

# Management of the High-Risk Pulmonary Embolism in the Acute Phase—Respiratory, Hemodynamic and Mechanical Support



**Dr. Anoush Dehnadi**

MD., FCCM

**@DRDEHNADI\_ICU**

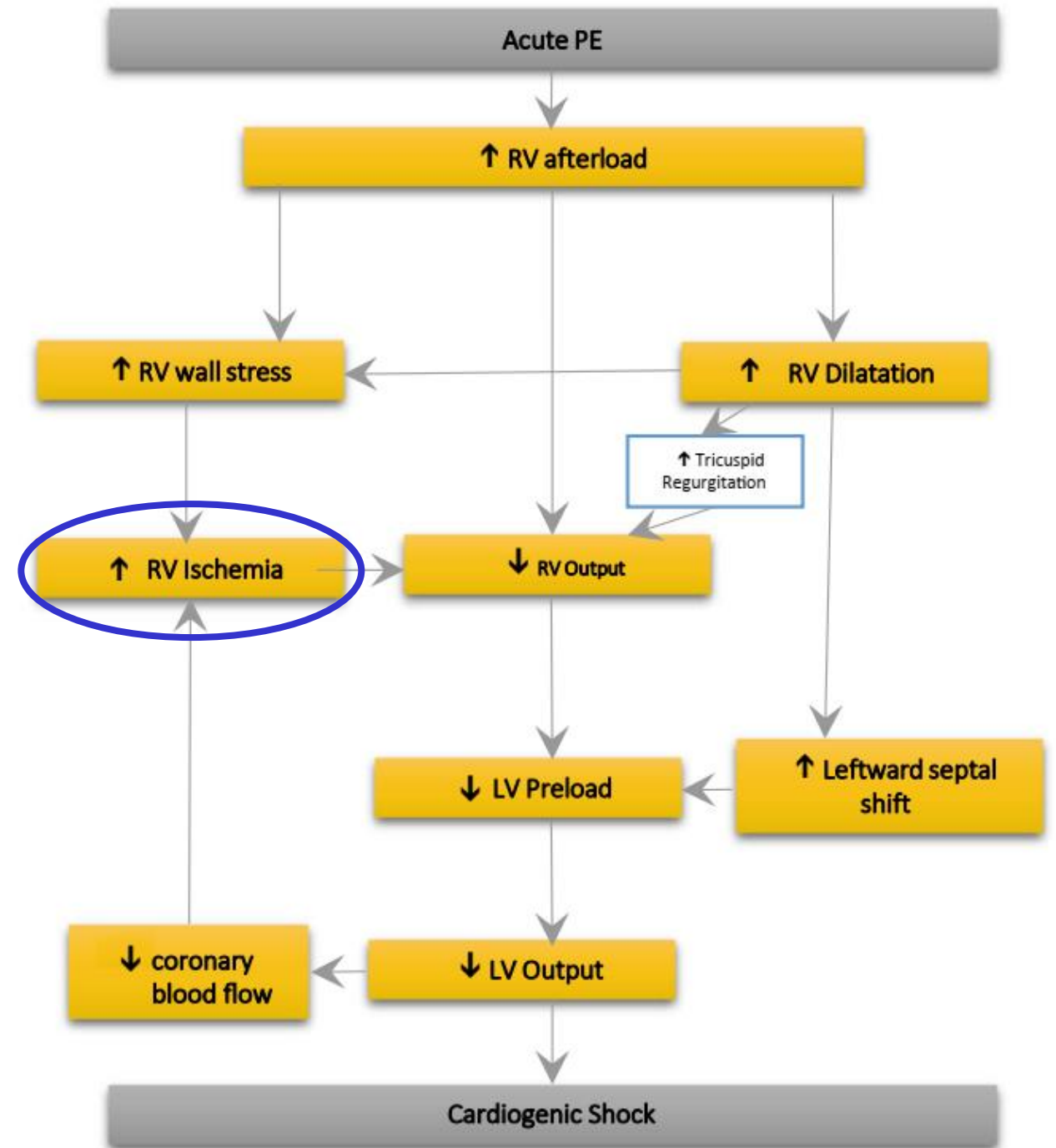
05.04.1402

- ❑ **VTE:** an incidence of 112 cases per 100,000 in USA
- ❑ VTE is responsible for nearly 1.2 million cases in the US every year, with 370,000 cases being Pulmonary Embolism (PE)
- ❑ **High-risk PE (massive PE):** **hemodynamic instability + a severe ventilation/perfusion mismatch causing hypoxemia**

