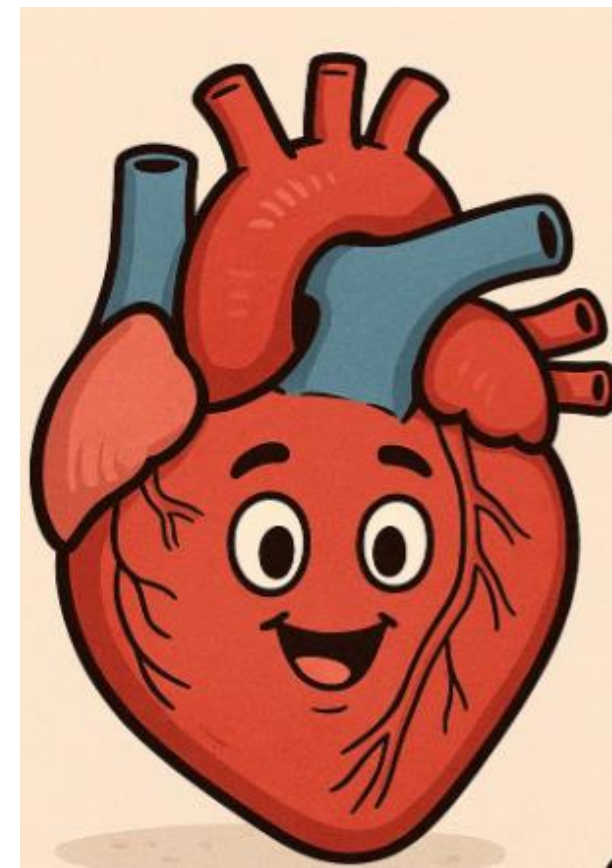
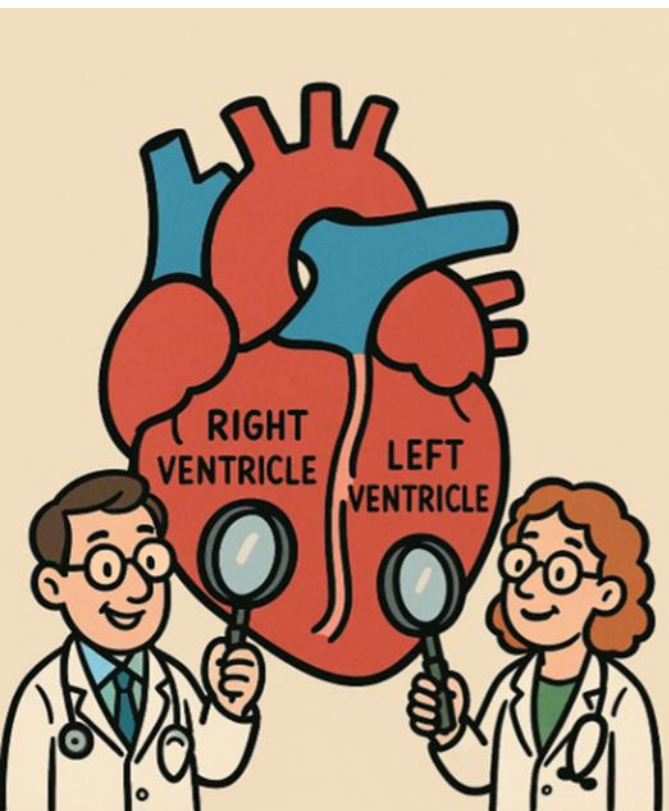


Diagnosis and Management of Acute Right ventricular dysfunction in ICU

인제의대 해운대백병원
장항제

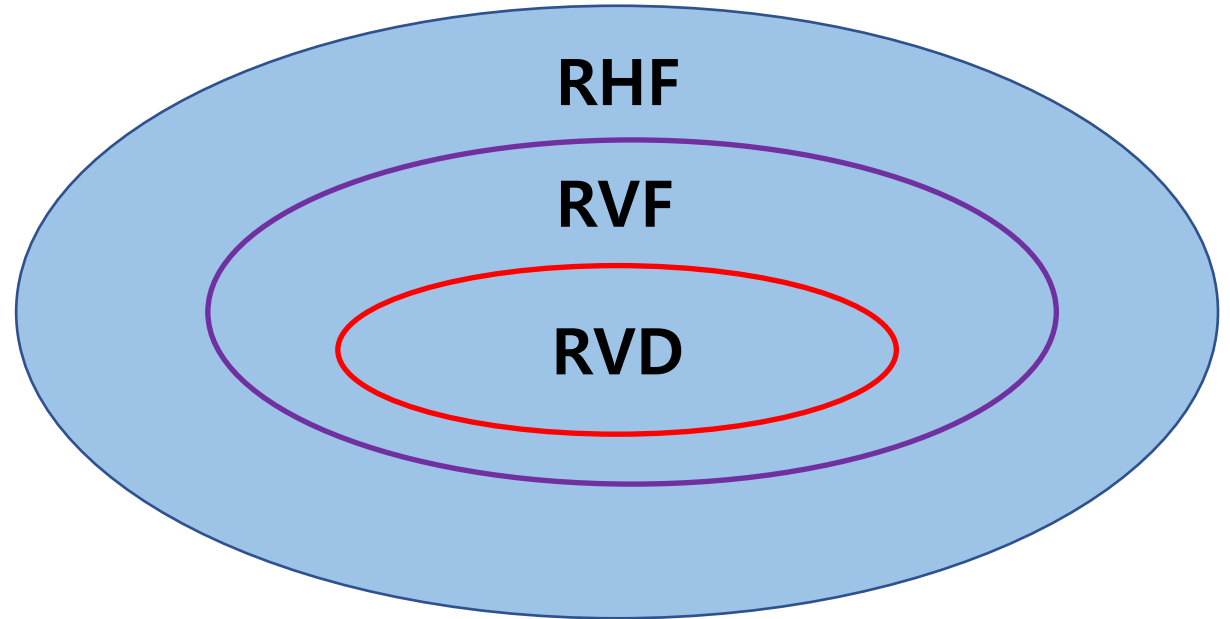


Contents

- 정의/병태생리
- ICU 에서 흔히 겪는 질환/상황
- 진단
 - 호흡기 intensivist 가 bedside 에서 echo assessment
 - Pulmonary artery catheterization
- 주요 증례
- 치료/대처

Terminology

- RV dysfunction (RVD)
- RV failure (RVF)



- Right heart failure (RHF)

: a clinical syndrome due to an alteration of structure and/or function of the right heart circulatory system

Acute Cor Pulmonale (ACP)

■ Definition: Acute Cor Pulmonale (ACP)

Acute cor pulmonale (ACP) is defined as:

A sudden dysfunction of the right ventricle (RV) resulting from an acute increase in pulmonary vascular resistance (PVR), leading to RV dilation, impaired RV output, and potential hemodynamic collapse, in the absence of left heart failure.

🫀 Key Features

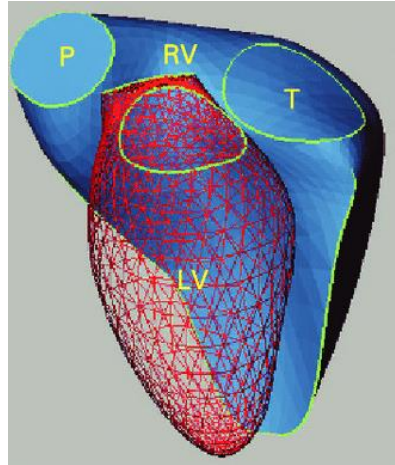
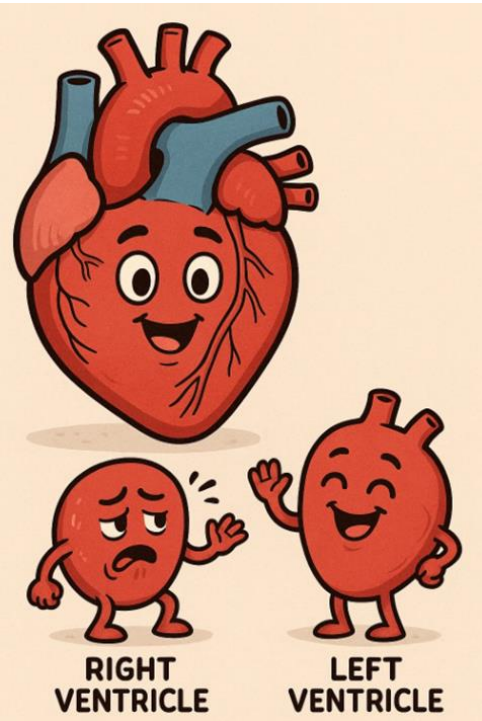
- **Cause:** Acute pressure overload of the RV
- **Common precipitants:**
 - **Massive pulmonary embolism (PE)**
 - **Severe acute respiratory distress syndrome (ARDS)** with hypoxic pulmonary vasoconstriction and high ventilator pressures
 - **Thoracic trauma** or acute pulmonary hypertension from other causes

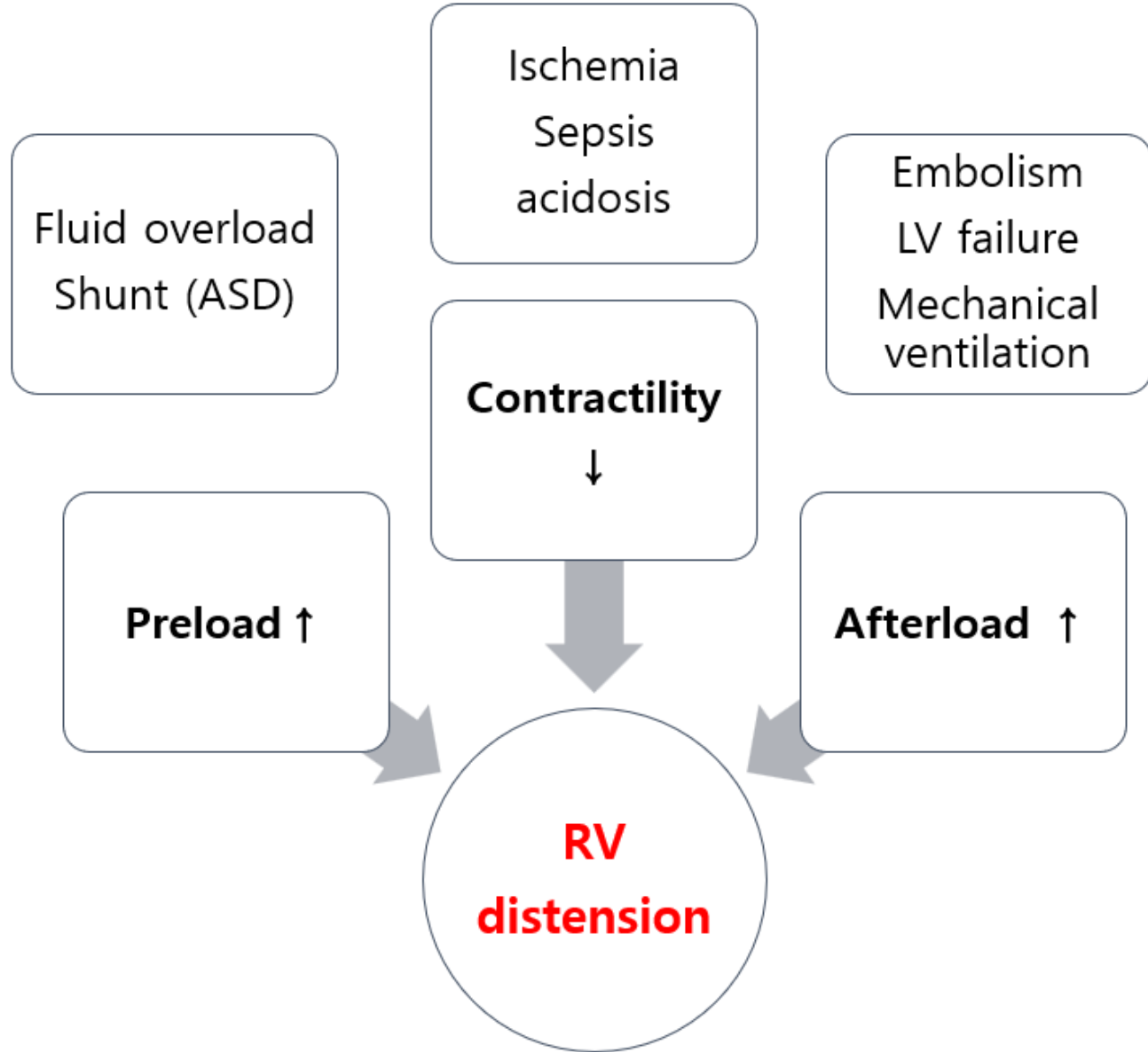
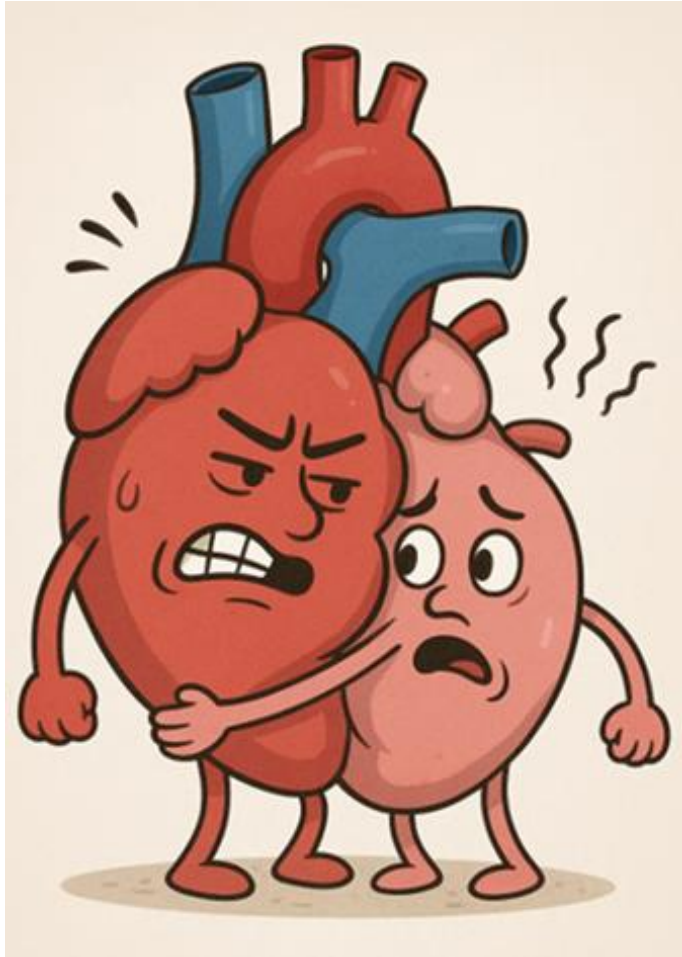
RV 생리학적 특성

