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# **Αντιμετώπιση του αιμοδυναμικά ασταθή ασθενή με Πνευμονική Εμβολή**

**Κονδύλη Ευμορφία ΜΕΘ ΠΑΓΝΗ  
Ιατρική σχολή Πανεπιστημίου Κρήτης**

# Massive Pulmonary Embolism

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## Acute PE with sustained hypotention

- SAP < 90 mmHg for at least 15 min
- **or** requiring inotropes , not due to to a cause other than PE, such as arrhythmia, hypovolemia, sepsis, or left ventricular [LV] dysfunction
- **Pulselessness, or persistent profound bradycardia (heart rate 40 bpm with signs or symptoms of shock.**

Jaff at all

## Definition of Submassive PE

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*Acute PE without systemic hypotension (SBP > 90 mm Hg) but with either*  
***RV dysfunction***  
***or Myocardial necrosis'***

***RV dysfunction means the presence of at least 1 of the following:***

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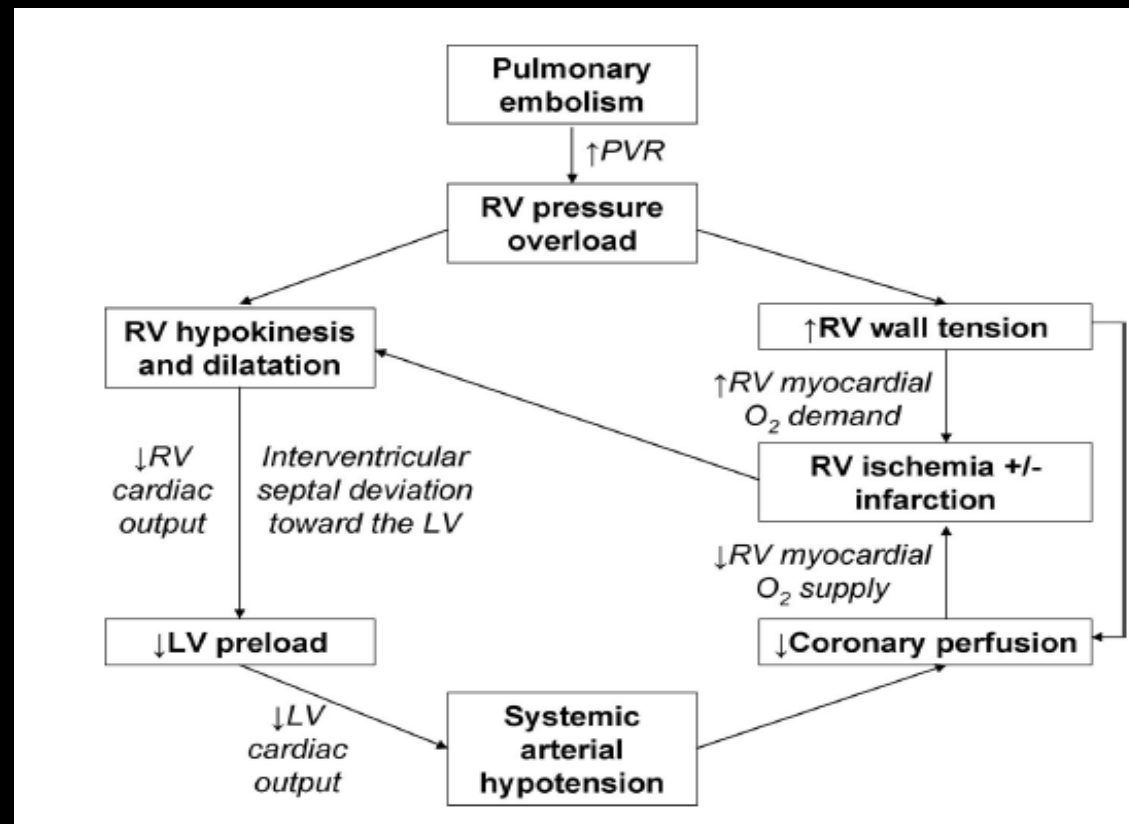
- 1. RV dilation (RV/LV >0.9) or RV systolic dysfunction on echocardiography**
- 1. RV dilation on CT (RV/LV >0.9)**
- 2. Elevation of BNP (90 pg/mL)**
- 3. Elevation of N-terminal pro-BNP (500 pg/mL);**
- 4. or ECG changes**

# ***Myocardial necrosis is defined as:***

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1. Elevation of troponin I (0.4 ng/mL) or
2. Elevation of troponin T (0.1 ng/mL)