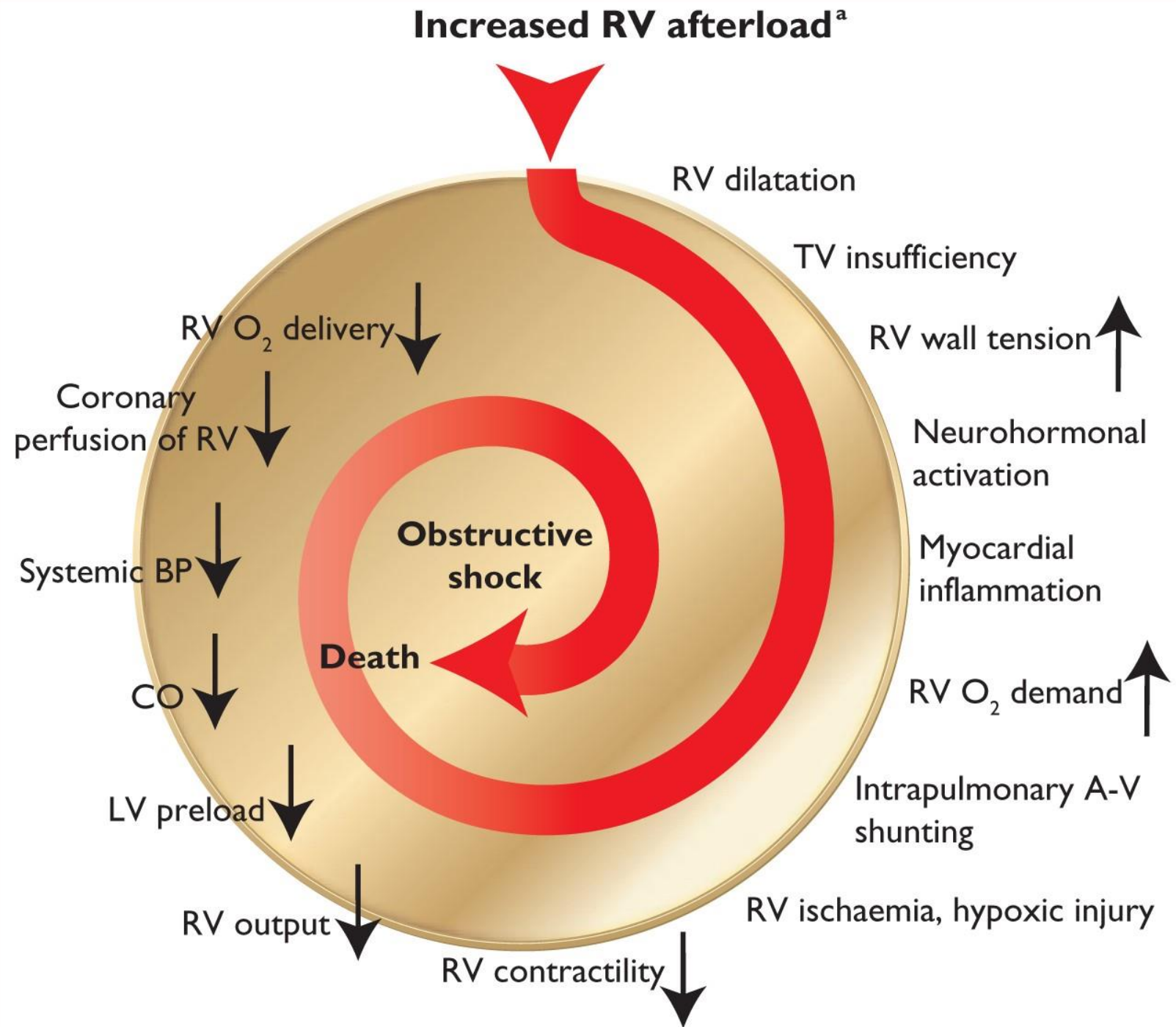
### **Hemodynamic instability:**

- **Obstructive shock:**
  - ✓ **systolic BP of  $< 90$  mmHg, or the need for vasopressors to maintain systolic BP over 90 mmHg, in face of adequate fluid loading) or**
- **persistent hypotension (Systolic BP  $< 90$  mmHg or SBP drop over 40 mmHg, lasting  $> 15$  min & not caused by new-onset arrhythmia, hypovolemia, or sepsis)**

