>

Degree of right ventricular dilation compared with left ventricular dilation <sup>32</sup>

Tricuspid annular velocity (systolic and/or diastolic) or excursion, or echo right ventricular descent (shortening) <sup>30,34,79-81</sup>

Right ventricular index of myocardial performance <sup>80,82,83</sup>

Doppler-estimated dP/dt <sup>84</sup>

Tricuspid regurgitation <sup>85-87</sup>

Doppler echo-derived right ventricular tissue displacement and strain <sup>88</sup>

Right atrial size <sup>85,89</sup>

Radionuclide angiographic, invasive angiographic, or echo/catheterization pressure-volume or pressure-area loops <sup>90-92</sup>

Brain natriuretic peptide level <sup>15-19</sup>

Heart rate variability <sup>93</sup>

## How can I detect a RVF in acute settings: *Biology*?

- Plasma lactate seems much higher than expected by systemic hemodynamic
- Elevated liver enzymes
- Decrease in PT
- Oliguria

## **How can I detect a RVF in acute settings: *Biology*?**

- Plasma lactate seems much higher than expected by systemic hemodynamic
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## **How to diagnose RVF in the context of advanced LVF?**

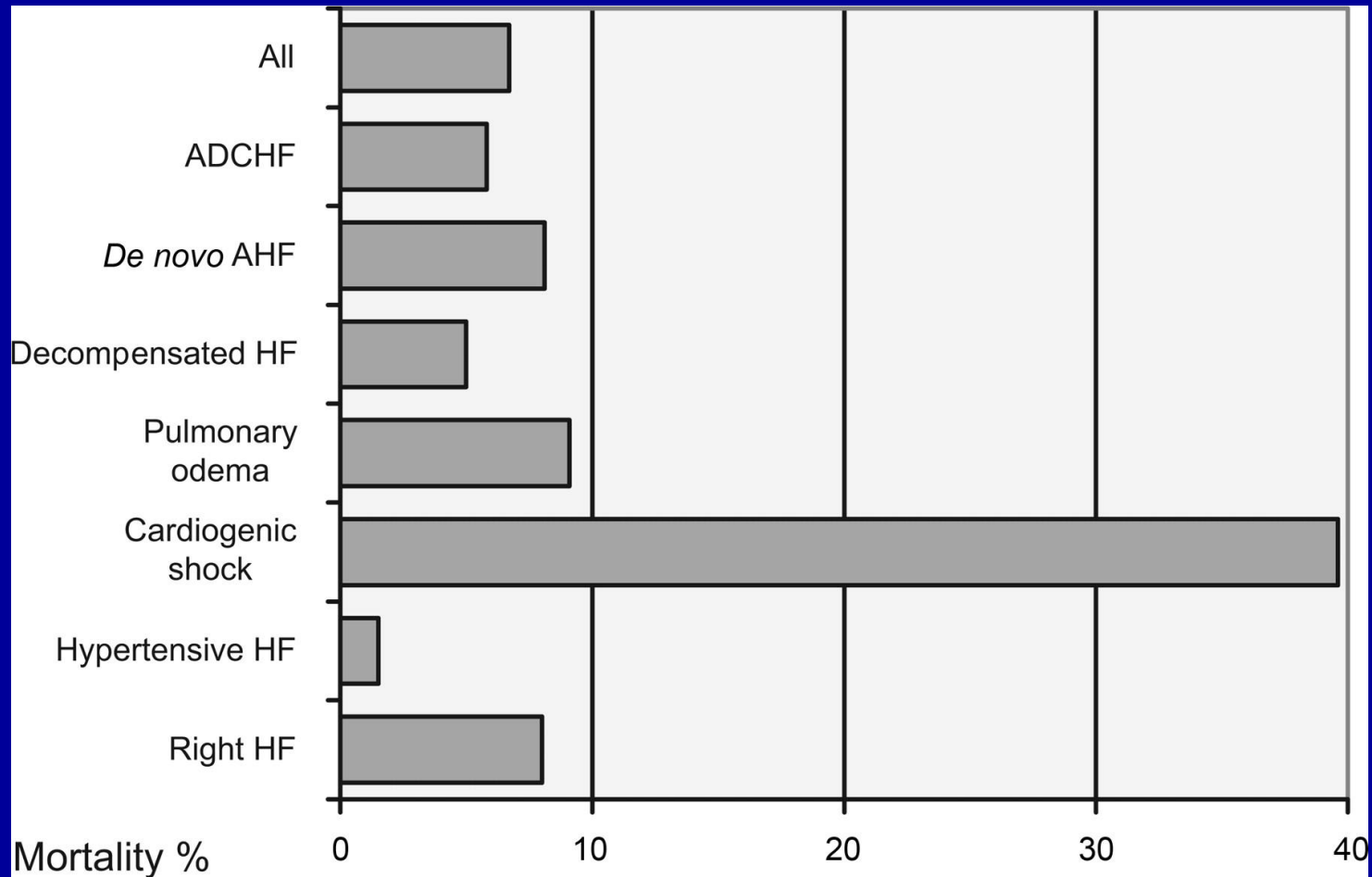
- Inability to reduce congestion
- Persistent organ dysfunction
  - Renal failure
  - Liver failure