1. Passive crescent chamber promoting venous return
From the systemic to pulmonary circulation

2. 수축력 reserve 없다

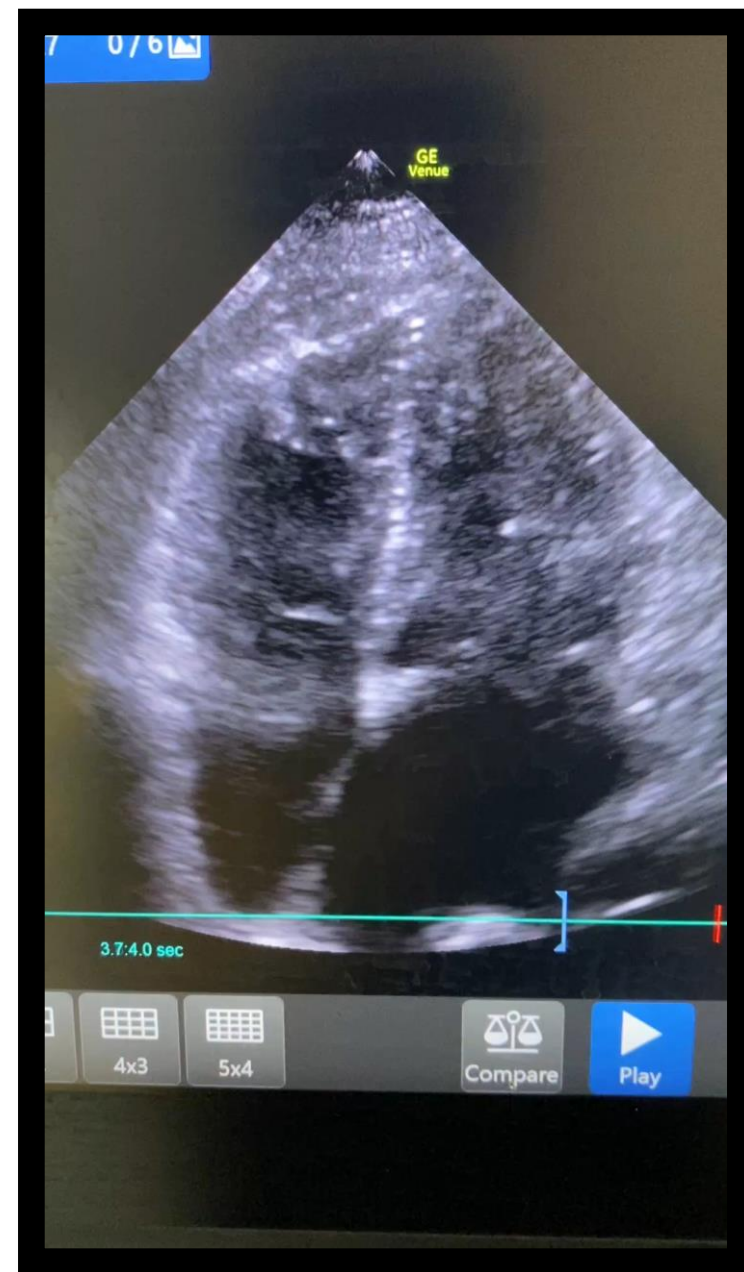
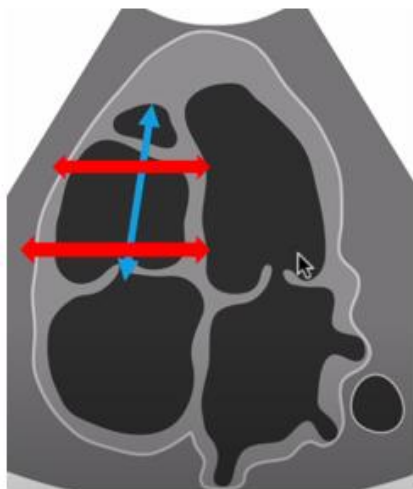
3. 후부하가 증가하면 우심실을 팽창시키는 방법 외의
급성 적응 기전이 없다





RV size

- Normal $< 2/3$ of LV
- Mild normal \leftrightarrow moderately
- Moderately RV = LV
- Severely RV $>$ LV



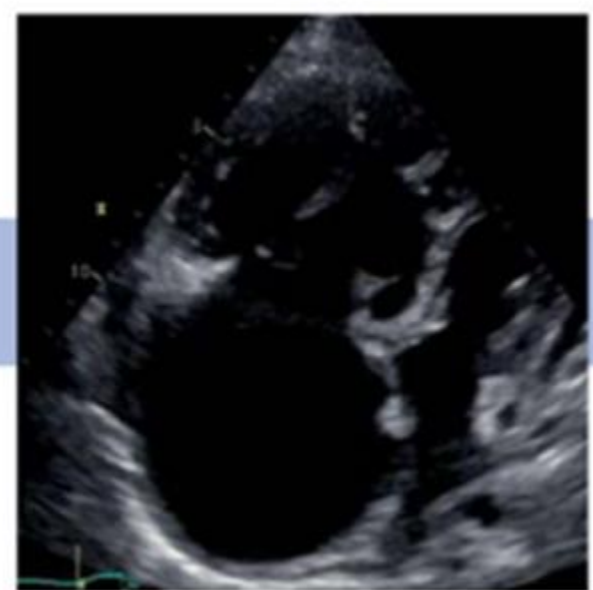
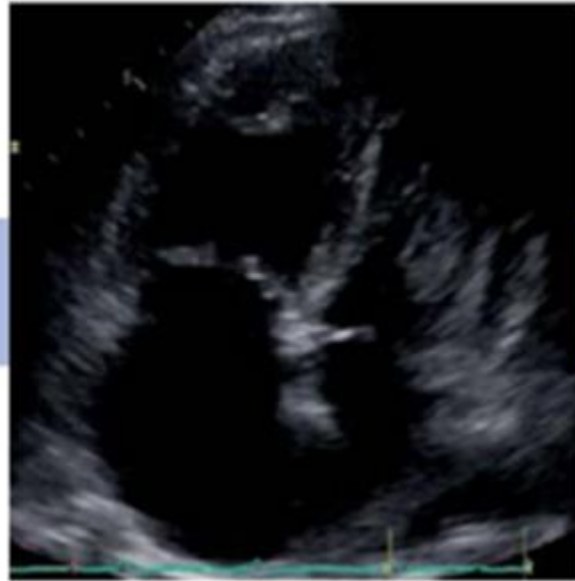
Normal RV size with RV dysfunction (**No afterload ↑**)

- inferior myocardial infarction (MI)
- Stress CMP/Myocarditis involving RV

- while it may be normal in size,
- It can still exhibit dysfunction.
- often associated with hemodynamic compromise, hypotension, clear lungs, RAP ↑ despite the LV maintaining relatively normal function.

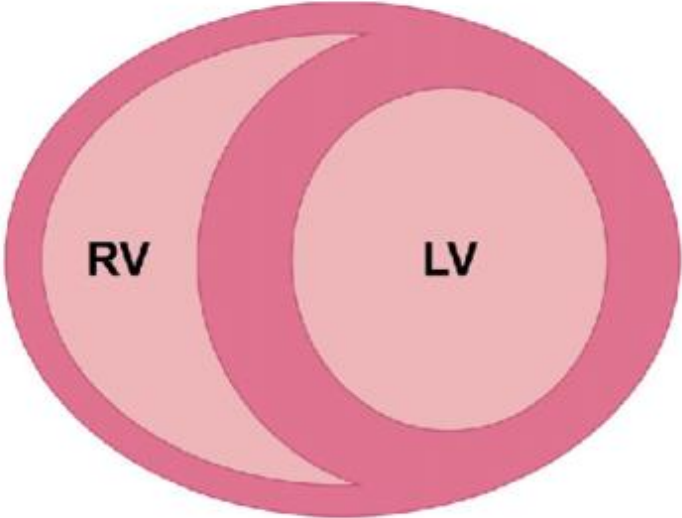


RV/RA dilatation → RV dysfunction

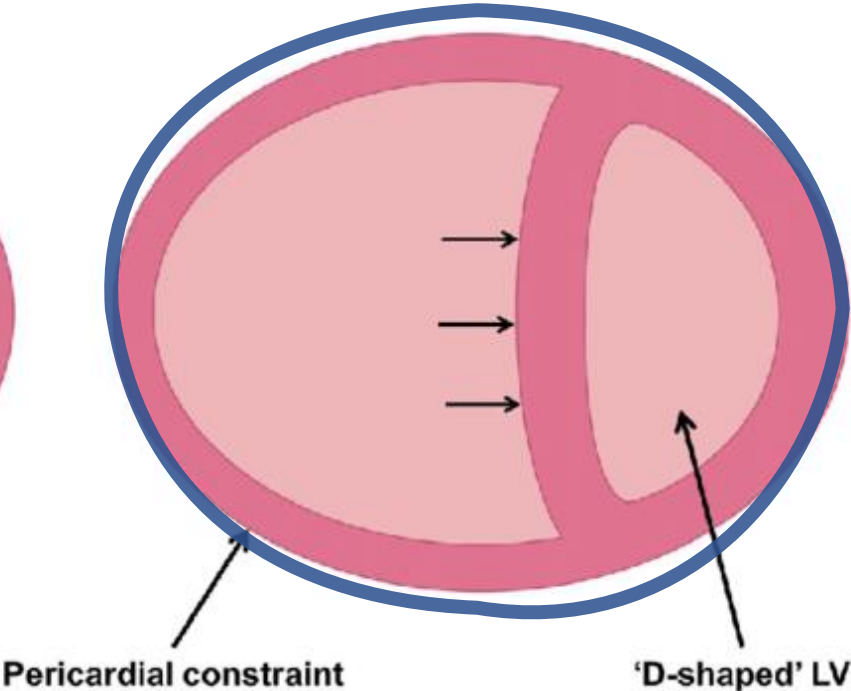


ventricular interdependence

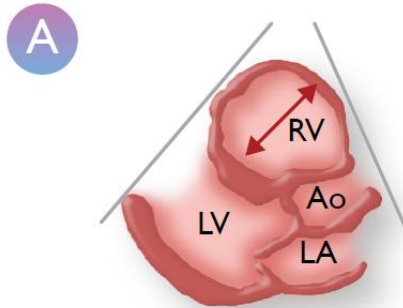
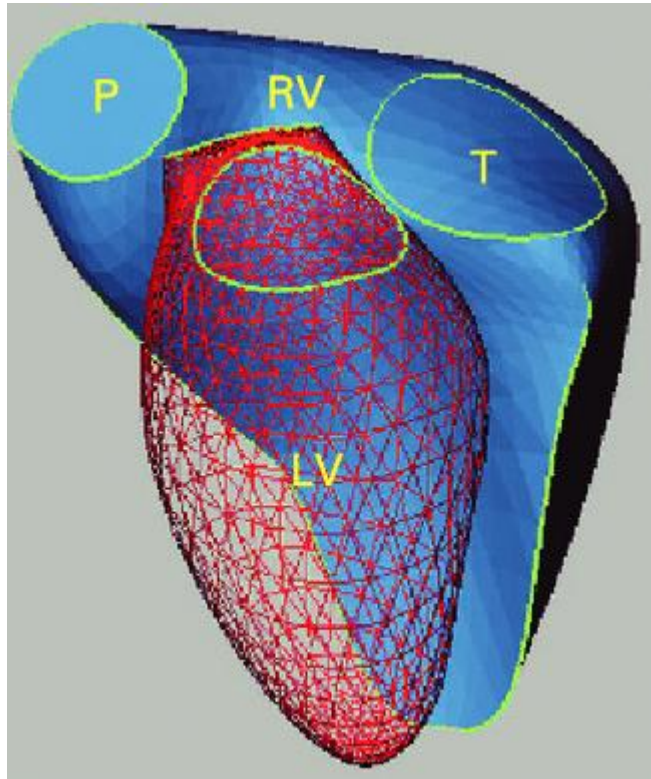
Normal heart



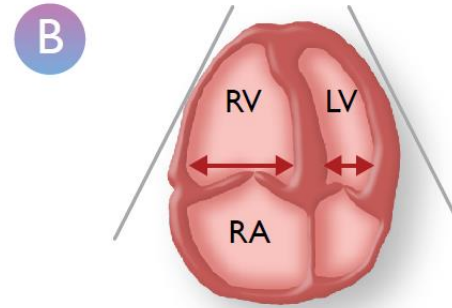
RV overload and dilatation



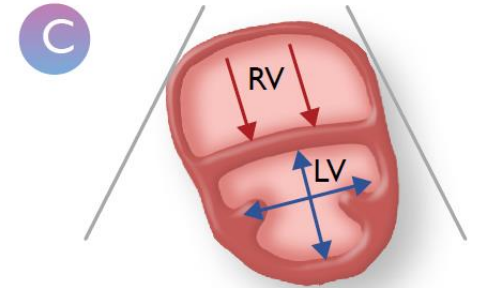
Right heart function: **RV size, RA size, IVC diameter**



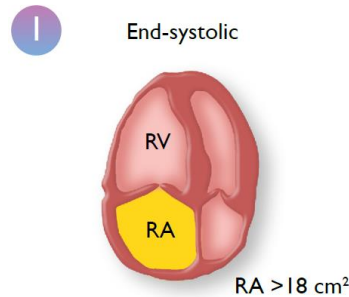
Enlarged right ventricle;
parasternal long-axis view



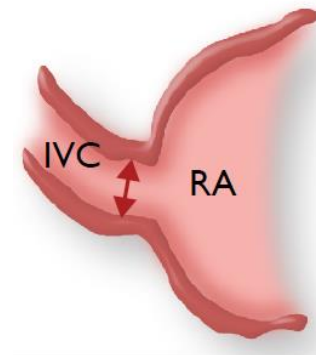
Dilated RV with basal RV/LV
ratio > 1.0 ;
four-chamber view



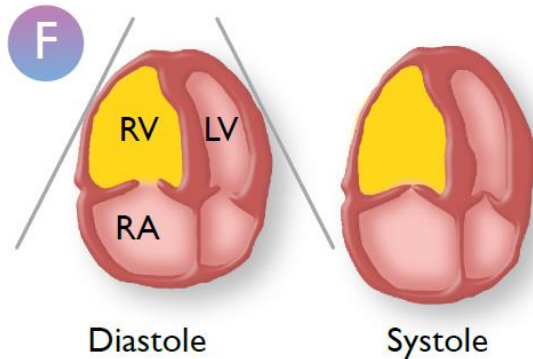
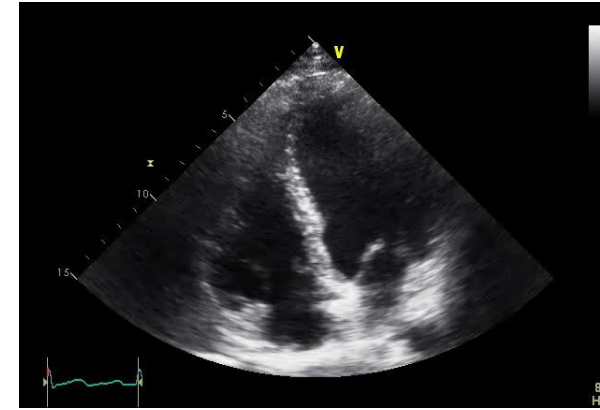
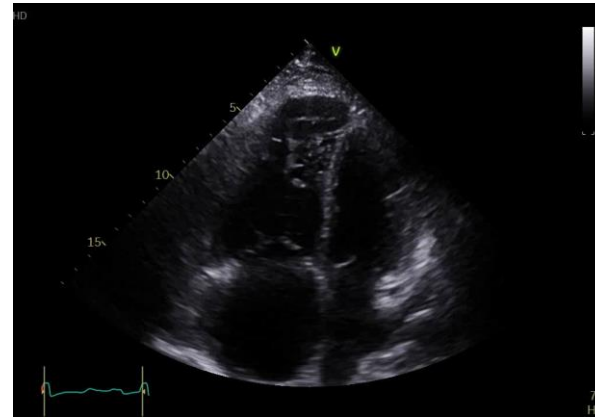
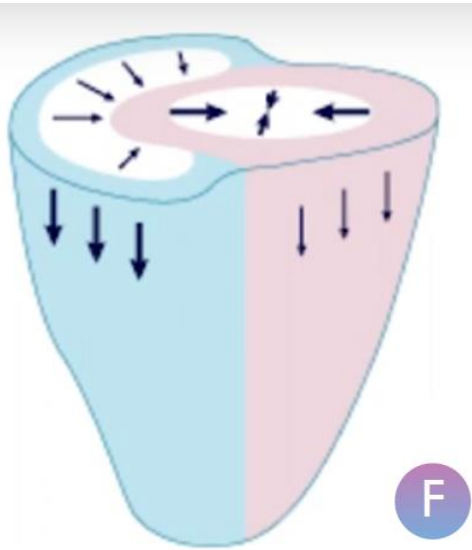
Flattened interventricular septum
(arrows) leading to 'D-shaped' LV;
decreased LV eccentricity index;
parasternal short-axis view



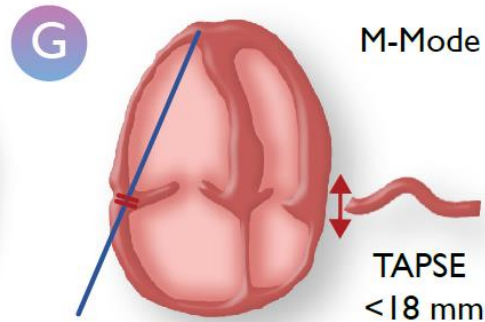
Enlarged right atrial area
($> 18 \text{ cm}^2$);
four-chamber view