# Pathophysiology of the hemodynamic compromise in high-risk PE



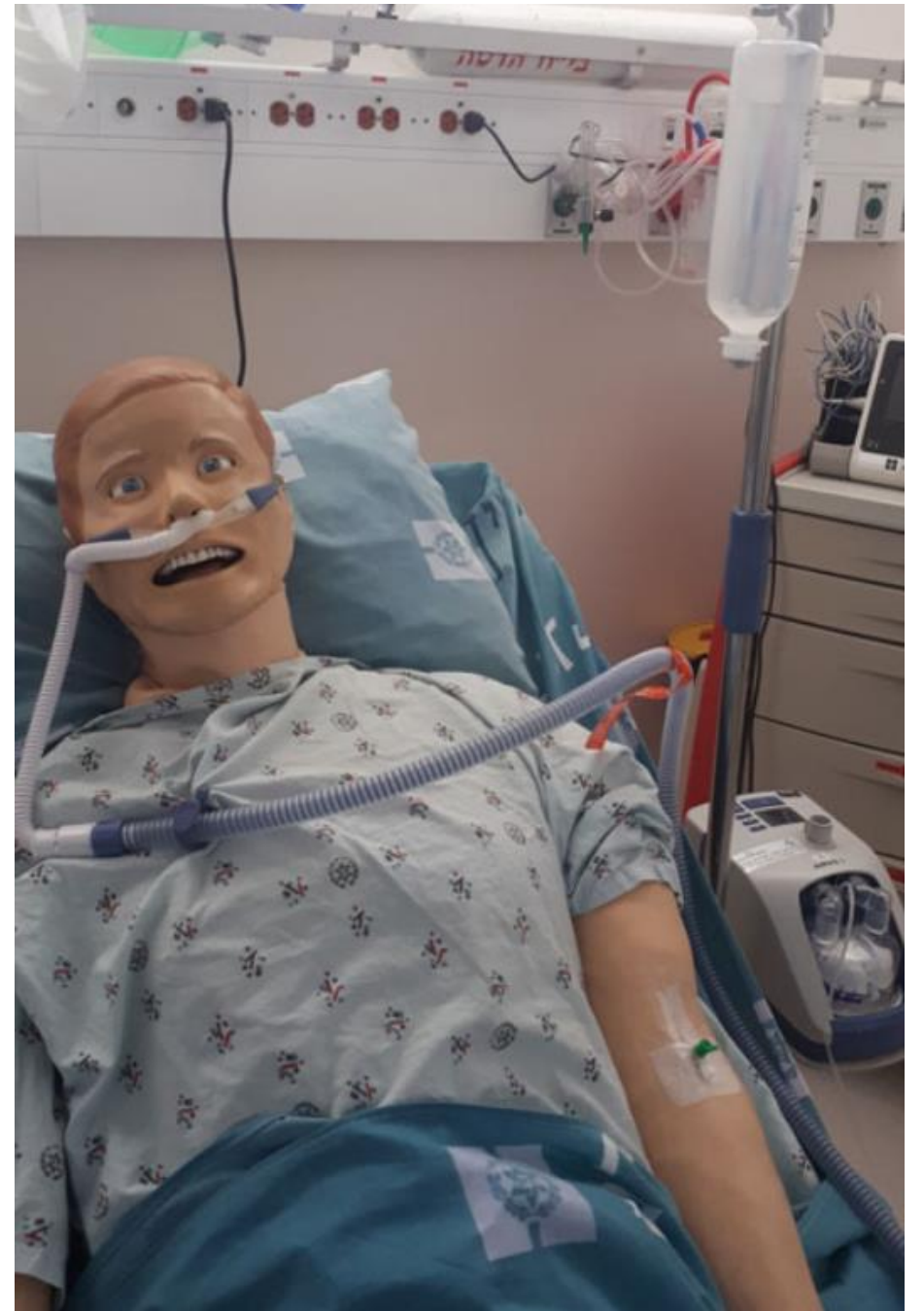
Key factors contributing to **hemodynamic collapse and death** in acute PE



# Respiratory Support

- ❑ **Aggressive management of hypoxemia and hypercapnia is critical**
- ❑ **HFNC:** high flow (up to 60 L/min) of warm and humidified air, together with a high FIO<sub>2</sub> (up to 95%), decreasing the patient's work of breathing
- ❑ **ETI + PPV ⇒ decrease in cardiac output & MAP + increase PVR**