# Pathophysiology OF RV dysfunction in massive /submassive PE

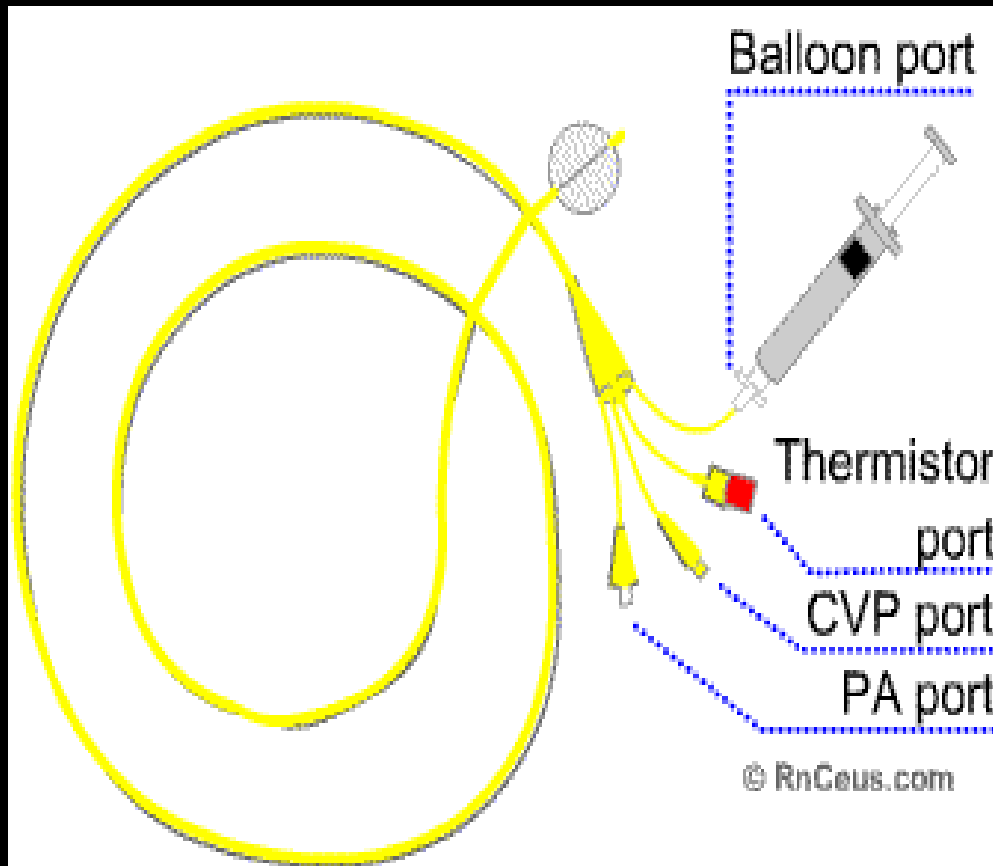


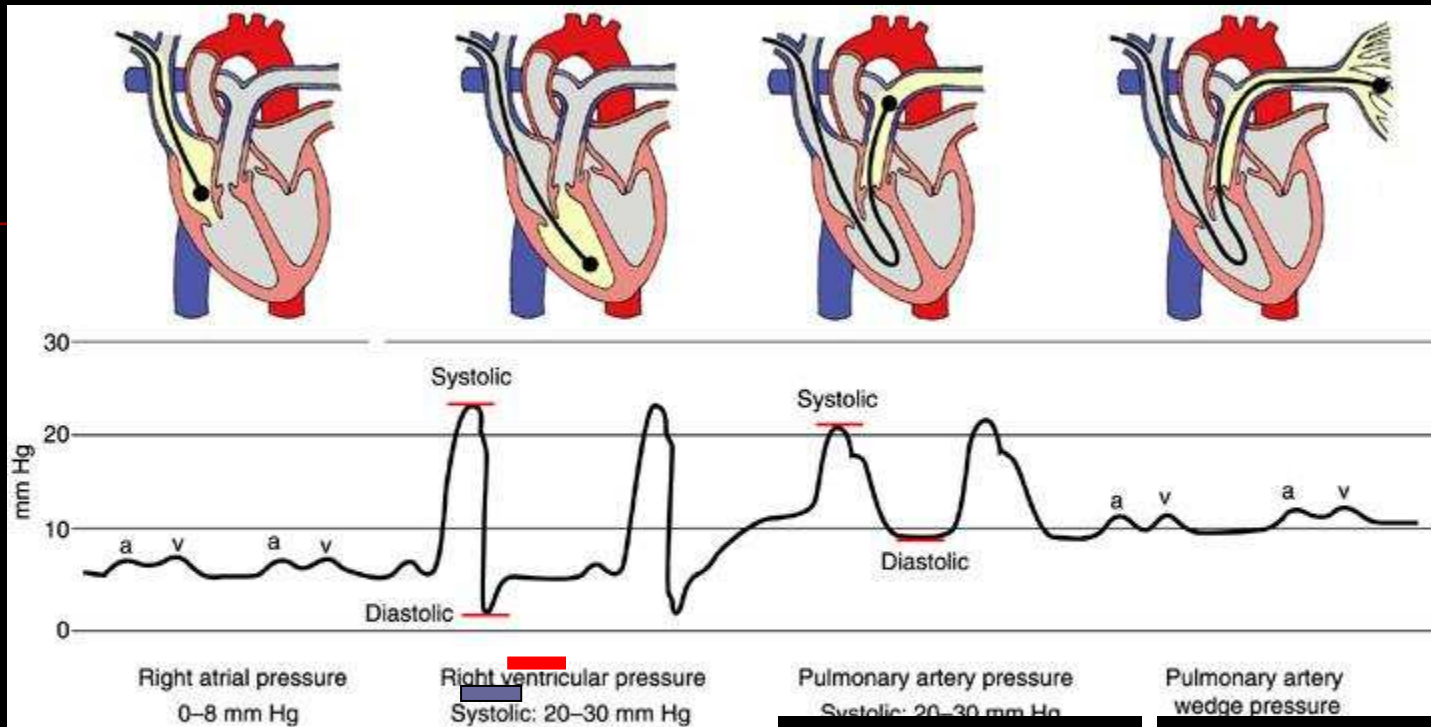
# RVD- EVALUATION

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- Pulmonary arterial catheterization
- Echocardiography

# Pulmonary arterial catheterization





Πίεση  
Δεξιού Κόλπου

0-5 mmHg

Πίεση Δεξιά  
κοιλίας

Συστολική 20-  
30 mmHg  
Διαστολική 0-8  
mmHg

Πίεση  
πνευμονικής  
αρτηρίας

Συστολική 20-  
30 mmHg  
Διαστολική 8-  
15 mmHg

Πίεση  
ενσφήνωσης  
πνευμονικών  
τριχοειδών  
8-12 mmHg

# Hemodynamic parameters

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- **PVR= mean PAP-Pwedge / CO**