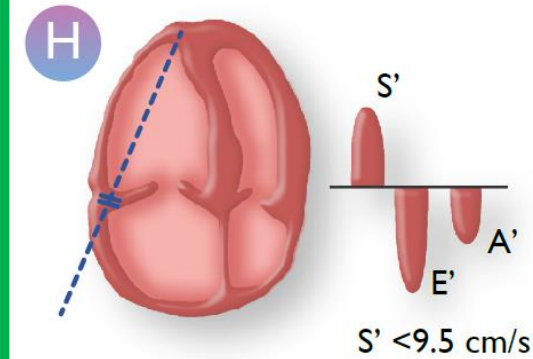
Right heart function – **RV contractility**



Reduced right ventricular fractional area change (<35%); four-chamber view



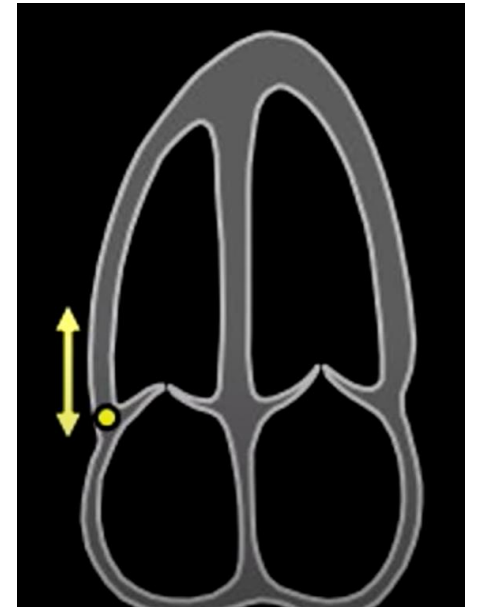
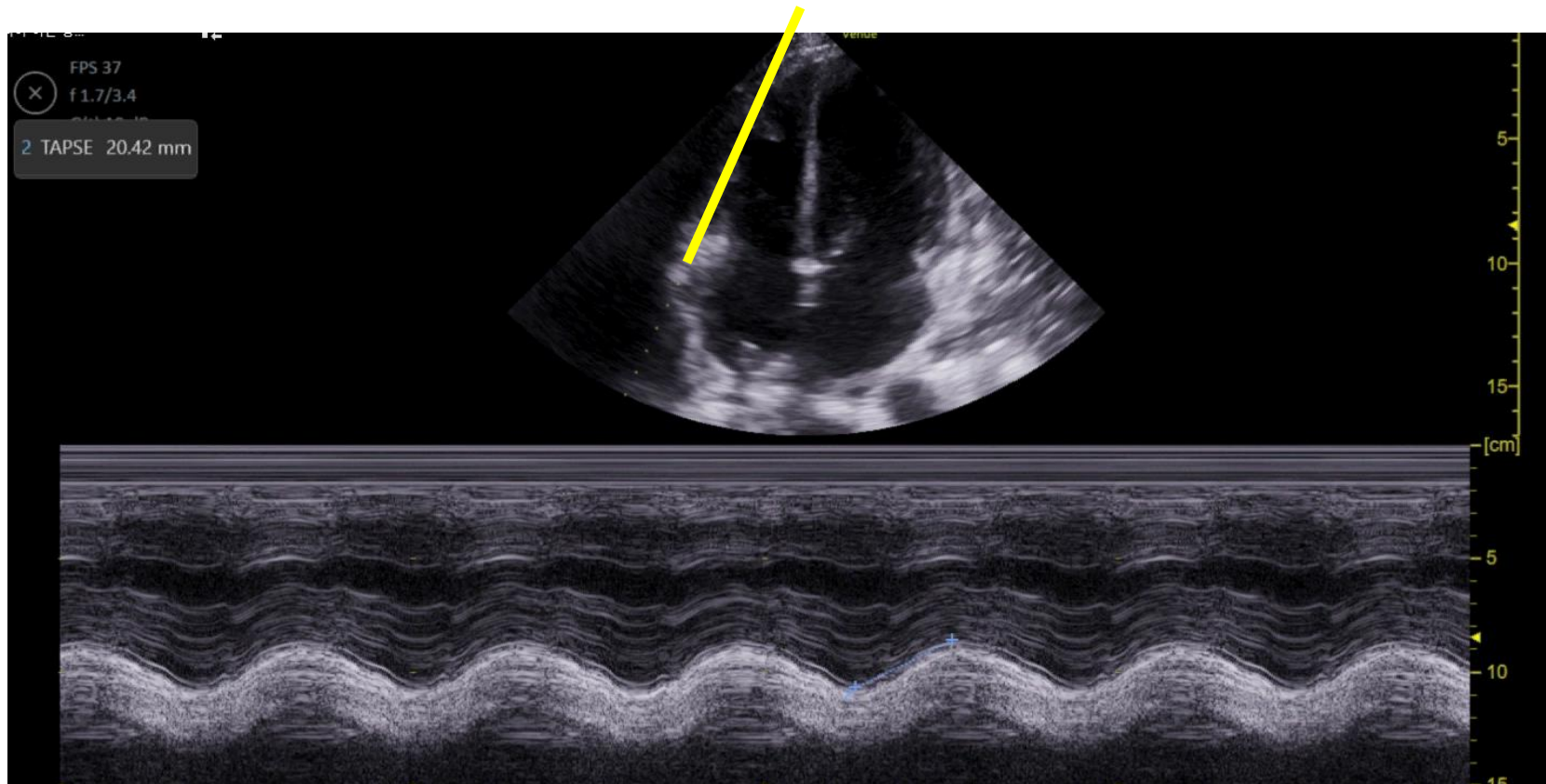
Decreased tricuspid annular plane systolic excursion (TAPSE) measured with M-Mode (<18 mm)



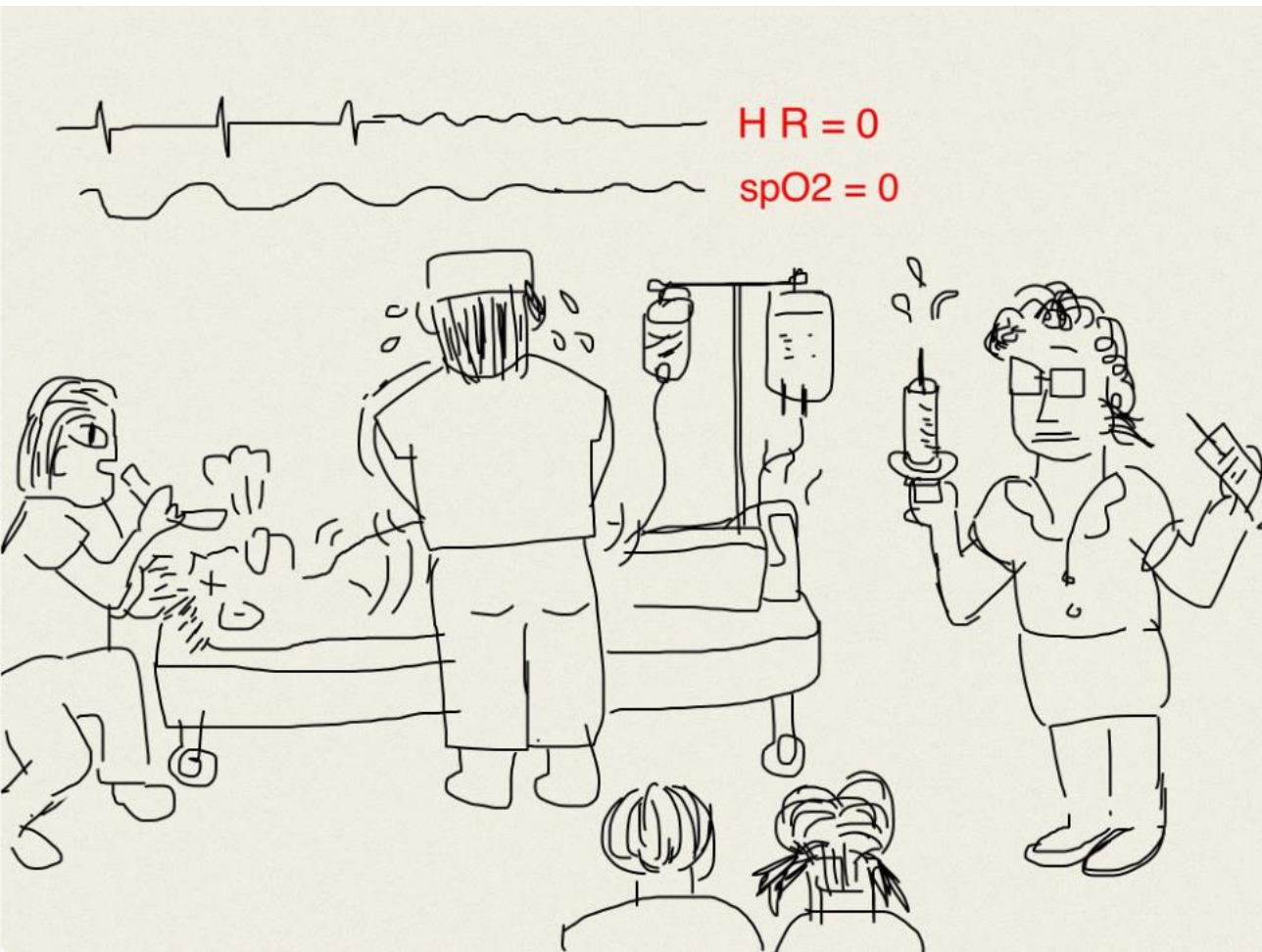
Decreased peak systolic (S') velocity of tricuspid annulus (<9.5 cm/s) measured with tissue Doppler

Bedside Echo RV Function evaluation by intensivist

TAPSE : Tricuspid Annular Plane Systolic Excursion < 18 mm

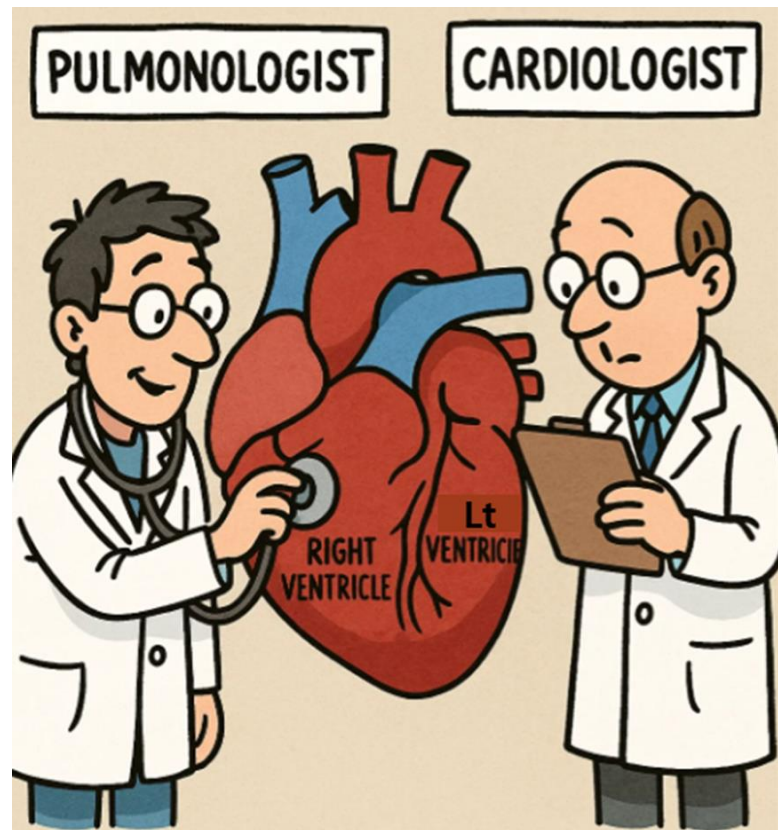


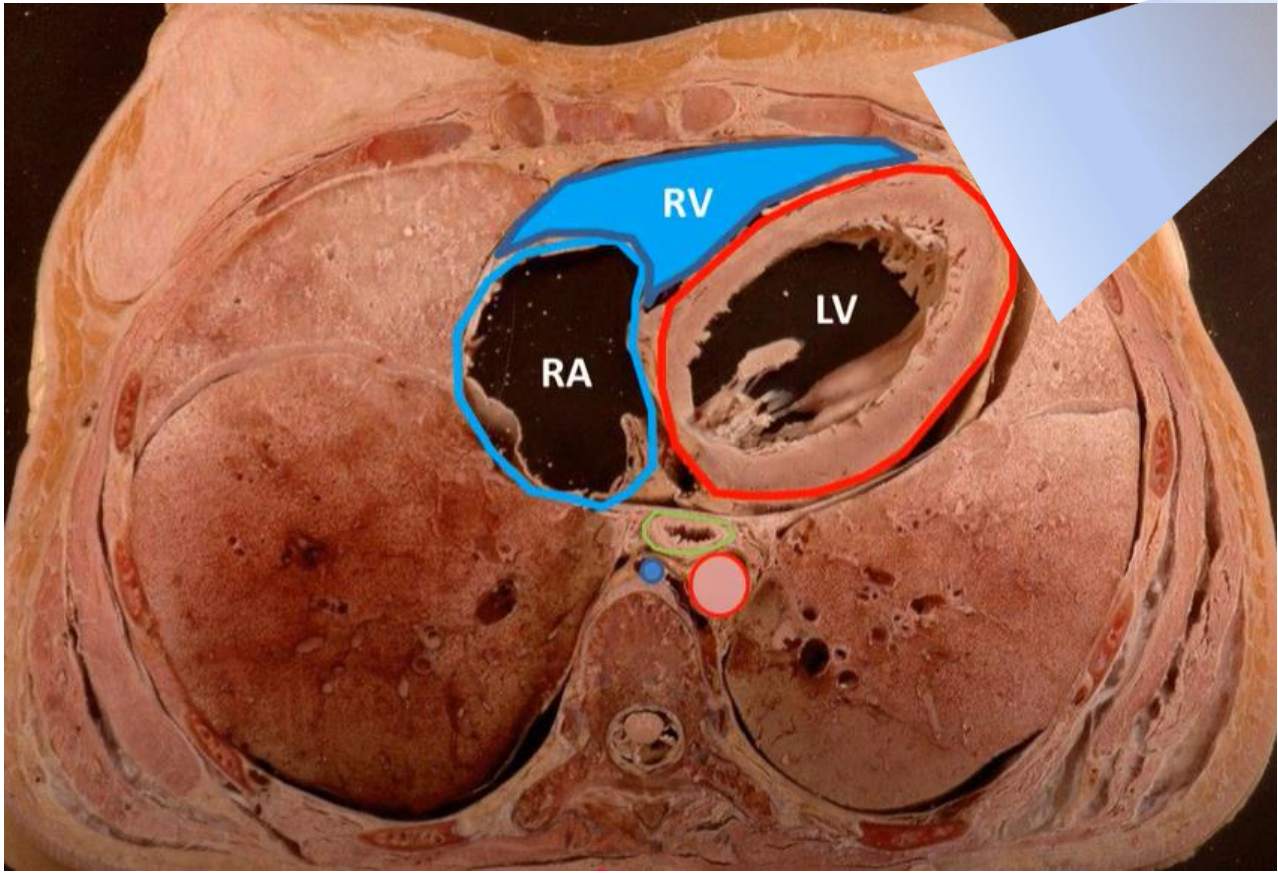
Sudden cardiac arrest in general ward
→ transfer to ICU after ROSC



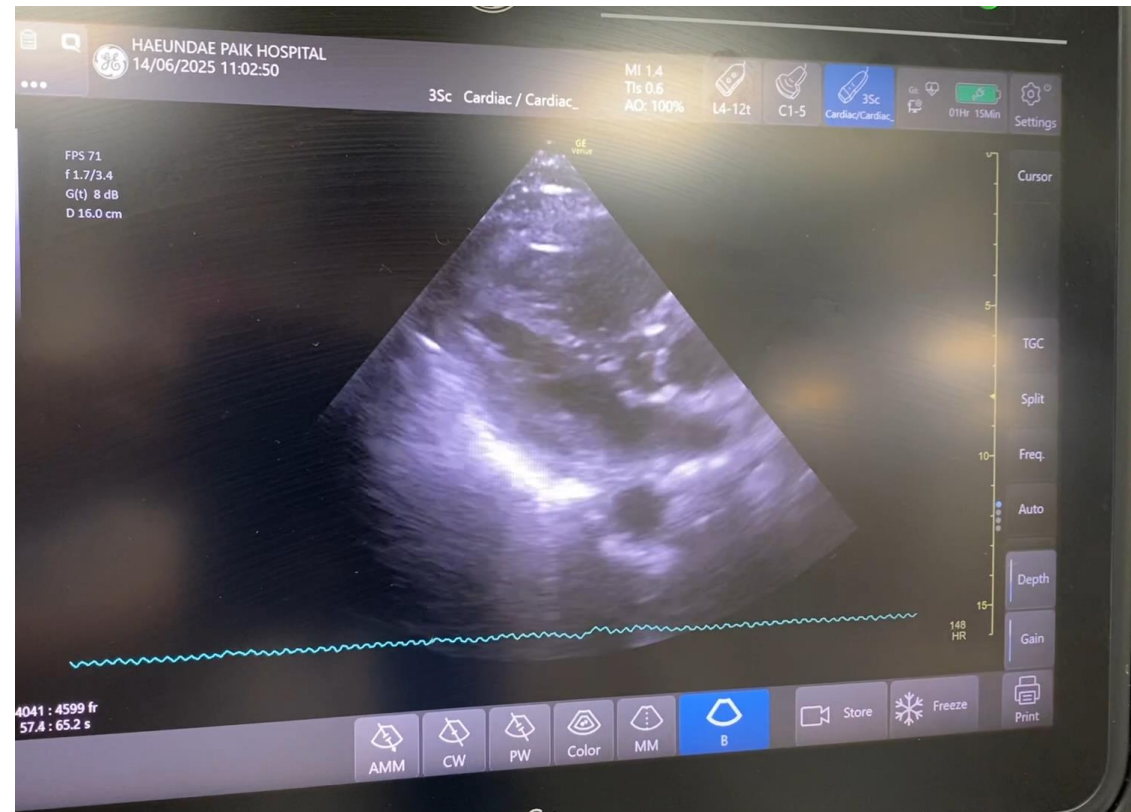
Hyperdynamic LV!!