# HFNC: high flow Nasal Canula



# Acute Hemodynamic Collapse After Induction of General Anesthesia for Emergent Pulmonary Embolectomy

Rosenberger, Peter MD<sup>\*</sup>; Shernan, Stanton K. MD<sup>\*</sup>; Shekar, Prem S. MD<sup>†</sup>; Tuli, Jayshree K. Sc<sup>‡</sup>; Weissmüller, Thomas MD<sup>§</sup>; Aranki, Sary F.<sup>†</sup>; Eltzschig, Holger K. MD, PhD<sup>§</sup>

- **19% of 52 PATIENTS** undergoing emergent pulmonary embolectomy for high-risk PE **developing refractory hemodynamic collapse after GA induction**

**preparing and draping the patient before GA induction.**

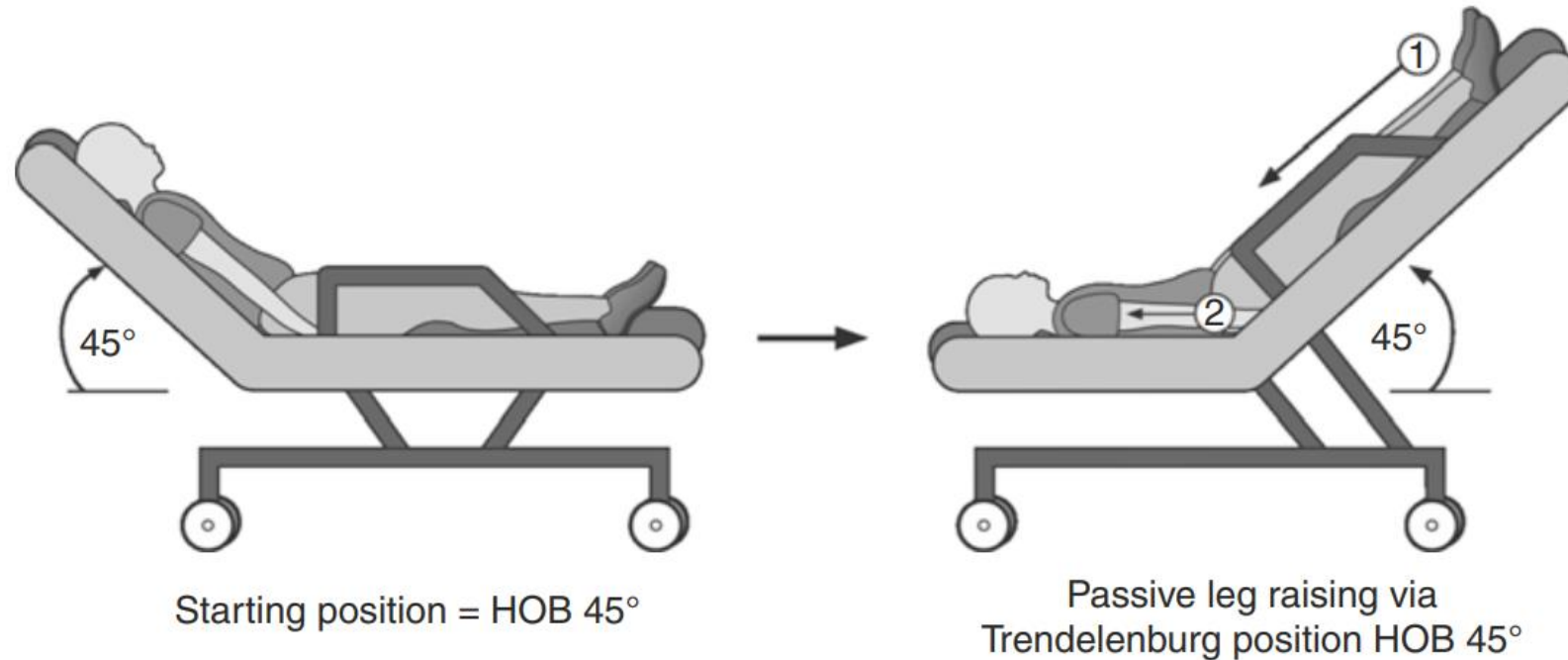
## Recommendations for Intubation in high-risk PE patients