( Wood units =  $1\text{mmHg}\cdot\text{min}\cdot\text{L}^{-1}$  )

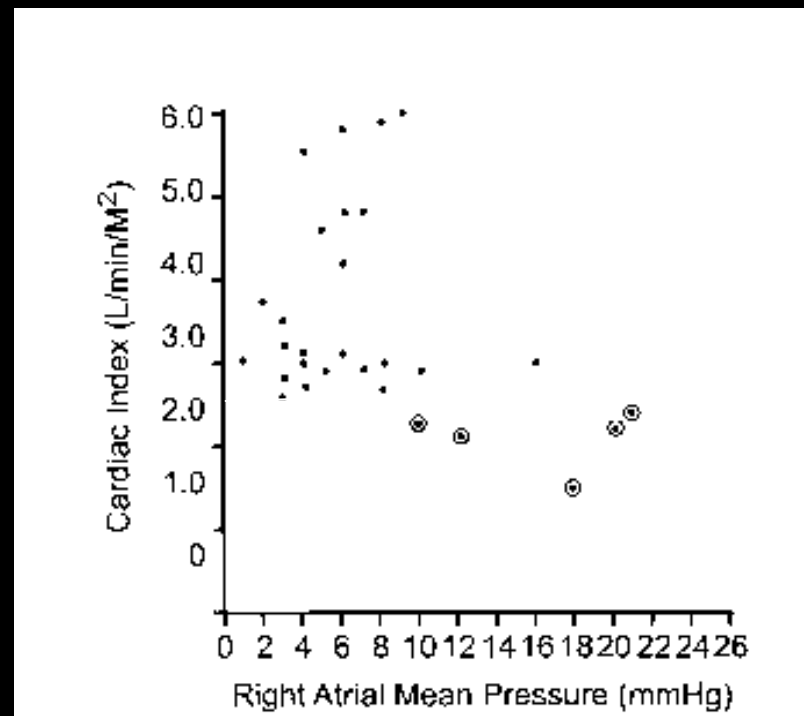
- **Cardiac Output**

(Thermodilution      Fick Method)