What the hell in RV ?

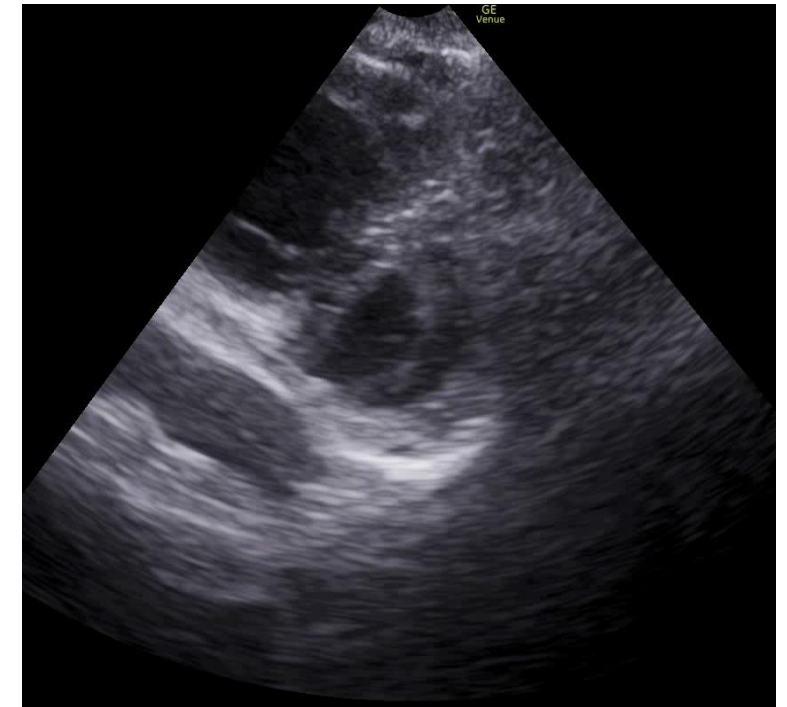
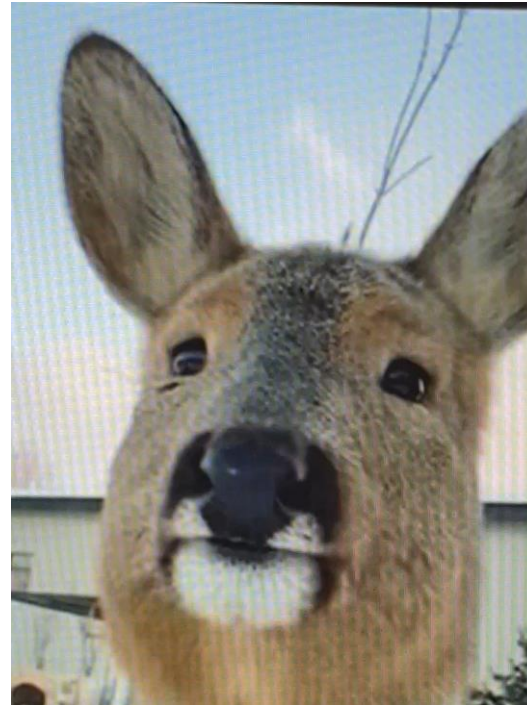




Massive PTE impending cardiac arrest

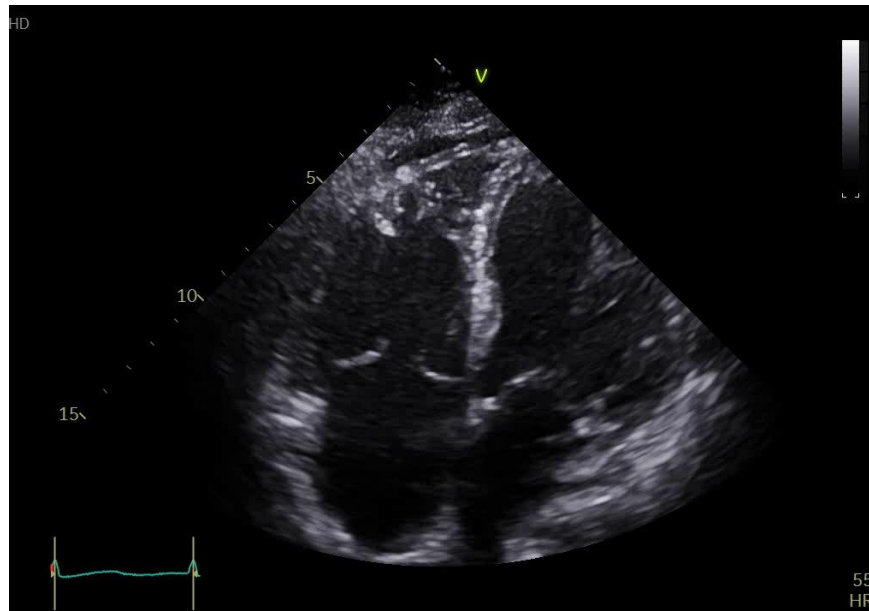


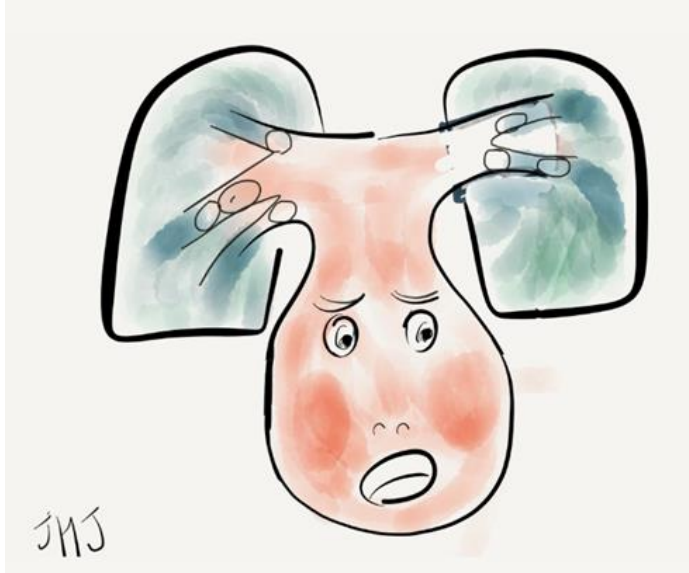
Massive PTE impending cardiac arrest



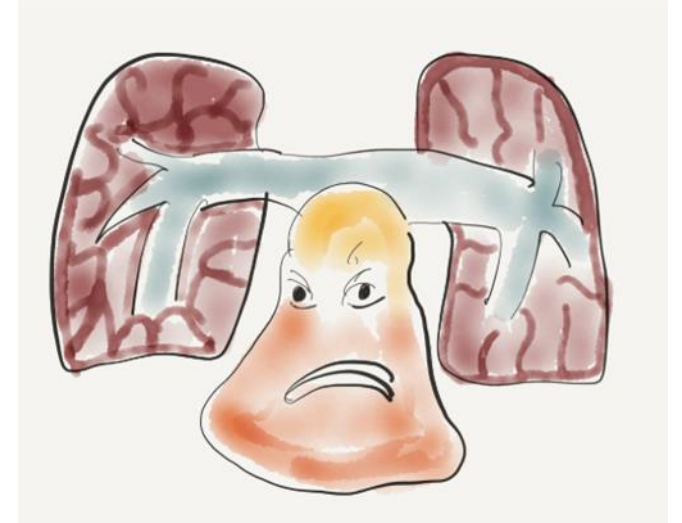
“Sudden deterioration in mechanically ventilated patients”

RV dysfunction in bedside echo





Massive PTE

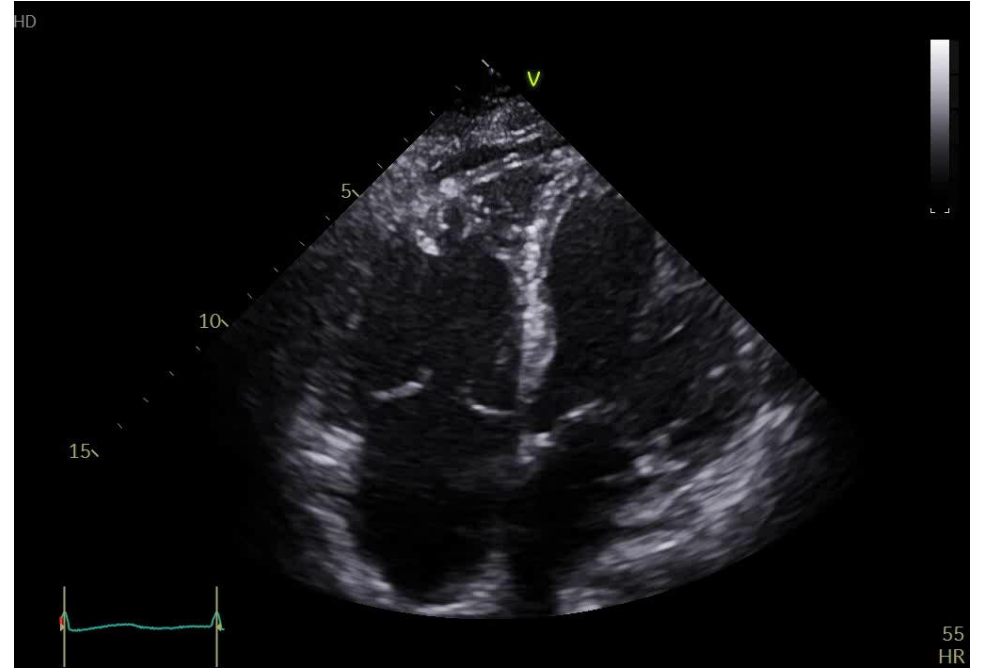


**Massive aspiration
Bronchospasm
Sepsis**



Echo 로 감별 가능할까? **McConnell's Sign**

- is an echocardiographic finding that is
- **highly specific for acute pulmonary embolism**
- 하지만 다른 원인에서도 보일 수 있을까?

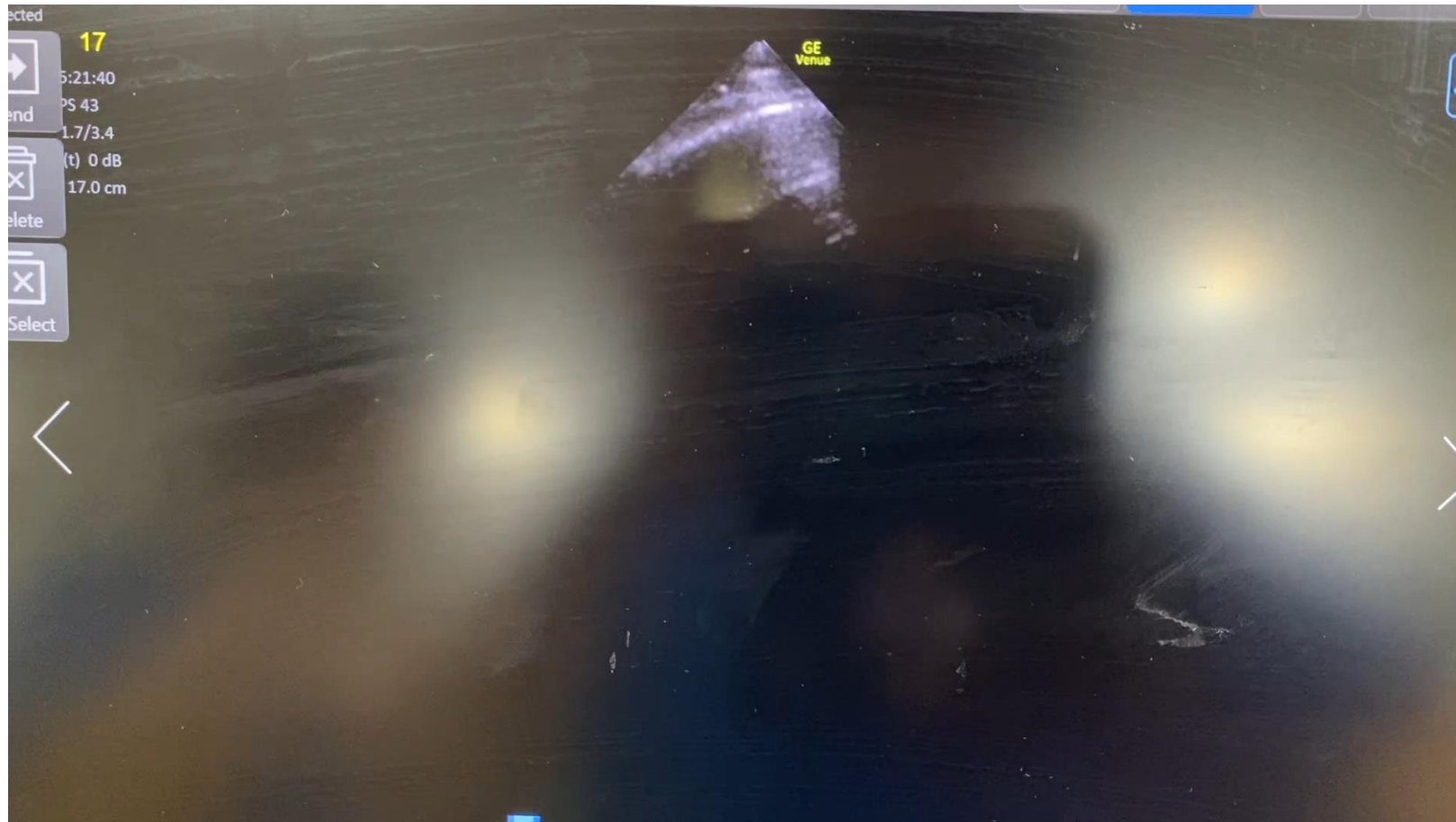


Physiologic Mechanism

- In **acute PE**, sudden rise in pulmonary vascular resistance leads to **RV pressure overload**.
- The **RV free wall becomes stunned or akinetic**, while the **apex retains motion** due to shared fibers with the LV.
- This regional difference creates a "trampoline-like" appearance.