# Factors Relating to Survival in Chronic Congestive Cardiomyopathy

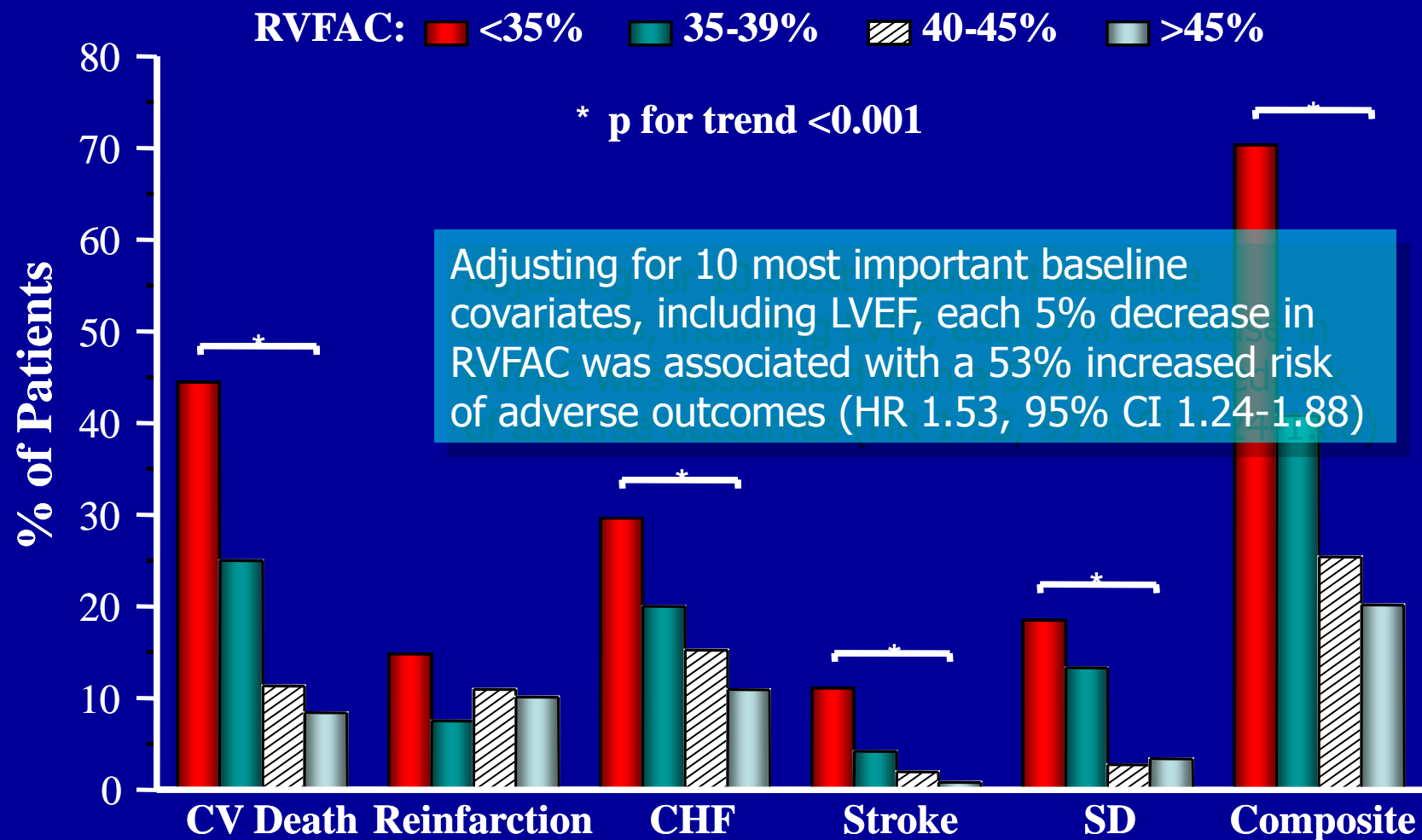
Factors	Survivors	Non-Survivors	<i>p-value</i>
LVEF	18.7±6.8 %	18.1±10.3 %	NS
<b>RVEF</b>	<b>41.4±23.0 %</b>	<b>23.9±10.2 %</b>	<b>&lt; 0.01</b>
LV dyskinesia	20.9±11.8 %	29.7±15.0 %	< 0.05
Ventricular arrhythmia	50%	44%	NS
Functional class (NYHA)	3.0 ± 0.7	3.6 ± 0.5	< 0.01
Diabetes	19 %	22 %	NS
Hypertension	19 %	33 %	NS
Previous inferior infarction	17 %	42 %	NS
COPD	6 %	33 %	NS