Pitfalls	How to overcome the pitfalls
Preventing Systemic Hypotension During Induction	Choosing Etomidate or Ketamine as induction agents
	Optimizing blood pressure with vasopressors before induction
Minimizing hypoxemia during intubation	Using Apneic Oxygenation, performance of intubation by the most skilled provider
Preventing abrupt increases in PVR during positive pressure ventilation	Maintaining normal PaO <sub>2</sub> and PaCO <sub>2</sub> , using the lowest possible PEEP, using tidal volumes of 6–8 ml/kg to prevent atelectasis and overdistention

# Hemodynamic Support

- 1- Judicious fluid resuscitation,
  - 2- inotrope/vasopressor support
  - 3- Extra—Corporeal Life Support [ECMO]
- Correction of the underlying cause

# 1-Fluid Management



## Fluid responsiveness:

- ✓ cardiac output response to **Passive Leg Raise (PLR) test** or assessment of IVC diameter
- ✓ **PLR test:** a) It consists of moving a patient from the semi-recumbent position to a position where the legs are lifted at 45° and the trunk is horizontal.  
b) Monitoring of stroke volume is required as a positive passive leg raise test is defined by an increase in stroke volume of at least 10%

# Fluid Bolus

infusion of 4–6 ml/kg given over a maximum of 20 min

## Fluid Challenge

- a dynamic functional test to assess a patient's fluid responsiveness by giving a fluid bolus of at least **4 ml/kg over 5–10 min**
- Simultaneously monitoring the hemodynamic status to be able to identify fluid responsive patients

## Fluid Responsiveness

- a condition in which a patient will respond to fluid administration by **a significant increase in stroke volume[CO]**  $\geq 15\%$  for the fluid challenge, **10% for the passive leg raising test**

## 2-Vasopressor and Inotrope support

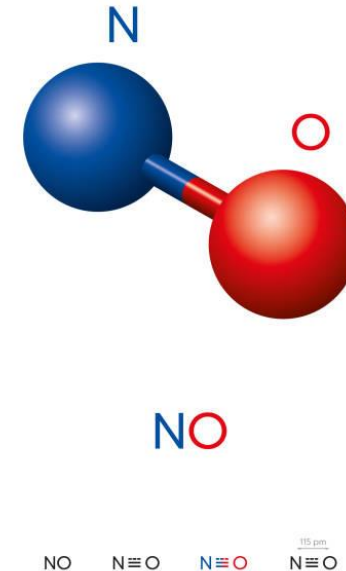
- ❑ The purpose of vasopressors in high-risk PE is to increase SVR and MAP and prevent RV ischemia.
- ❑ Noradrenaline is the main vasopressor recommended for use in patients with high-risk PE [**a net increase in SVR/PVR ratio**]
- ❑ Vasopressin is also potentially beneficial[the absence of inotropic effect]
- ❑ Inotropes: secondary to the use of vasopressors, after blood pressure had been stabilized
- ❑ Dobutamine: main inotrope, improve right ventricular—pulmonary artery coupling and to decrease PVR, improve CO
- ❑ Catecholamines: worsening tachycardia ,atrial fibrillation, worsen RV ischemia

# Pulmonary Vasodilators

❑ Intravenous Epoprostenol had failed to demonstrate a significant effect in PE

## ❑ Inhaled nitric oxide (iNO):

- ✓ colorless gas with a sharp odor
- ✓ arterial relaxation [cGMP ↑],
- ✓ correction of V/Q mismatch,
- ✓ anti-platelet aggregation activity