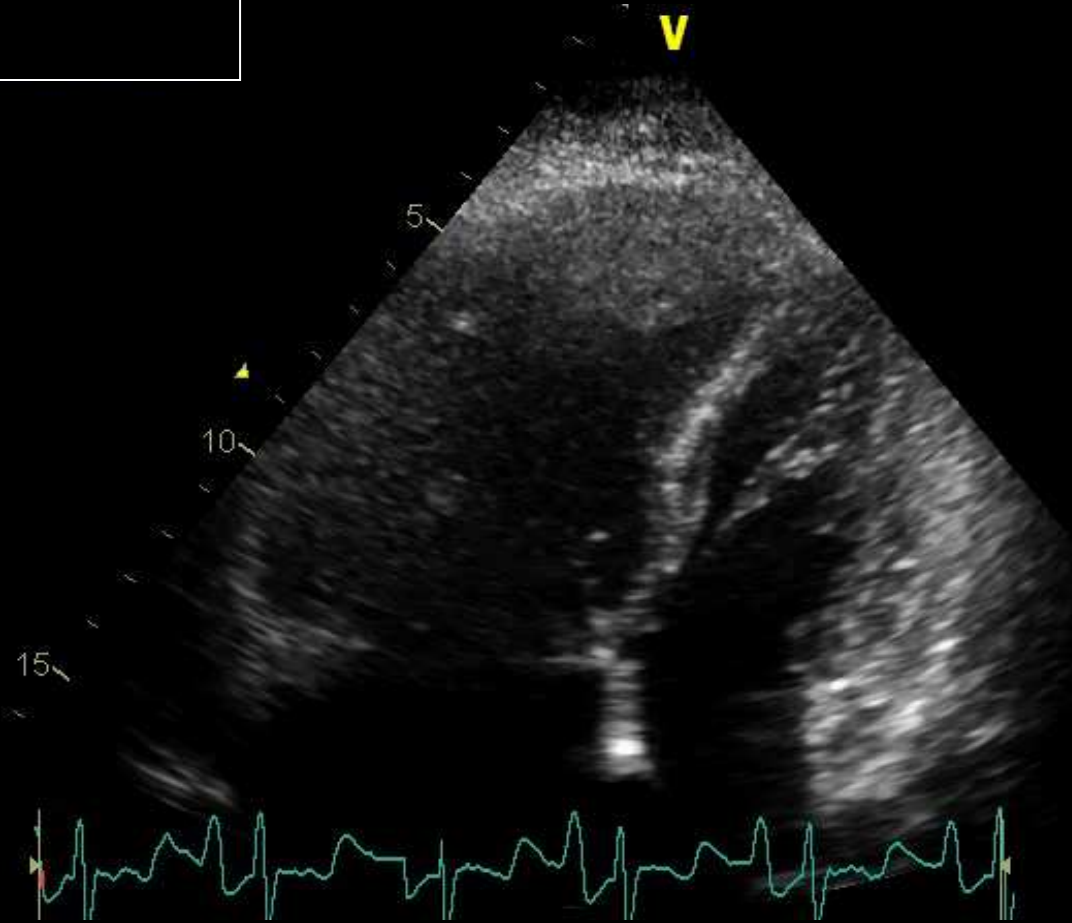
**SvO<sub>2</sub>**

# Pulmonary arterial catheterization

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# Echocardiography



2:130 90 HR

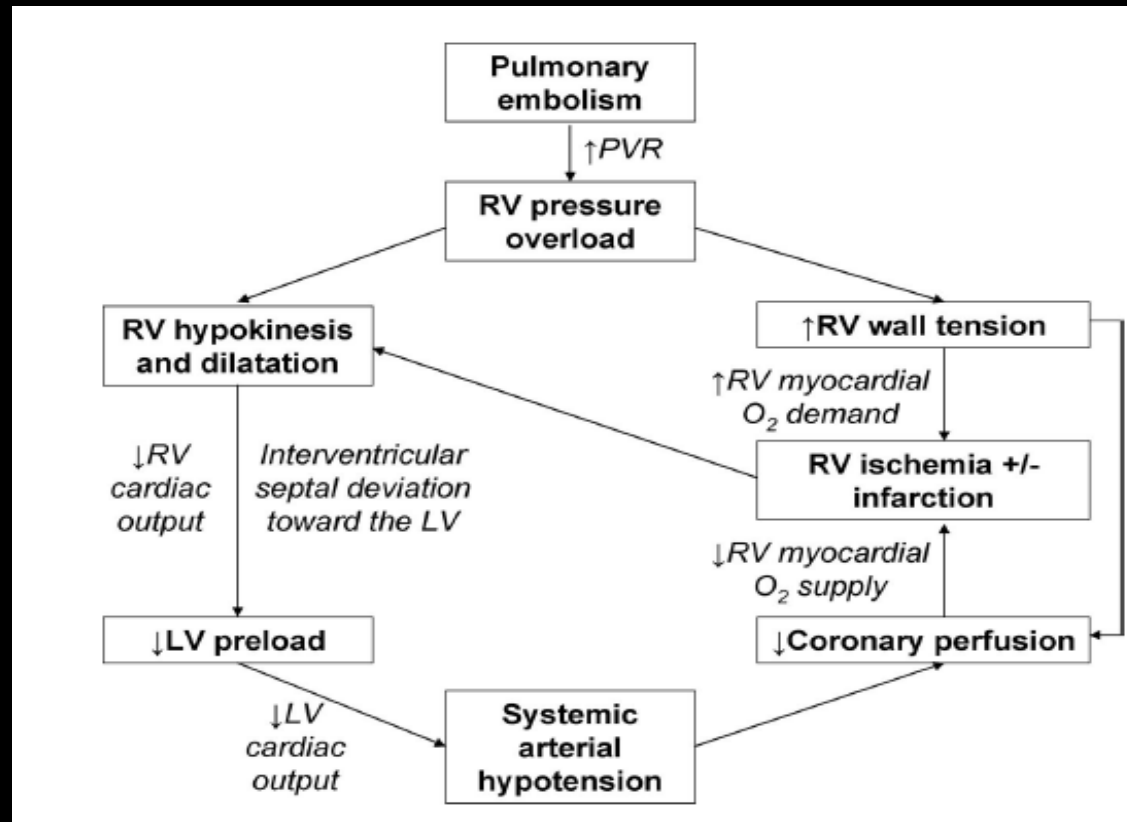
# Management of acute RVD

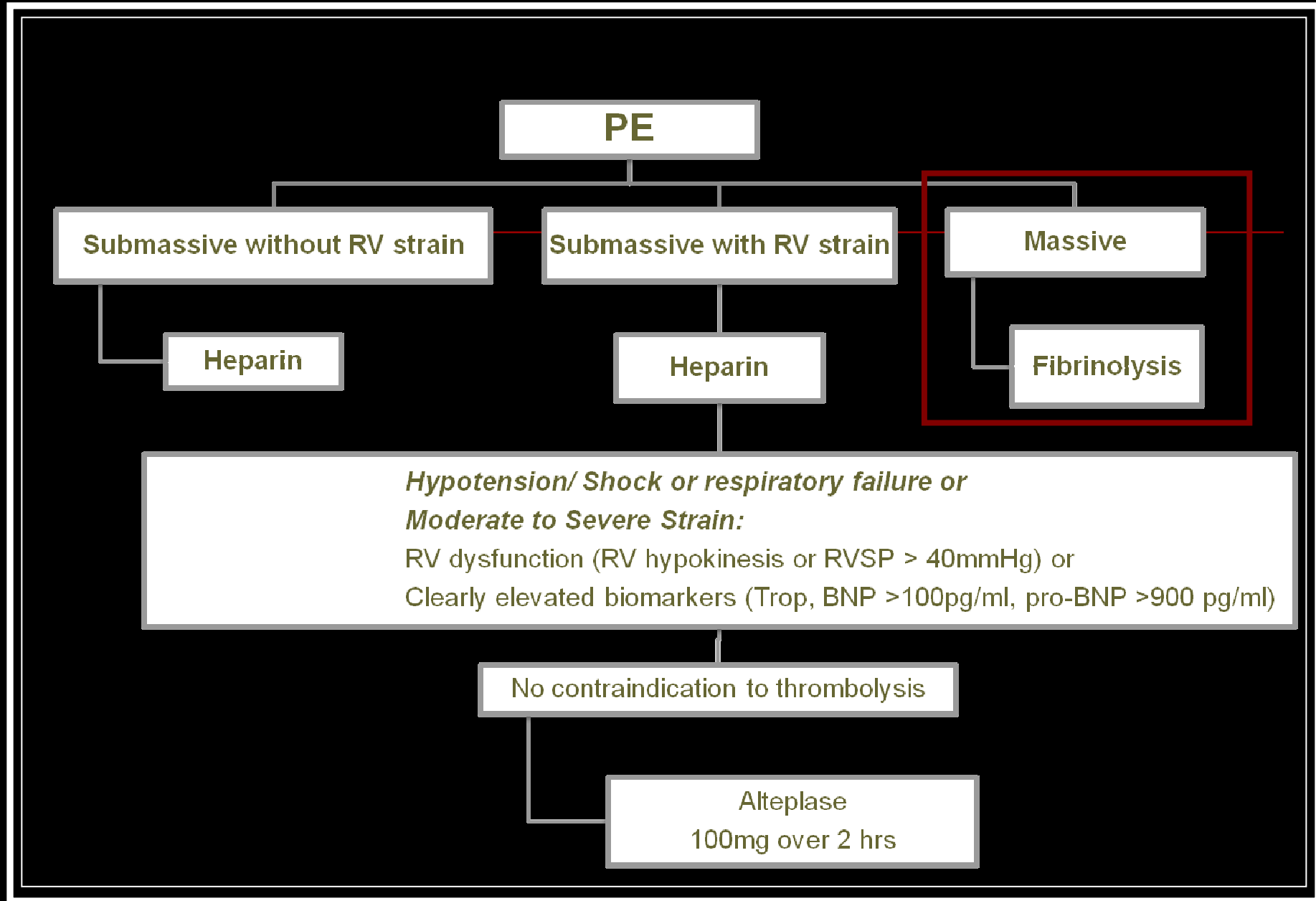
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The main goal