No pulmonary embolism, Cardiac arrest due to PH crisis

“Pulmonary capillary hemangiomatosis”



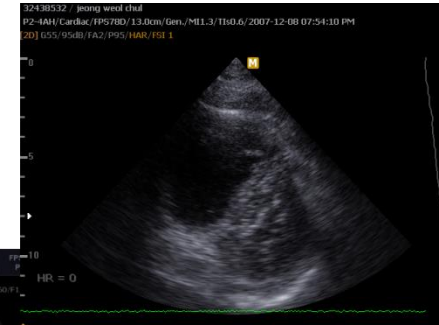
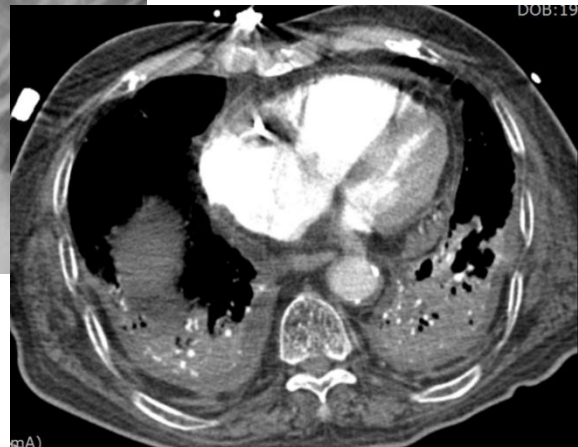
A R D S

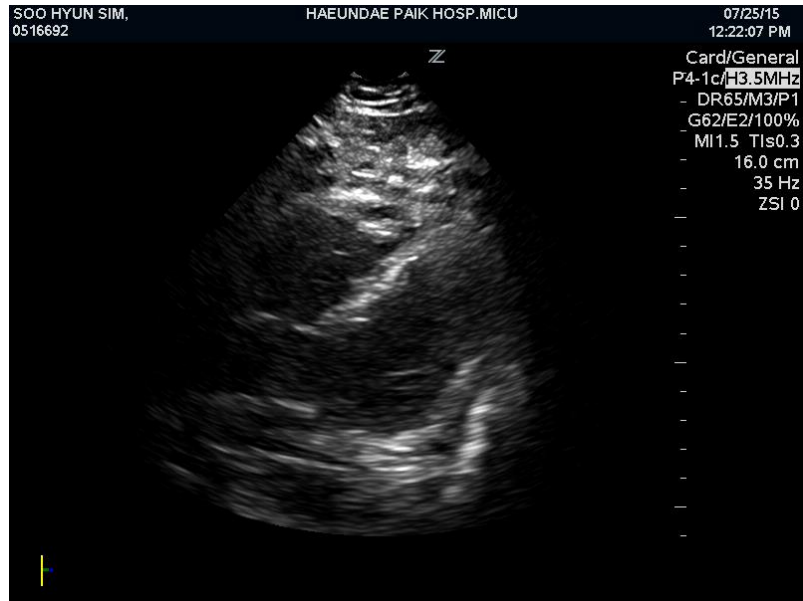
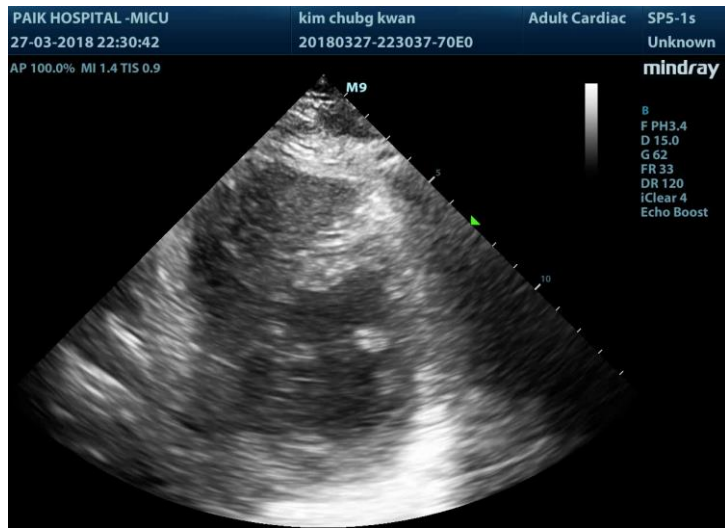
Severe pneumonia ARDS with septic shock

Hypoxic pulmonary vasoconstriction

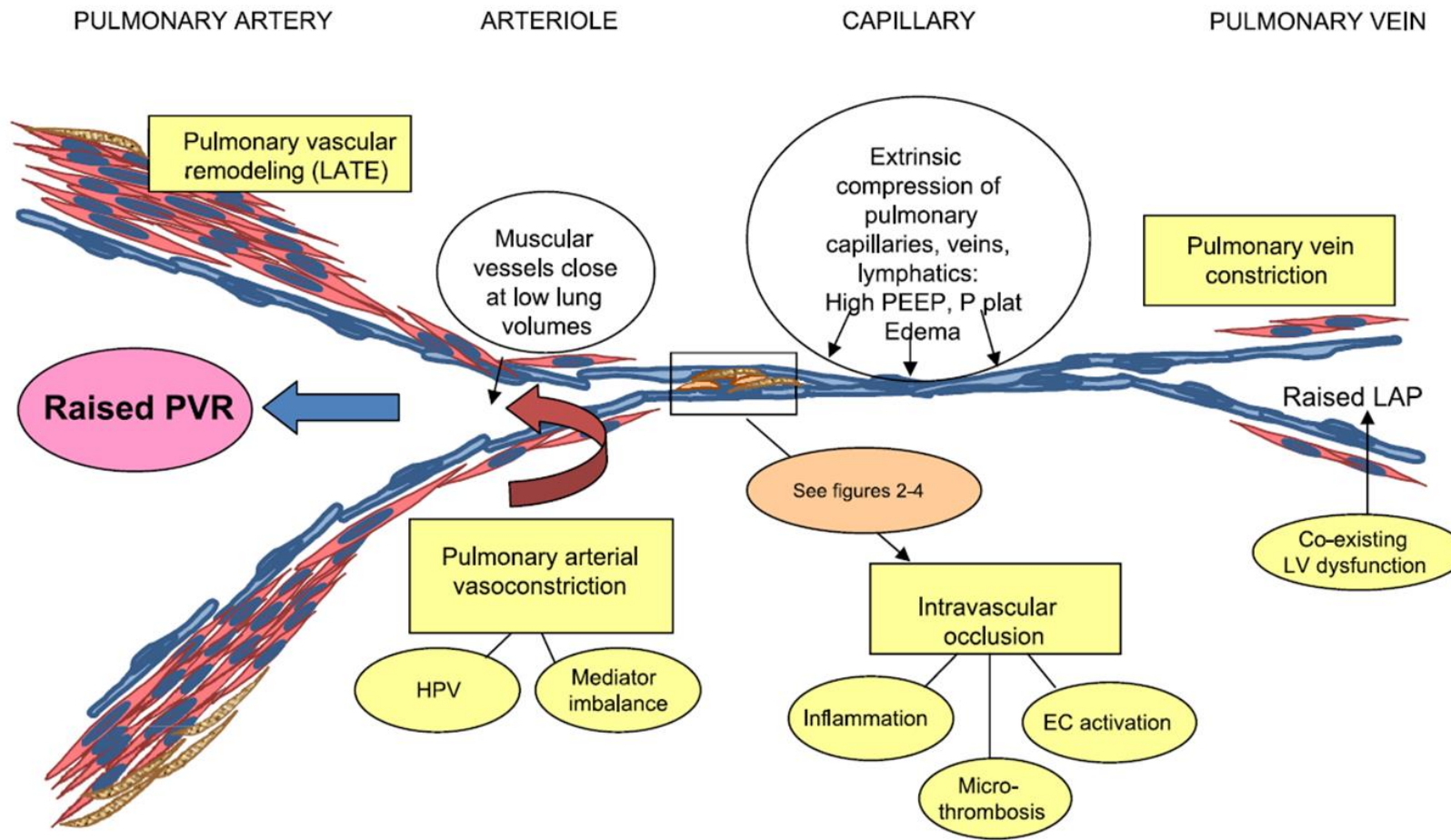
→ RV pressure overload

→ **acute cor pulmonale**

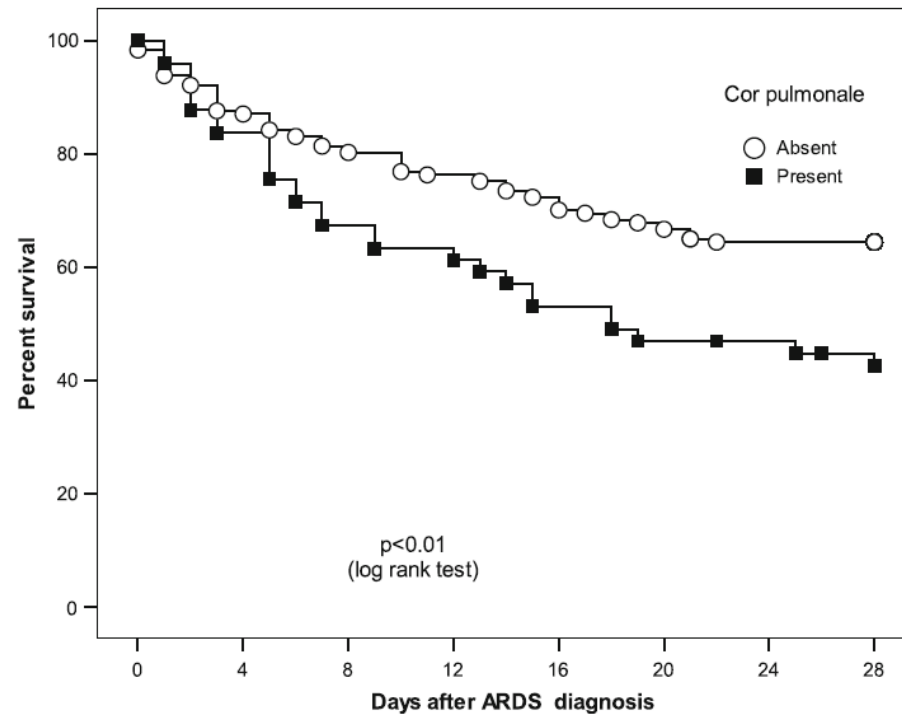




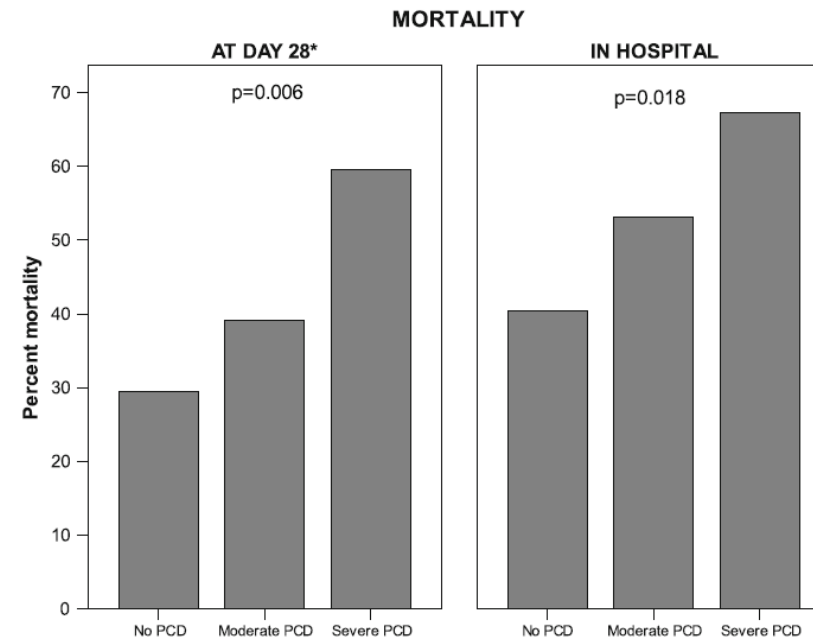
Pathophysiology of PH in ARDS



Prevalence and prognosis of cor pulmonale during protective ventilation for acute respiratory distress syndrome

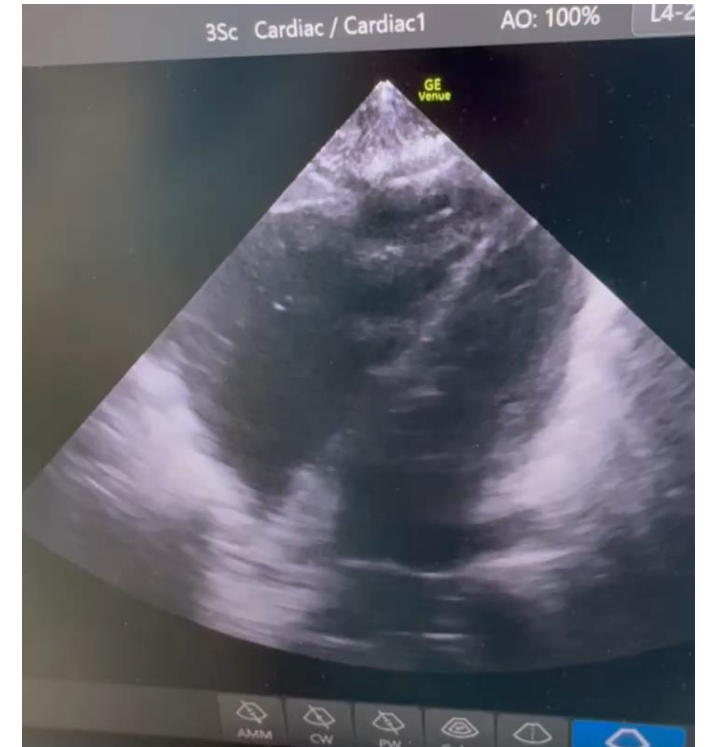


Number at risk									
Cor pulmonale									
No	Yes	0	4	8	12	16	20	24	28
177	49	154	143	135	127	119	113	112	
		41	33	31	26	23	22	19	



RV dysfunction as a complication of **ARDS**

- More gradual than in patients with massive PE
- Usually occurring at least 48 hours after ventilator support
- 25% incidence of acute right ventricular failure
- reversible in patients who recovered



RV dysfunction as a complication of **ARDS**

- The initial magnitude of PH was not an indicator of mortality rate
- **Mean PAP** increased in non-survivors
- not in survivors when followed for 7 days
- Development of PH in ARDS patients
 - seems to be **a sign of poor prognosis**

Armand Mekontso Dessap
Florence Boissier
Cyril Charron
Emmanuelle Bégot
Xavier Repessé
Annick Legras
Christian Brun-Buisson
Philippe Vignon
Antoine Vieillard-Baron

Acute cor pulmonale during protective ventilation for acute respiratory distress syndrome: prevalence, predictors, and clinical impact

- Independently associated with **acute cor pulmonale**

Table 3 The acute cor pulmonale risk score

Parameter	Score
Pneumonia as cause of ARDS	1
Driving pressure ≥ 18 cmH ₂ O ^a	1
PaO ₂ /FiO ₂ ratio <150 mmHg	1
PaCO ₂ ≥ 48 mmHg	1
Total score	0–4

Prone Positioning Unloads the Right Ventricle in Severe ARDS*

Antoine Vieillard-Baron, MD; Cyril Charron, MD; Vincent Caille, MD; Guillaume Belliard, MD; Bernard Page, MD; and François Jardin, MD

CHEST 2007; 132:1440 –1446

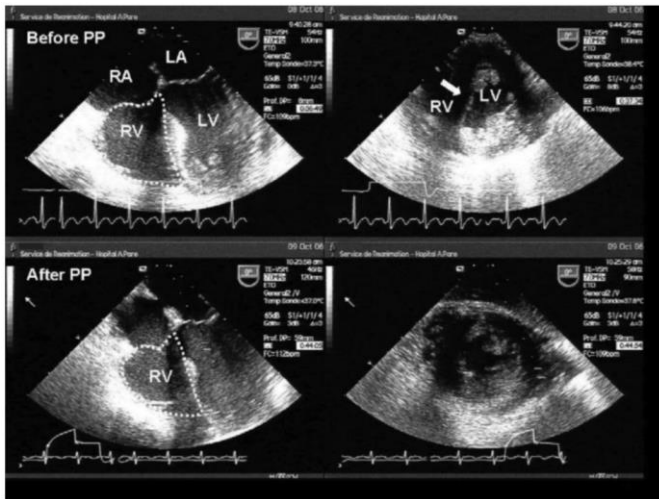
42 ARDS patients treated by Prone position (PP)
21 patients (50%) had **acute cor pulmonale**

significant airway pressure ↓ PaCO₂ ↓.