# Management of Right Ventricular Failure in Pulmonary Embolism

Steven Zhao, MD<sup>a</sup>, Oren Friedman, MD<sup>b,\*</sup>

Crit Care Clin 36 (2020) 505–515

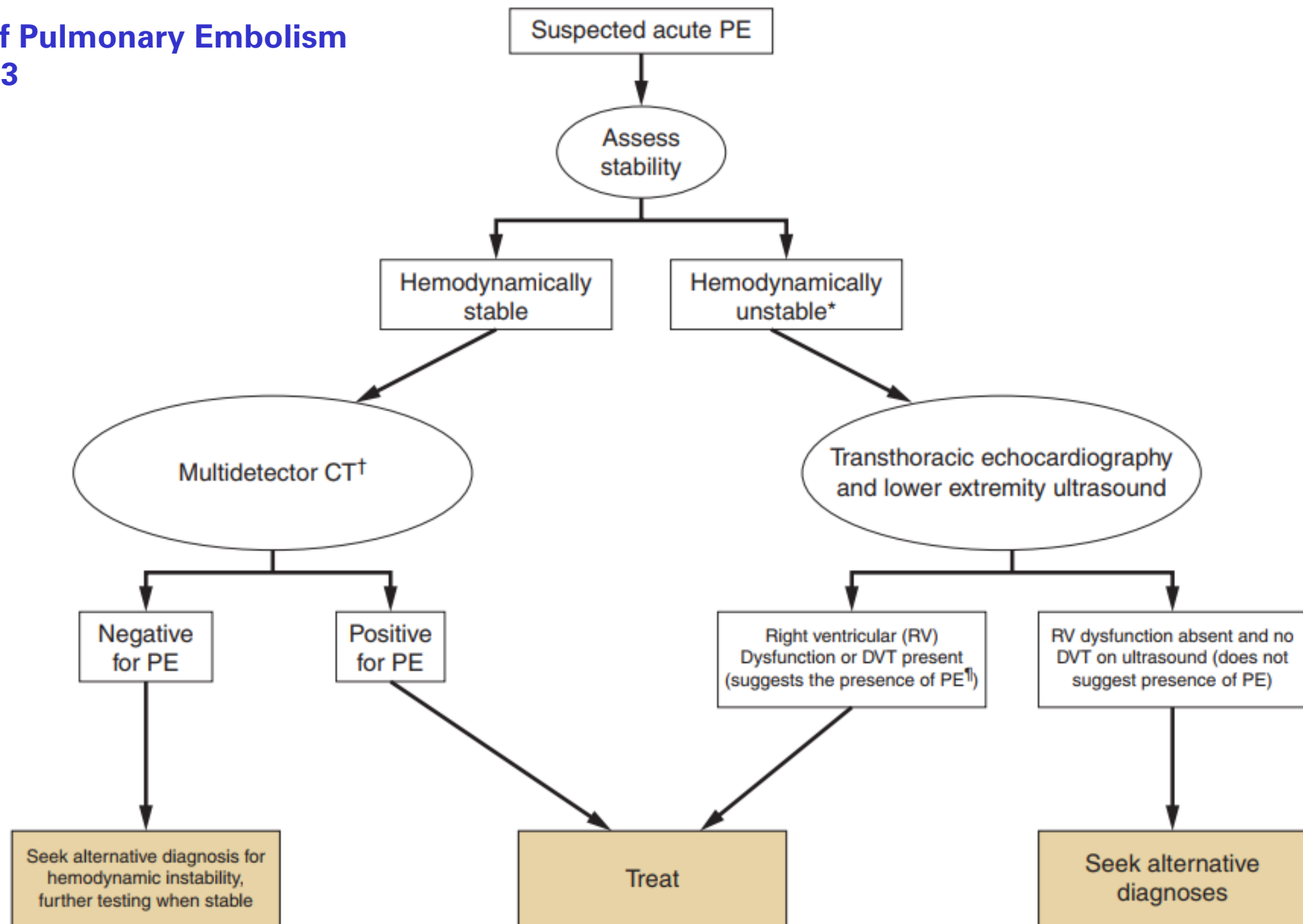
<https://doi.org/10.1016/j.ccc.2020.02.006>