# In-hospital mortality in EHFS II by history of HF and clinical class

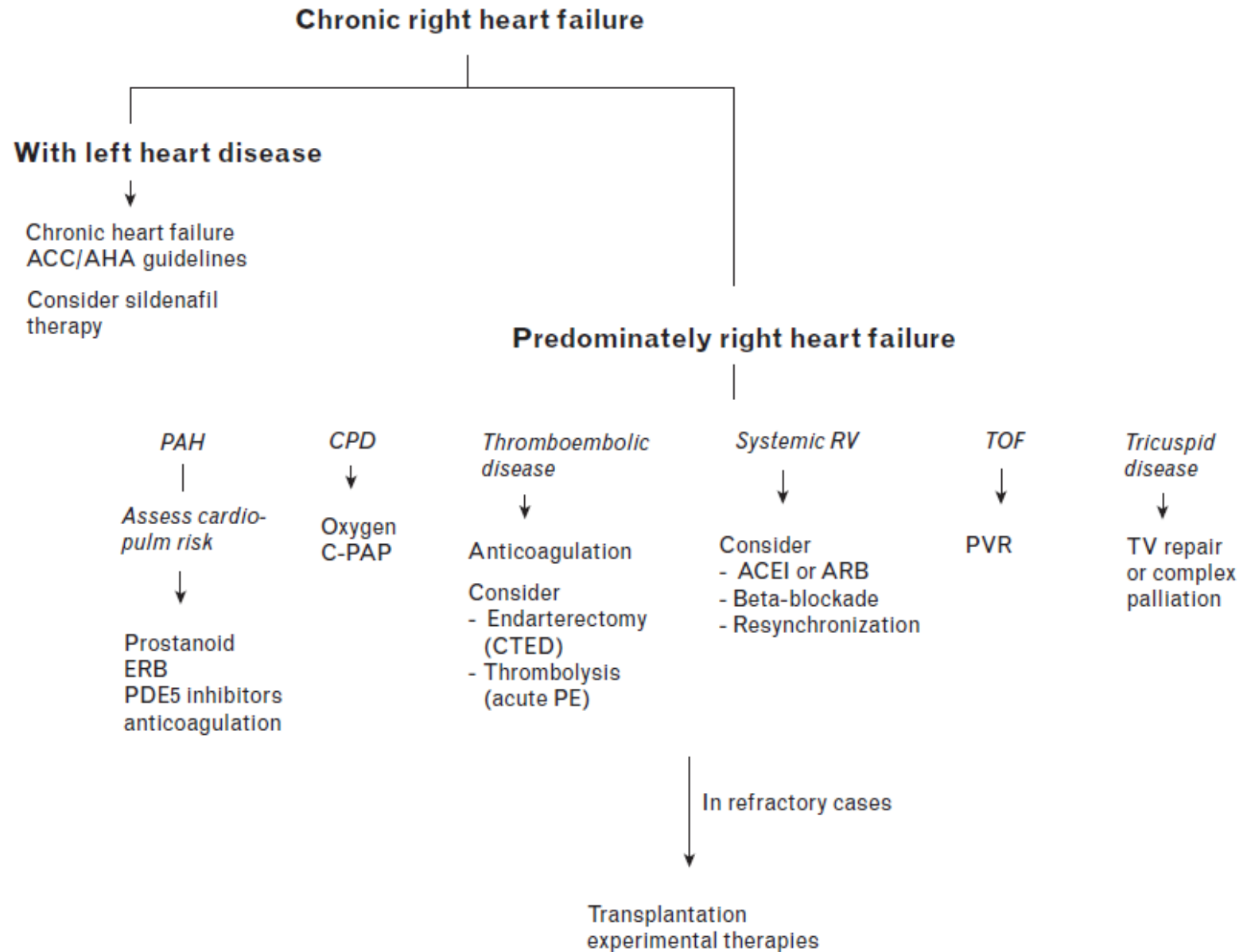


Niemenen, M. S. et al. EHJ 2006 27:2725

# RV Function and CV Events



# RV dysfunction/ failure: treatment



# Treatment of LVF versus RVF

## LV failure

- Often decrease BP
- NIV often needed
- Volume loading might be needed

## RV failure

- Often increase Blood Pressure (Right Coronary Artery!)
- Caution if volume loading: it might aggravate RVF

# Treatment of RVF

- **What I should do**

- Decrease RV afterload: to decrease  $MVO_2$
- Decrease volemia: to decrease RVV and TR
- Improve contractility
- Increase MAP: to improve RC and organ PP

- **What I should avoid**

- Mechanical ventilation as much as possible
- Volume loading

# Right Heart: Cinderella of HF

## RV dysfunction / failure

- Syndrome of congestion
- High Morbidity and Mortality
- Heterogenous aetiology
- Not EBM therapy
- Much less studied than the Left !