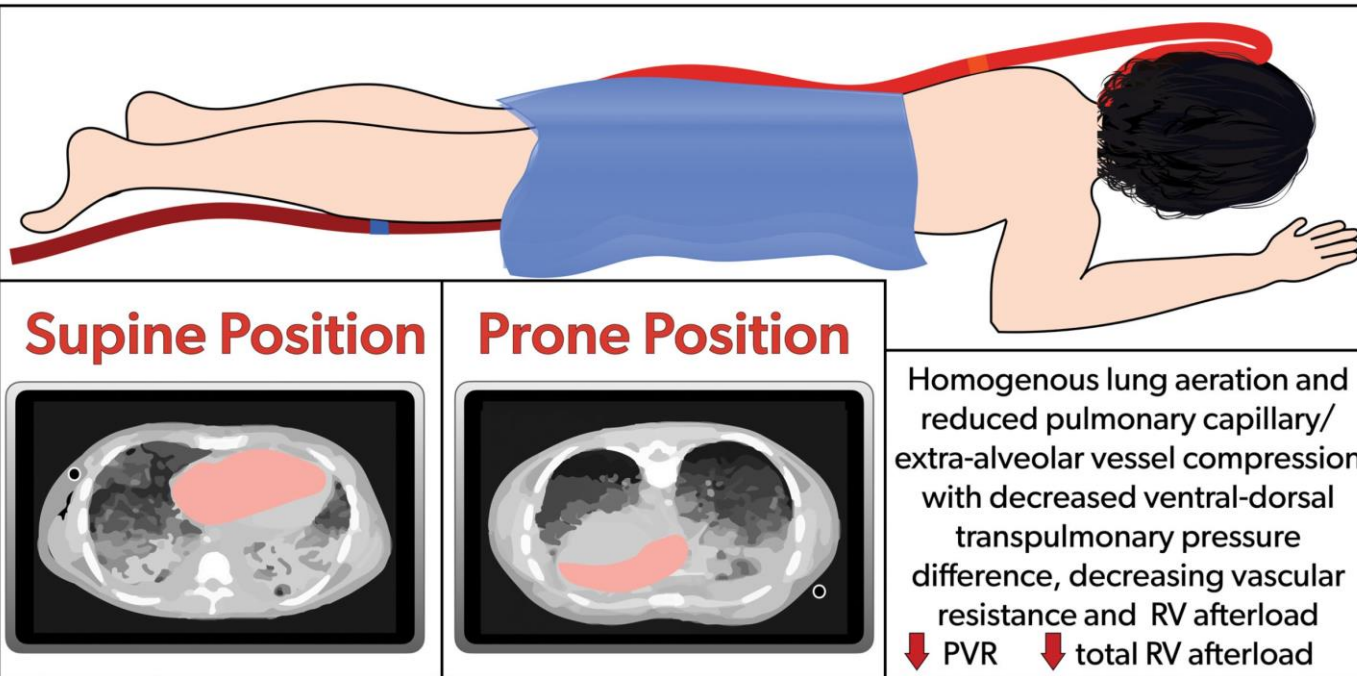
produced a significant decrease in mean **RV enlargement** after 18 h of PP
(p = 0.000)

significant reduction in mean **septal dyskinesia** after 18 h of PP
(p = 0.000)

Conclusion: In the most severe forms of ARDS,
PP was an efficient means of controlling RV pressure overload.



Prone Positioning and Right Ventricular Protection During Extracorporeal Membrane Oxygenation for Severe ARDS



Recruitment of collapsed alveoli in dorsal lung regions
→ overdistension ↓ atelectotrauma

Increased thoracic circulation blood volume
→ Pulmonary microvasculature ↑
→ PVR ↓ RV afterload ↓

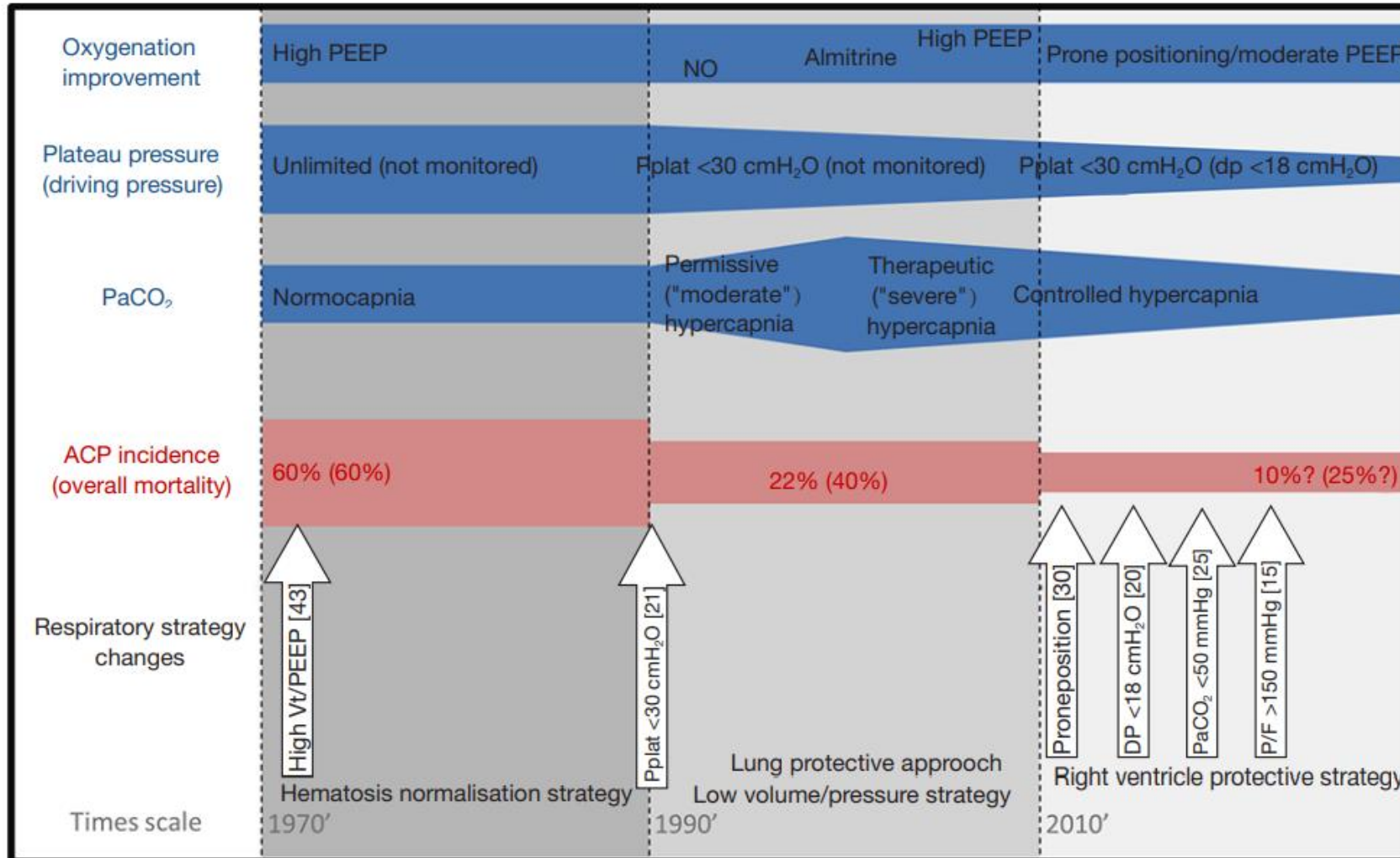
RV-protective ventilatory strategy

1. Lung-Protective Ventilation:

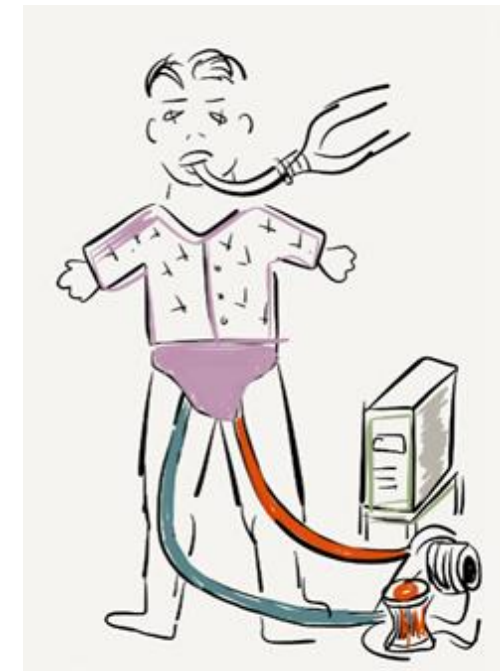
- **Low tidal volume ventilation:** Reducing tidal volume (typically to 6 mL/kg of predicted body weight) helps minimize alveolar overdistension and barotrauma, which can increase RV afterload.
- **Optimal PEEP titration:** Positive end-expiratory pressure (PEEP) is used to prevent alveolar collapse, but excessive PEEP can increase RV afterload. Adjusting PEEP based on RV function and hemodynamic parameters is crucial.
- **Limiting plateau pressure:** Maintaining plateau pressure below 27-30 cmH₂O helps prevent overdistension and reduces the risk of barotrauma.
- **Permissive hypercapnia:** Accepting a slightly elevated PaCO₂ (permissive hypercapnia) can reduce the need for high ventilator settings and minimize RV strain.
- **Driving pressure:** Lowering driving pressure (plateau pressure - PEEP) is associated with improved lung mechanics and can be used as a target to optimize ventilation.

Changes in respiratory strategy

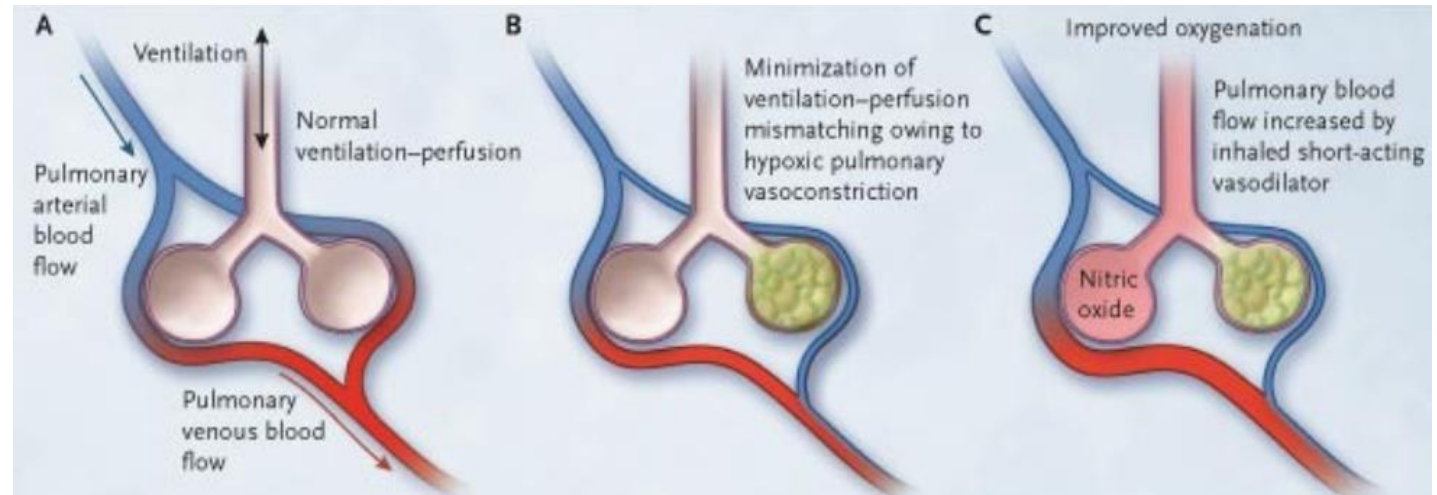
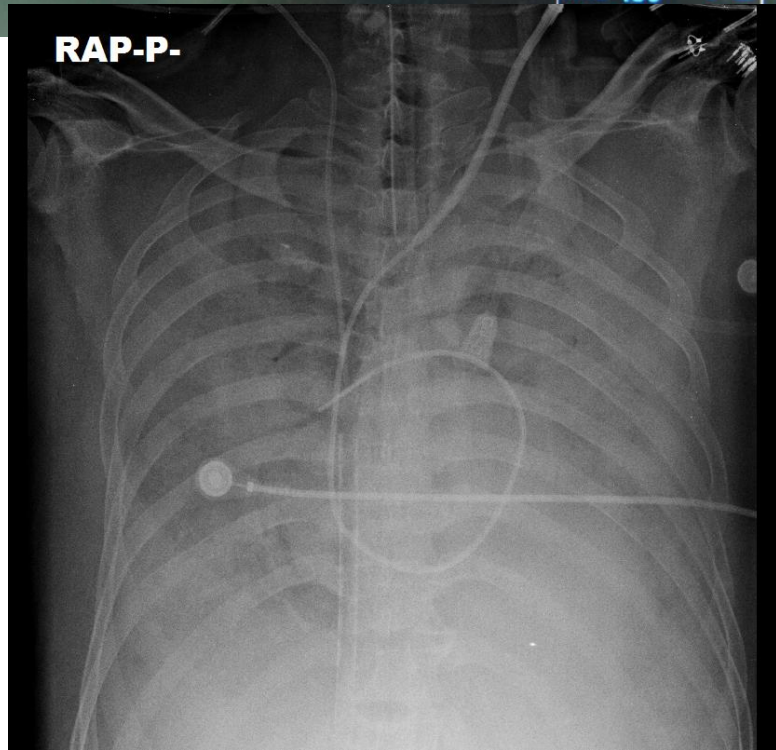
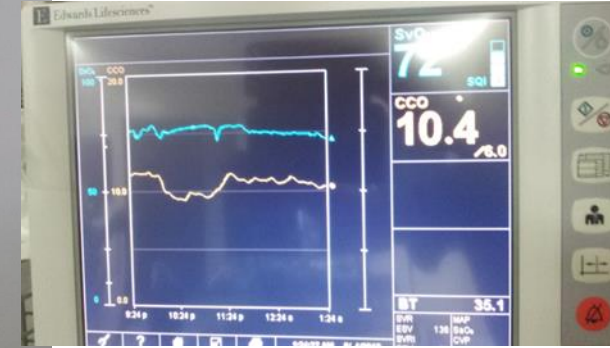
three main risk-identified factors for **acute cor pulmonale in ARDS**



V-V ECMO
ECCO2R



Pulmonary Vasodilator therapy — Nitric oxide gas, prostacycline inhalation



Mark J D Griffiths Timothy W Evans
N Engl J Med. 2005 Dec 22;353(25):2683-95

RESEARCH

Open Access



Inhaled prostacyclin therapy in the acute respiratory distress syndrome: a randomized controlled multicenter trial

Helene A. Haeberle¹, Stefanie Calov¹, Peter Martus², Lina Maria Serna-Higueta², Michael Koeppen¹, Almuth Goll¹, Alice Bernard¹, Alexander Zarbock³, Melanie Meersch³, Raphael Weiss³, Martin Mehrländer¹, Gernot Marx⁴, Christian Putensen⁵, Tamam Bakchoul⁷, Harry Magunia¹, Bernhard Nieswandt⁶, Valbona Mirakaj^{1†} and Peter Rosenberger^{1*†}

Conclusions:

The primary result of our study was negative.

Our data suggest that inhaled prostacyclin might be beneficial treatment in patients with COVID-19 induced ARDS.