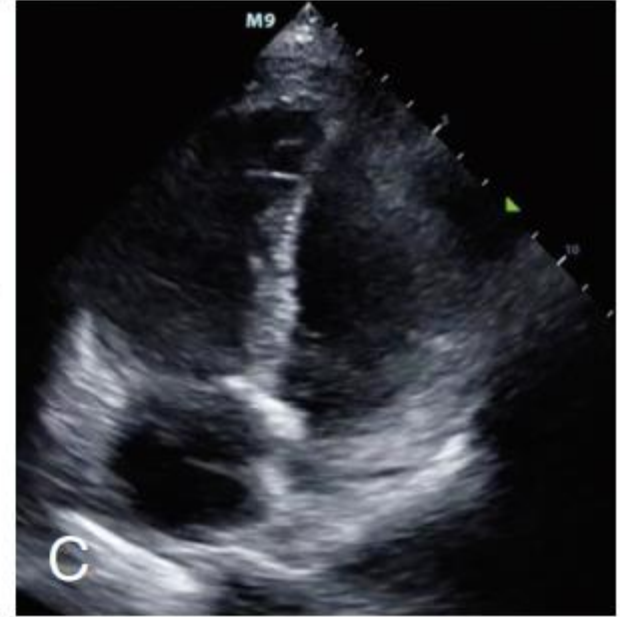
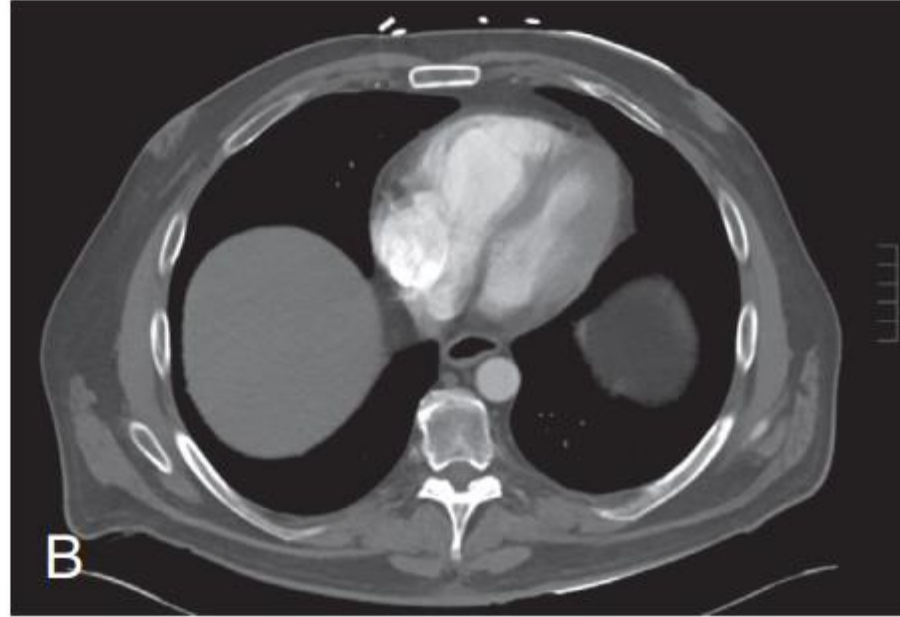
**Table 1**  
Treatment of acute RV failure

Goals	Treatment	Comments
Preload optimization	Judicious volume expansion with crystalloid	<ul style="list-style-type: none"><li>• Avoid volume overload</li><li>• If CVP available and increased, do not give fluids; consider diuretics</li></ul>
Maintain systemic pressure and coronary perfusion	NE 1–40 µg/min is first line	Add vasopressin 0.04 U/min when NE dose >15 µg/min
Augment cardiac output	Dobutamine 2–10 µg/kg/min is first line	<ul style="list-style-type: none"><li>• Add dobutamine only after NE has already been started</li><li>• Avoid milrinone because of vasodilatory effects</li><li>• Epinephrine 1–10 µg/min is second line</li></ul>
Pulmonary vasodilators	iNO 10–20 ppm	<ul style="list-style-type: none"><li>• Physiologic rationale exists for pulmonary vasodilators</li><li>• Evidence for benefit is lacking but no indication of harm</li><li>• Use inhaled pulmonary vasodilators to avoid VQ mismatch</li></ul>

# Diagnosis of Pulmonary Embolism

Vincent 2023





**A, Acute PE.** A large bilateral acute pulmonary embolism overlying the main pulmonary bifurcation ("saddle PE")

**B,** An enlarged right ventricle in the setting of acute pulmonary embolism

**C,** Echo showing acute right heart strain

# Cardiac Arrest Management

- 1-Same as other patients
- 2-the most useful initial test is bedside TTE
- 3-**In a highly unstable patient, TTE evidence of RV dysfunction is sufficient to prompt initiation of heparin + reperfusion strategy[thrombolysis and/or embolectomy ]**

# Cardiac Arrest Management

4-**In patients undergoing CPR**, the use of **systemic thrombolysis** should be considered [bolus of 0.6 mg/kg of tPA iv over 15 min]

5-After the administration of tPA, it is recommended to continue CPR for at least 60 min

6-Following the reperfusion treatment or preferably during the therapy, the patient should be started on **therapeutic hypothermia**

7-The use of ECMO [**VA-ECMO**] during or after CPR while definitive therapy is given, for hemodynamic support is an option



**AHA's My Life Check–Life's Simple 7**