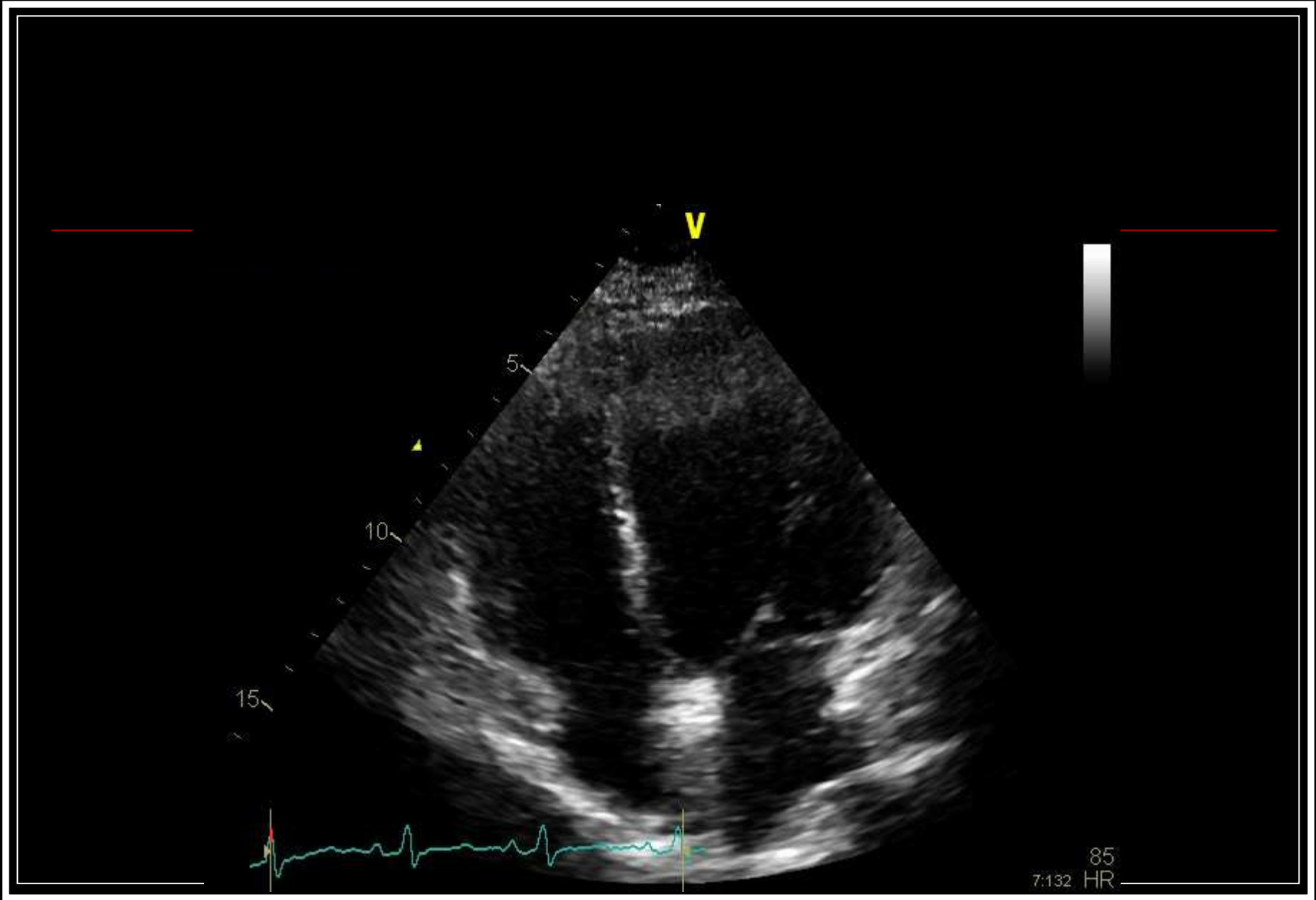
Breaking the vicious cycle of reduced CO by **restoring** adequate oxygen delivery to the myocardium and **reducing RV overload**