증례#1 84/F 구토 후 의식저하로 ICU 입원

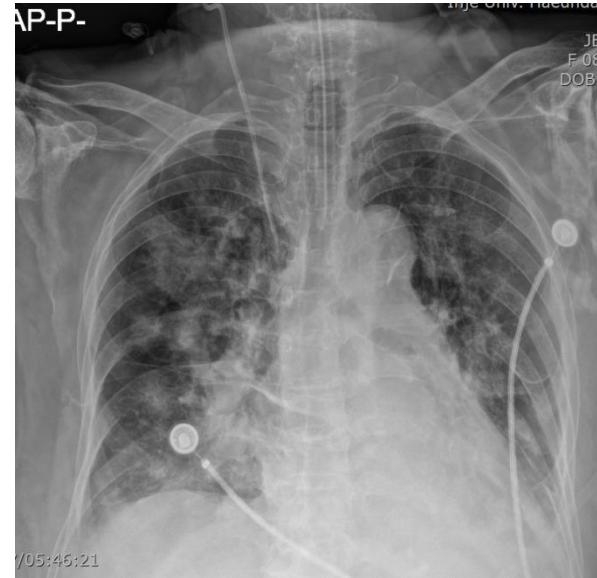
응급실 도착 직후
Intubation and MV

- Past history

HCV-liver cirrhosis (child A)

Parkinson disease
Major Depression
Hypertension

3년 전 aspiration pneumonia 로 입원 치료력

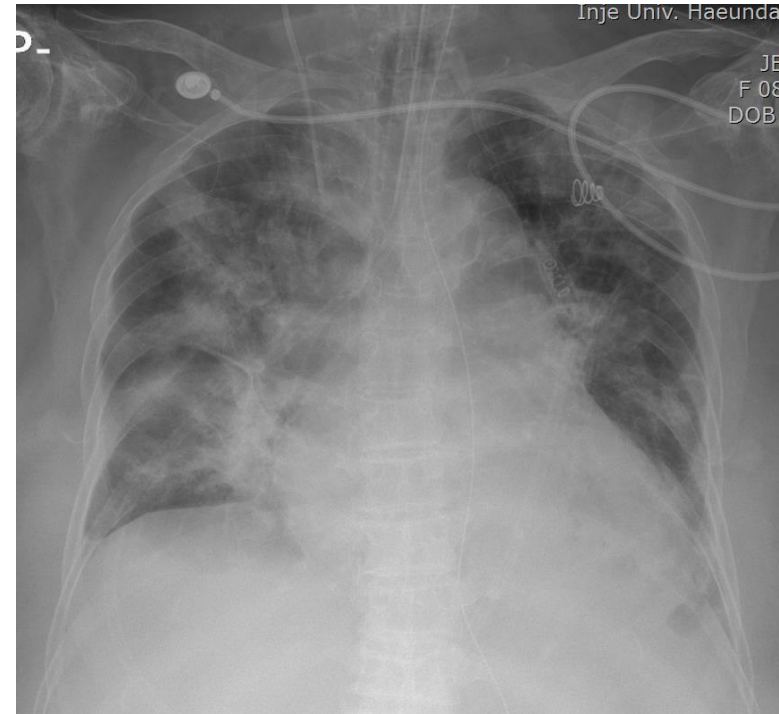
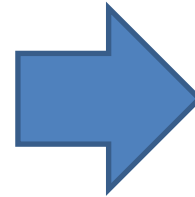
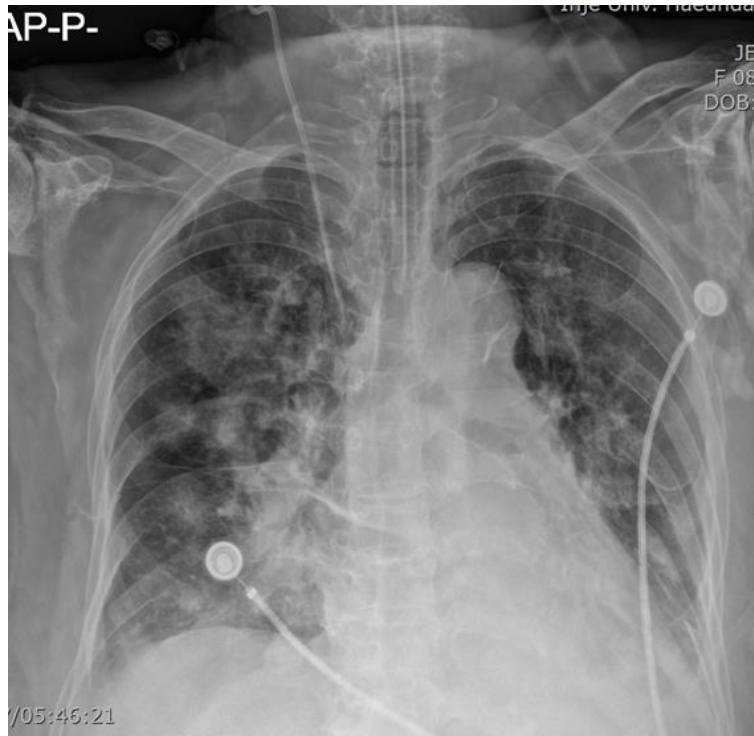


Dx: Aspiration pneumonia

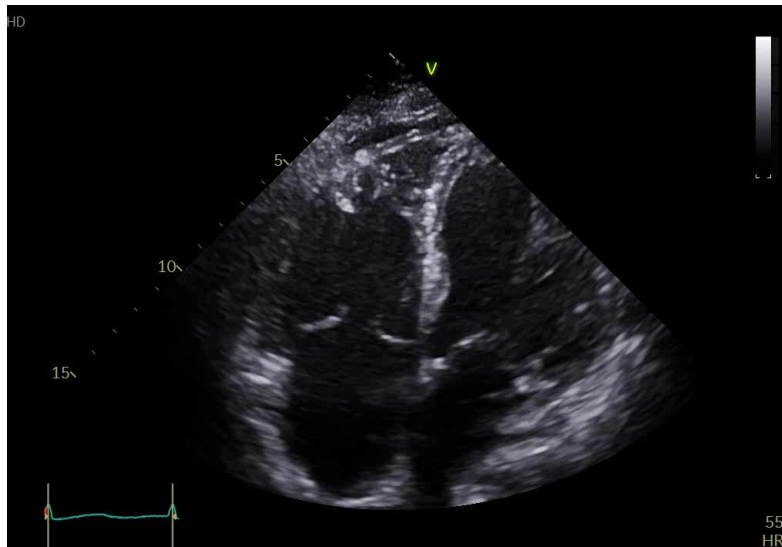
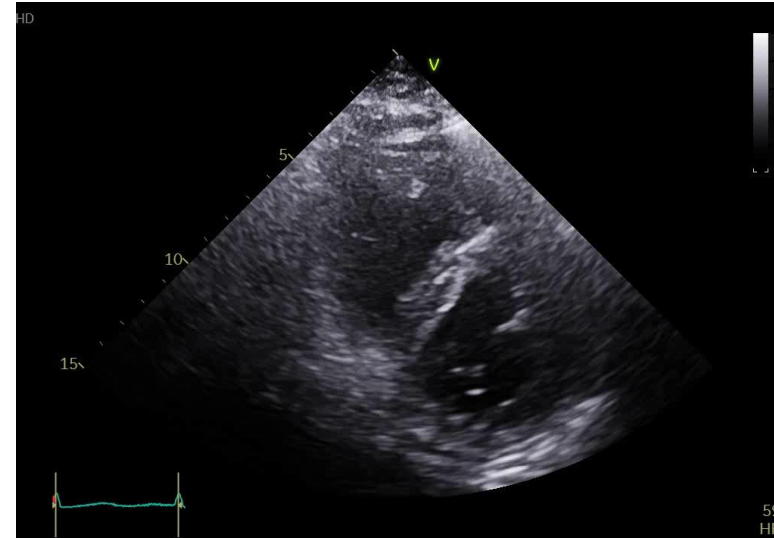
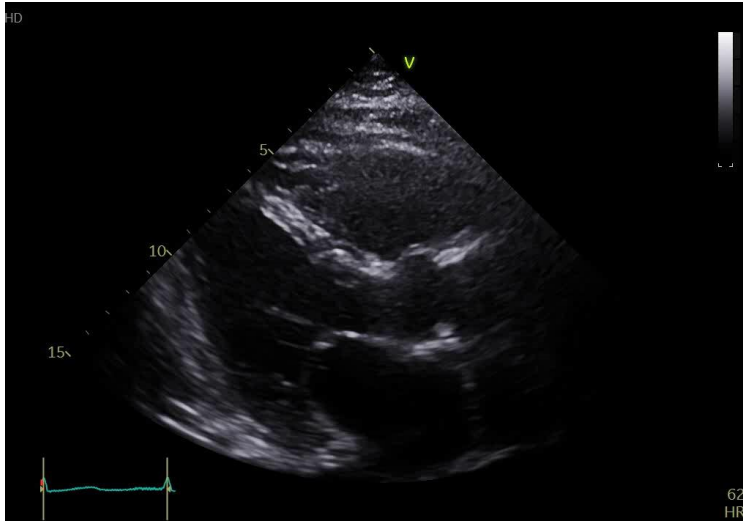


Worsening to severe ARDS
Hypoxemia despite FiO₂ 100%
High PEEP no response
Prone position no response
→ Desaturation, vital signs 악화로 보호자 임종 면회

Inhaled **nitric oxide** applied 10 ~20 ppm
Response (++) : PF ratio ↑ hemodynamic stable



Bedside Echo



Small amount pericardial effusion
IVC plethora +
No visible shunt flow

TAPSE 19.2 mm
Peak TR Vmax 2.8 m/s
RA area 24

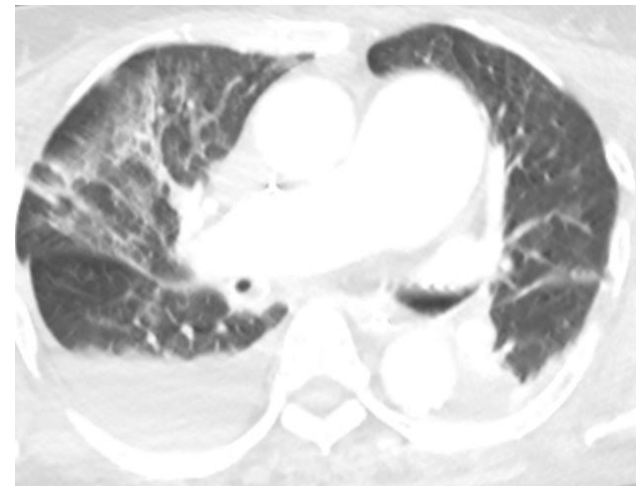
→ **Suggestive of acute RV pressure overloading.**
Recommend) embolism CT

Treatment and hospital course

- Antibiotics
- Steroid 2mg/kg/day 7 days
- Anticoagulation IV heparin
- Supportive care

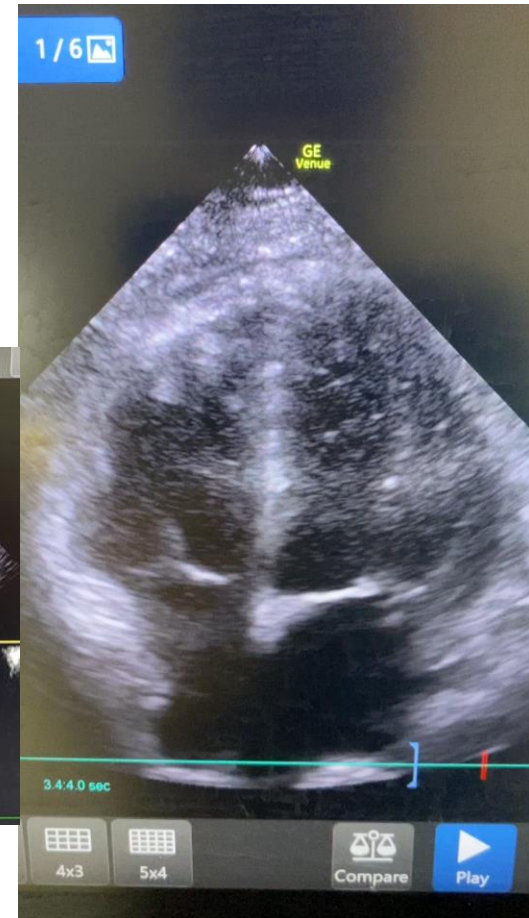
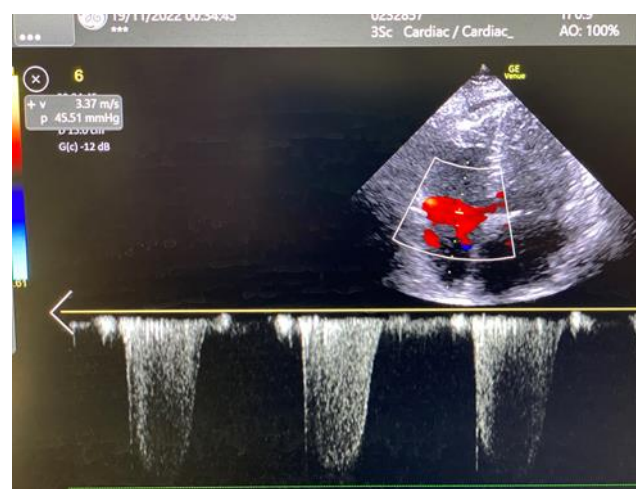
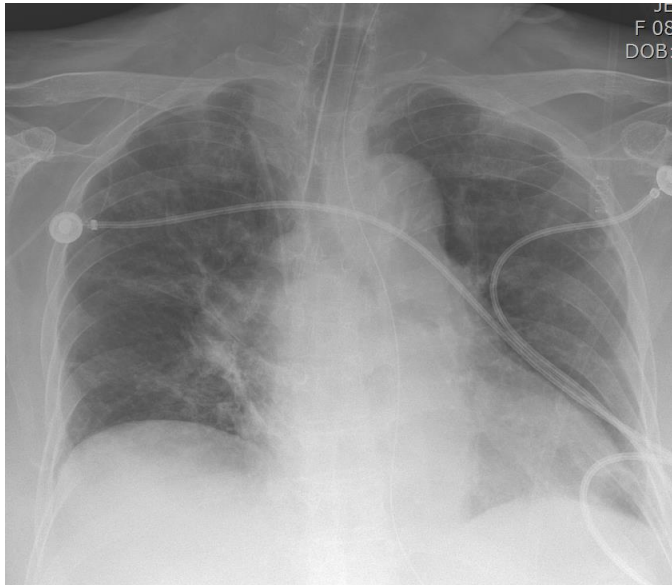
- 7일 경과 후 호전 awakening
- Pressure support ventilation
- FiO₂ 0.4

- Echo 시행 후 11일째 이동 가능하여 CTPA
No filling defect in pulmonary artery



Treatment and hospital course

- ICU 입원 31일째
On ventilator weaning
Fio2 0.3 PSV mode
Inhaled Iloprost 10mcg q6hr start



증례#1 정리

- Aspiration pneumonia ARDS
- Acute cor pulmonale
- Underlying PAH ?

HCV-LC associated portopulmonary hypertension ??

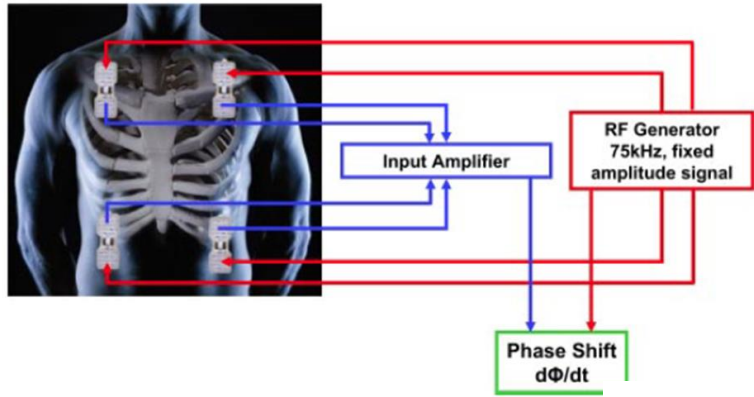


Invasive Hemodynamic monitoring

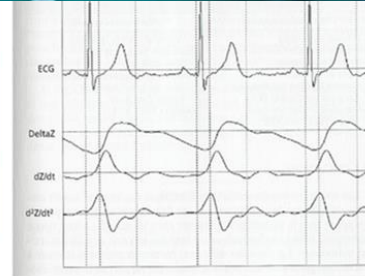
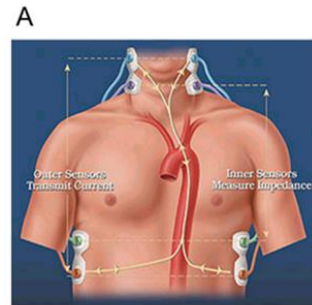
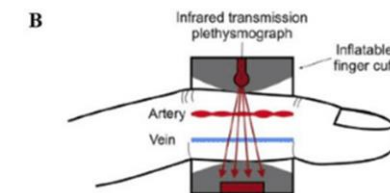
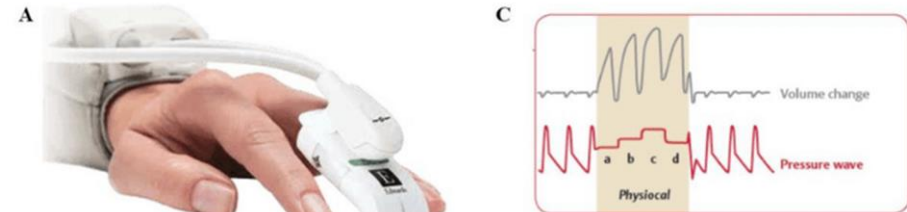
PA catheterization