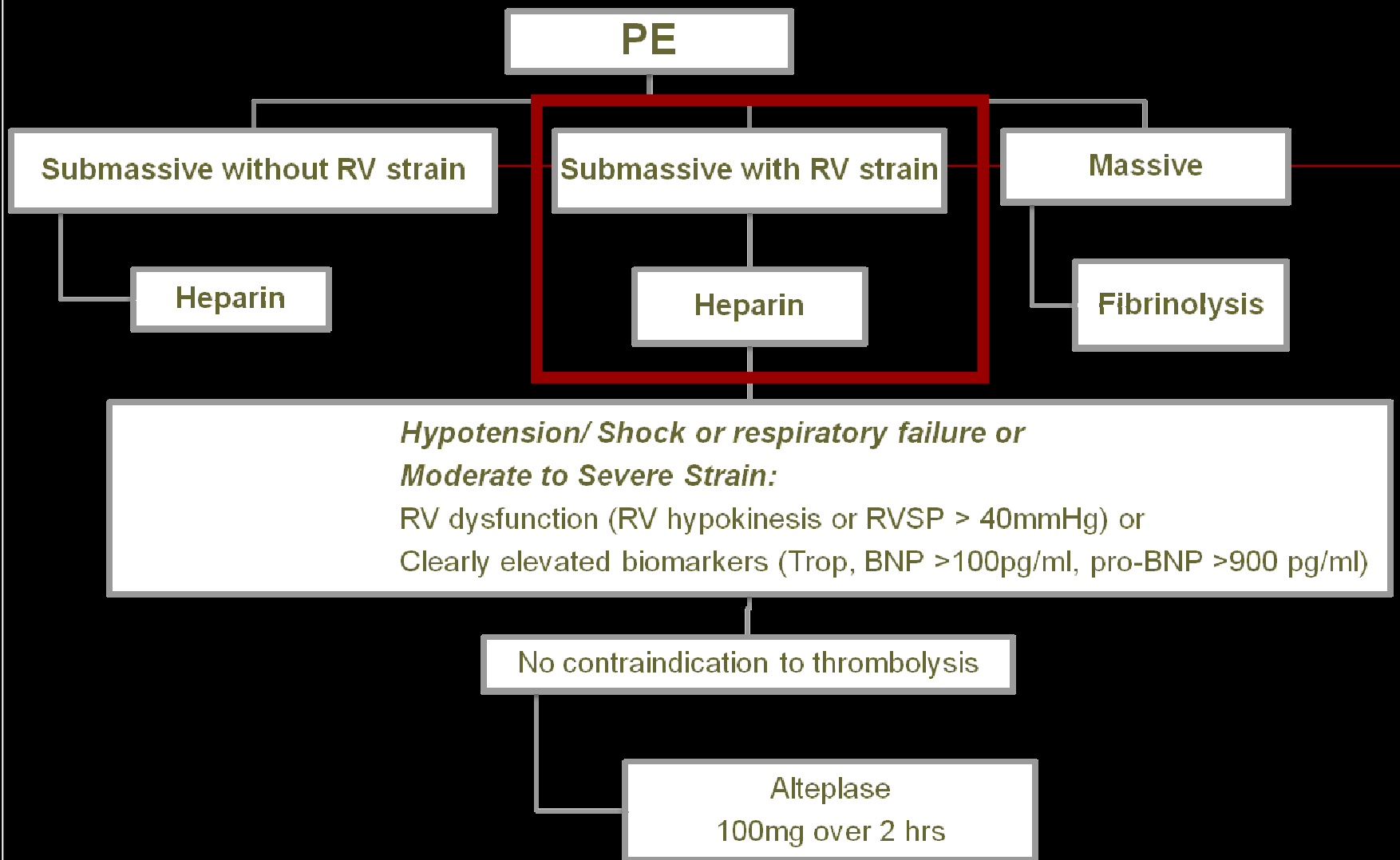
# Pathophysiology OF RV dysfunction in massive /submassive PE



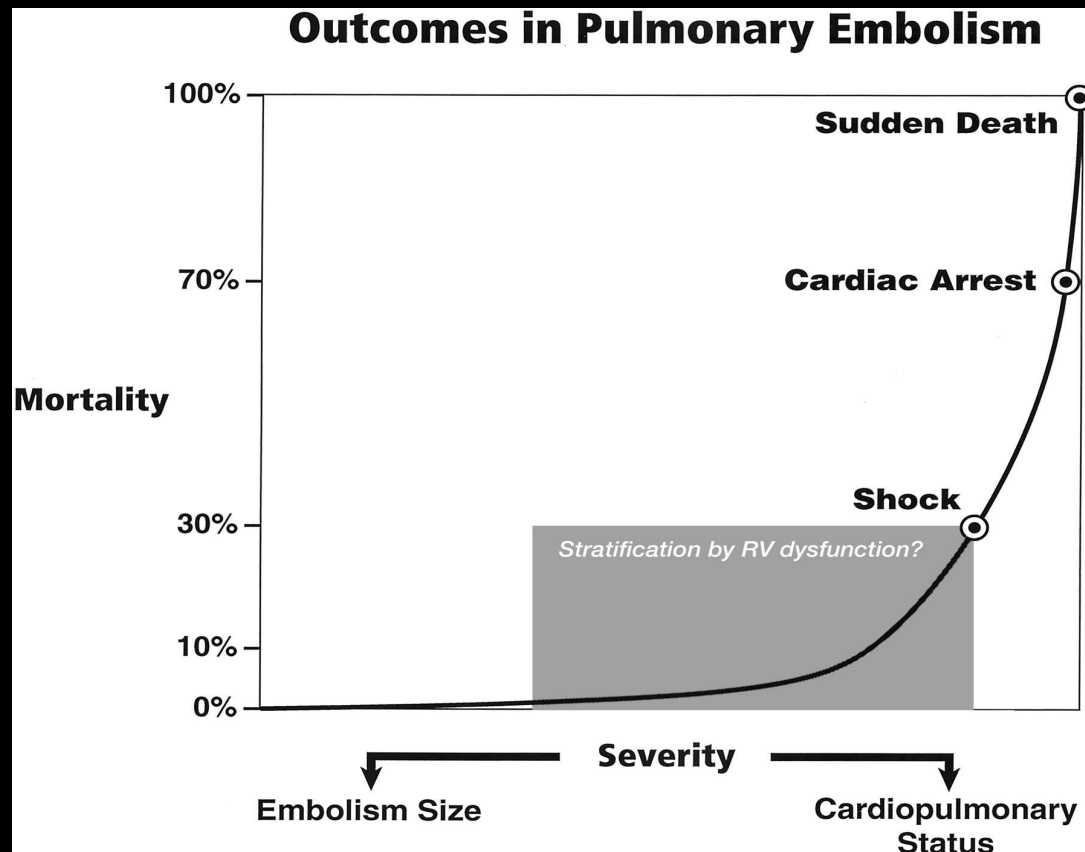


Modified from AHA scientific statement. Circulation 2011; 123

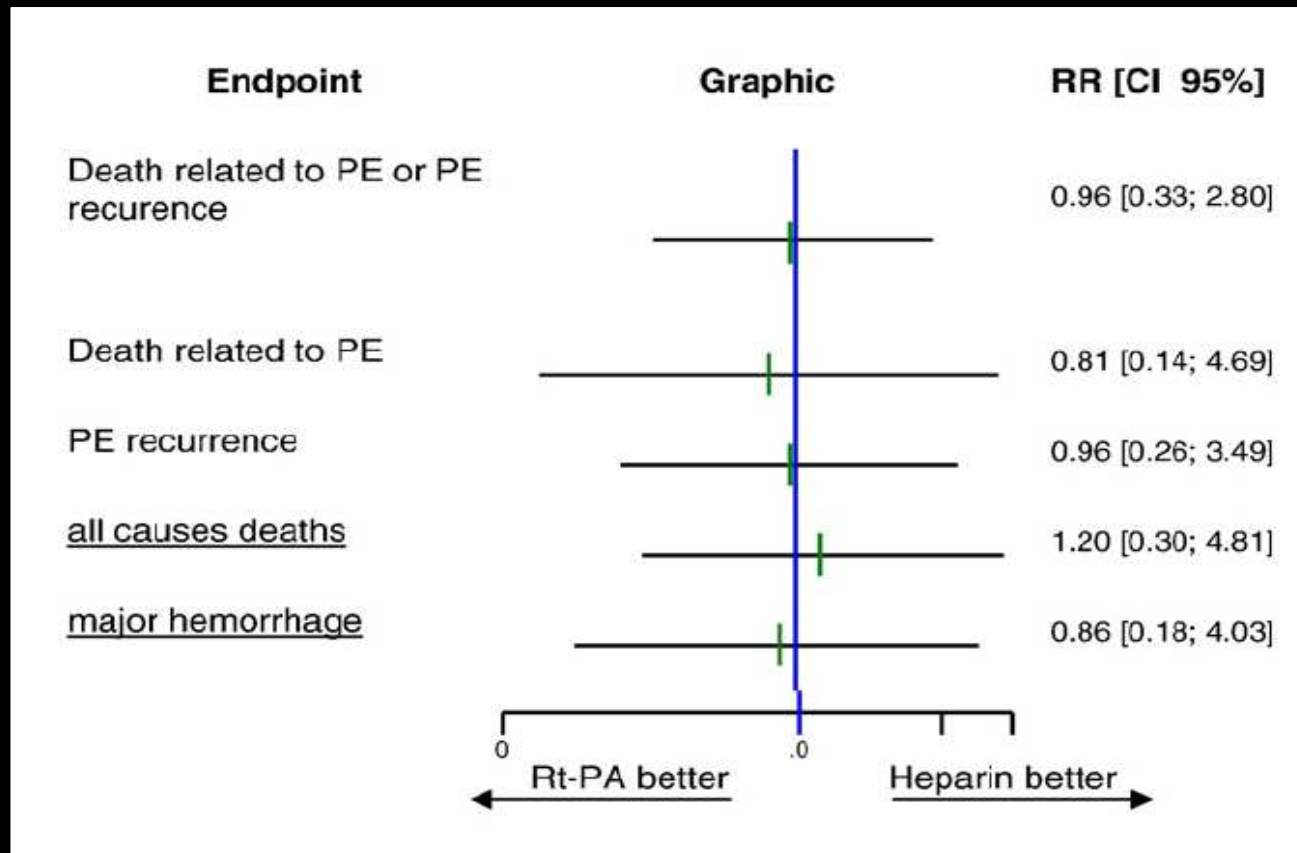




# Anticoagulation - Thrombolysis



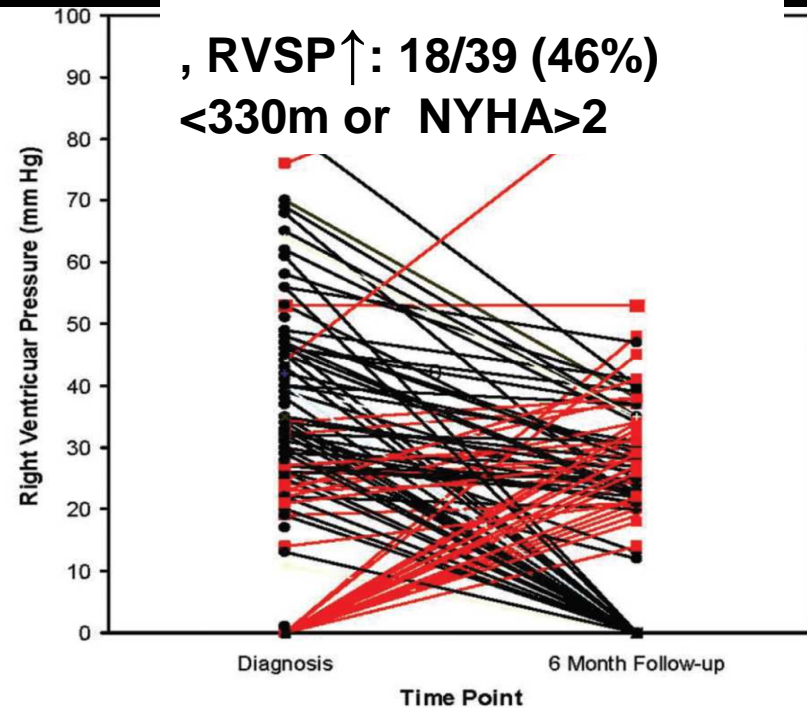
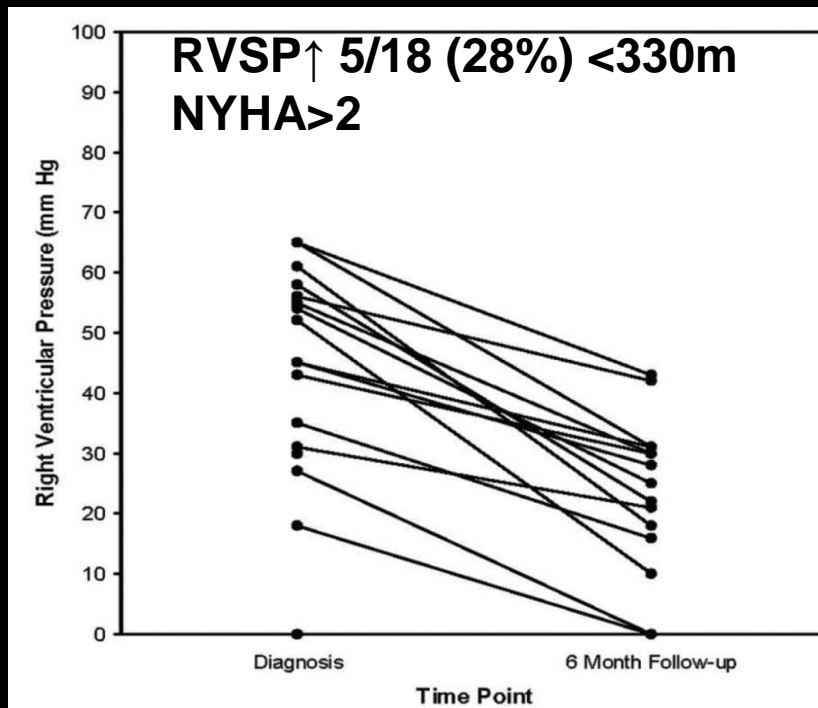
# Results for pts with RV dysfunction: 2 studies, 357 pts



# RVSP at baseline and 6 months later

Group with heparin + alteplase

Group with heparin and placebo



*Kline et al. Chest 2009; 136:1202-1210*

# Volume management

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- Volume overloading may further  
**Dilate RV**  
**Increase Tricuspid regurgitation**

A sharp increase in left or to right filling pressures may indicate when **further loading is detrimental**

# Positive Inotropic agents

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- **Dobutamine – Phosphodiesterase inhibitors**

Increase contractility **but** may increase oxygen consumption of the myocardium

- **Levosimendan**

# Vasopressors

TABLE 1. Effect of Volume and Norepinephrine on Right Ventricular

	CO (l · min <sup>-1</sup> )	SV (ml)	RVEDP (mmHg)	RVSP (mmHg)	PAP (mmHg)	PVR (mmHg · l <sup>-1</sup> · min)
Baseline (cond. A)	2.8 ± 1.6	17.4 ± 4.4	0.17 ± .4	19.4 ± 2.4	11.0 ± 2.3	1.9 ± 1
Volume (cond. B)	3.4 ± 0.6	23.5 ± 6.5*	1.3 ± .93	21.2 ± 1.6	12.3 ± 1.8	1.2 ± .1
Baseline (cond. C)	3.0 ± 1.5	18.7 ± 8.3	0.42 ± 2.5	18.7 ± 8.3	13.8 ± 4.4	1.7 ± 2.3
PVR (cond. D)	1.4 ± 0.2†	9.1 ± 1.3†	9.4 ± 4.8†	71.3 ± 6.8†	55.4 ± 5.8†	37.0 ± 5.9†
Volume (cond. E)	0.94 ± .4	6.3 ± 2.3†	11.9 ± 4.7†	52.1 ± 18.7†	42.3 ± 16.0†	36.7 ± 5.9
Norepinephrine (cond. F)	1.9 ± .2*	11.2 ± 2.1†	4.7 ± 4.3†	81.8 ± 15.5†	60.2 ± 7.1†	29.7 ± 4.1

# Conclusions

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- Massive PE :Heparine +Fibrinolysis
- Submassive PE : Heparine , Consider Fibrinolysis in High Risk Patients
- Avoid Volume overload
- Vasopressors