Non-invasive cardiac output monitoring + bedside Echo

Bioreactance CO Monitor



ClearSight System



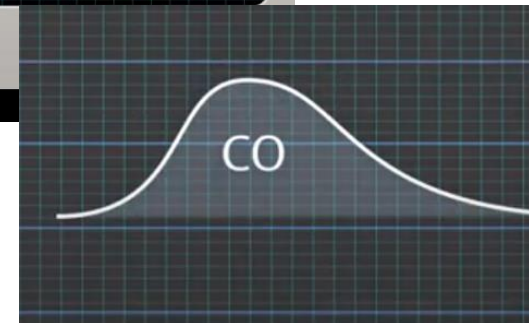
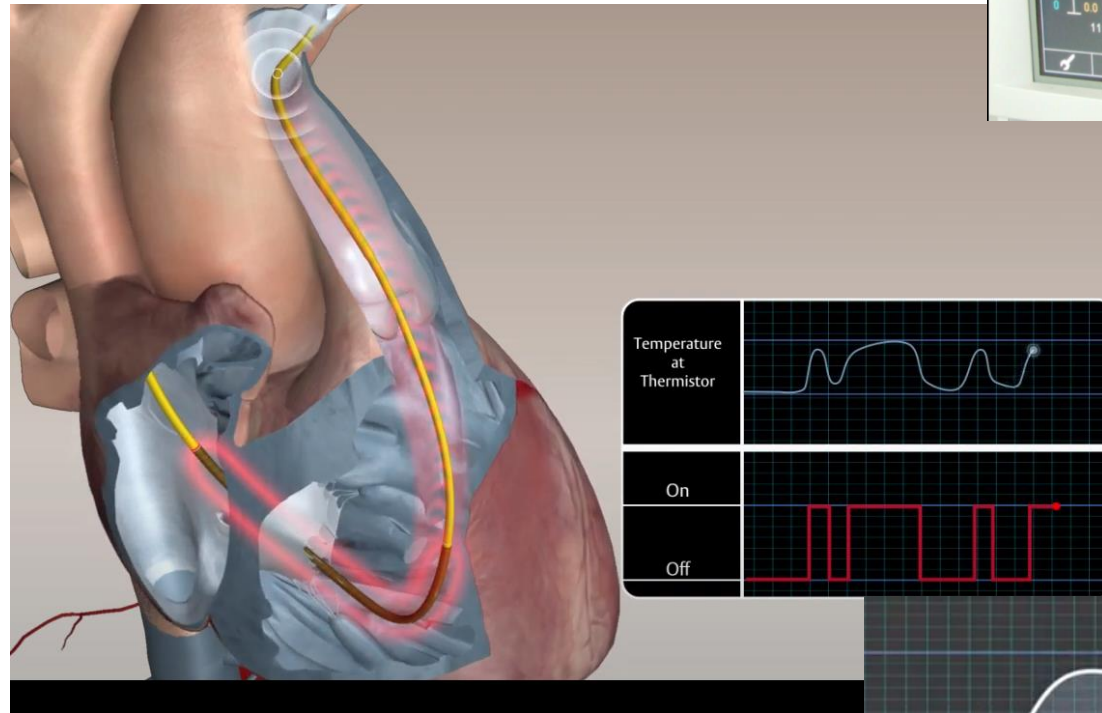
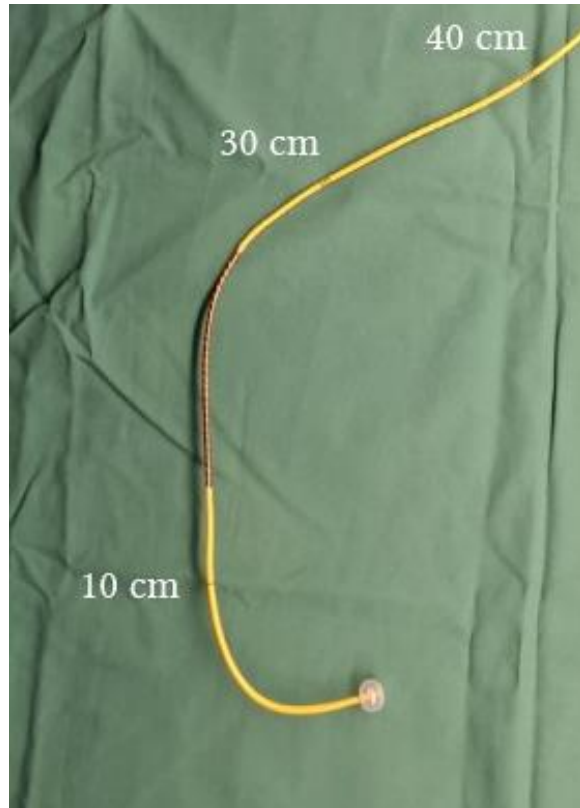
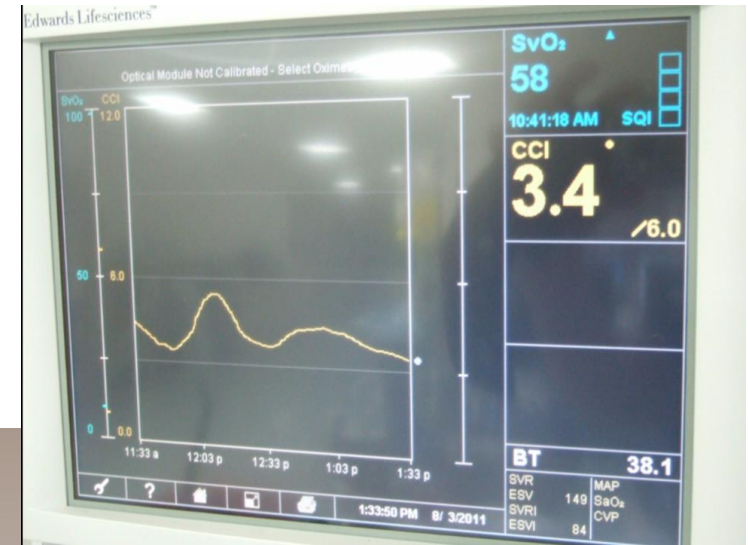
Journal and Va
Vol 28,
pp 755

Fig 1. Measurement of changes in thoracic electrical bioimpedance during a cardiac cycle. A: Placement of skin electrodes at the base thorax (emitters) and at the base of the neck (detectors). B: Recording of thoracic electrical bioimpedance changes during systole and (deltaZ) and its first (dZ/dt) and second (d^2Z/d^2t) derivatives over time.



PA catheterization in ICU

- Monitoring Continuous cardiac output



PA catheterization in ICU 아직도 필요한가?

Cardiac Profile		10:02pm	10/28/09
CCO	5.1 L/min	CCI	3.3 L/min/m ²
SVR	1166 dn-s/cm ⁵	SVRI	1798 dn-s-m ² /cm ⁵
PVR	126 dn-s/cm ⁵	PVRI	194 dn-s-m ² /cm ⁵
LVSWI	28 g-m/m ² /b	RVSWI	6 g-m/m ² /b
SV	47 mL/b	SVI	30 mL/b/m ²
MAP*	82 mmHg	HR*	109 bpm
CVP*	8 mmHg	Height	159 cm
MPAP	22 mmHg	Weight	53.9 kg
PAWP	14 mmHg	BSA	1.54 m ²

New Patient

Pulmonary artery catheterization (PAC) for adult patients in intensive care

Need to maximize fluid resuscitation
Complicated combined shock

1970년대~2000년 초반까지 ICU에서
많이 사용되었음

PA catheterization in ICU 아직도 필요한가?

- **Extreme critically ill or perioperative patients with very high risk (eg. Cardiac surgery, liver transplantation)**

Question >>

clinical or noninvasive modality 로 충분하지 않은가
PAC data 로 management 가 달라지는가?

Ex) severe septic shock with high dose vasopressor & inotropics
+ **stress induced cardiomyopathy** + **RV dysfunction**
ARDS on high PEEP

Fluid responsiveness

CO 올리고 vasopressor 줄이고

Septic shock due to Septic knee, lactic acidosis

Dobutamine, High dose vasopressor infusion
(norepinephrine, dopamine, vasopressin)

ARDS ventilator PEEP 16 cmH2O

CO 4.5 L/min PAP 47/23 PCWP 17

→ Normal saline 500ml full drip infusion

CO 5.2 L/min PAP 48/23 PCWP 18

→ Normal saline 1000 ml full drip infusion

CO 6.5 L/min PAP 48/23 PCWP 18

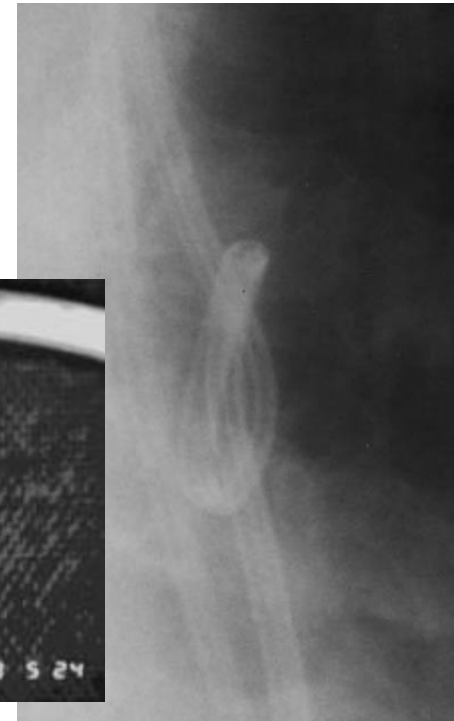
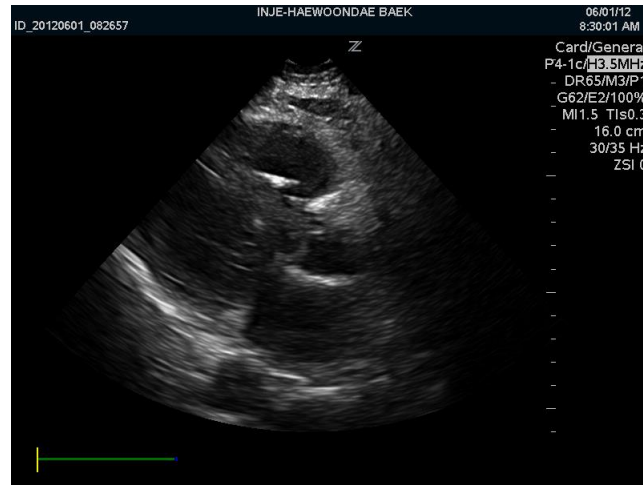
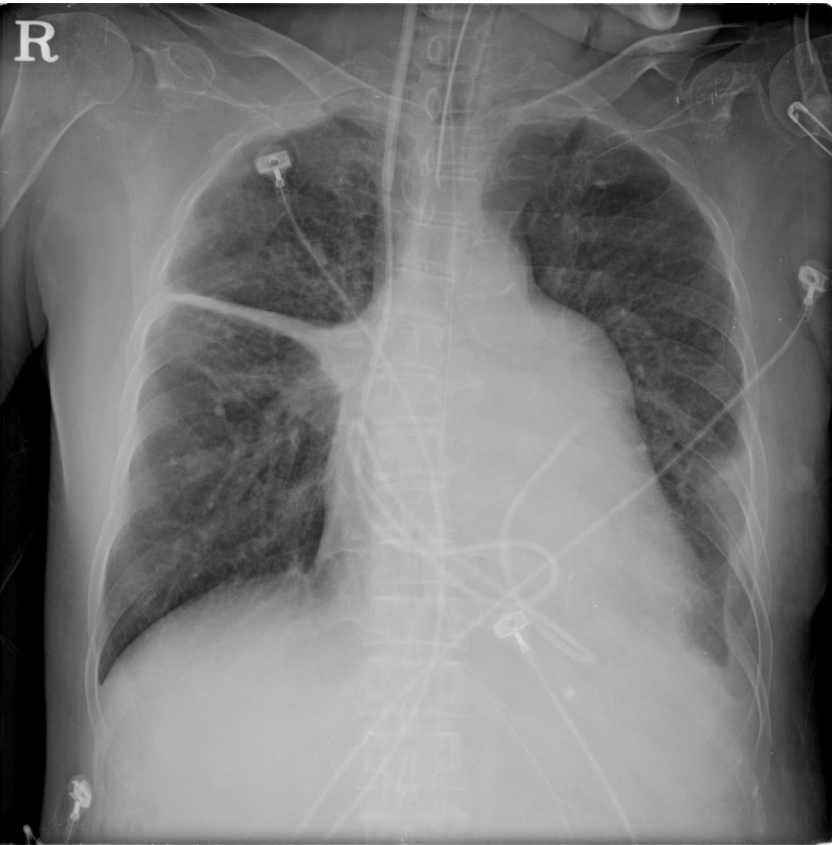
→ Normal saline 1000 ml infusion

CO 7.0 L/min PAP 50/25 PCWP 20



Complication of PA catheterization

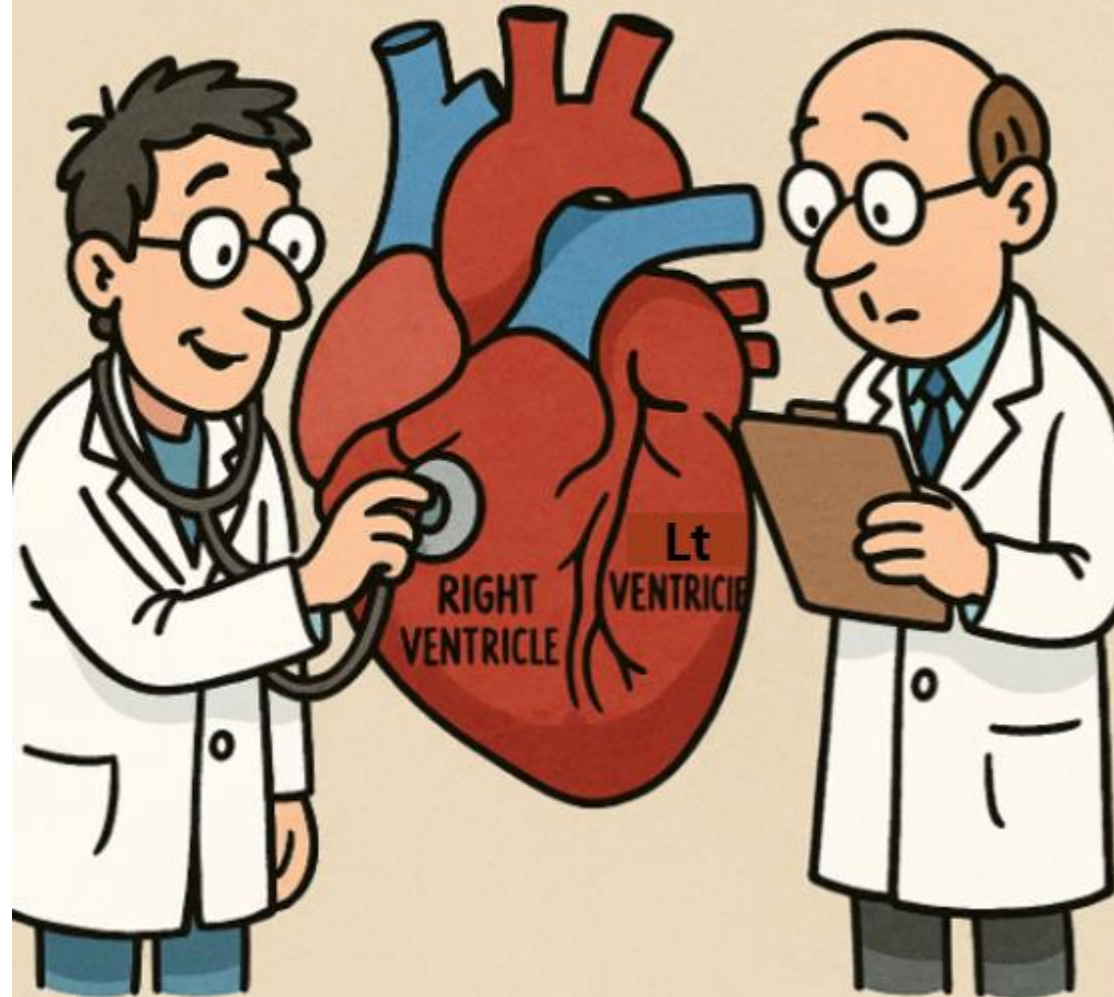
- Depend on experiences and skill of physician

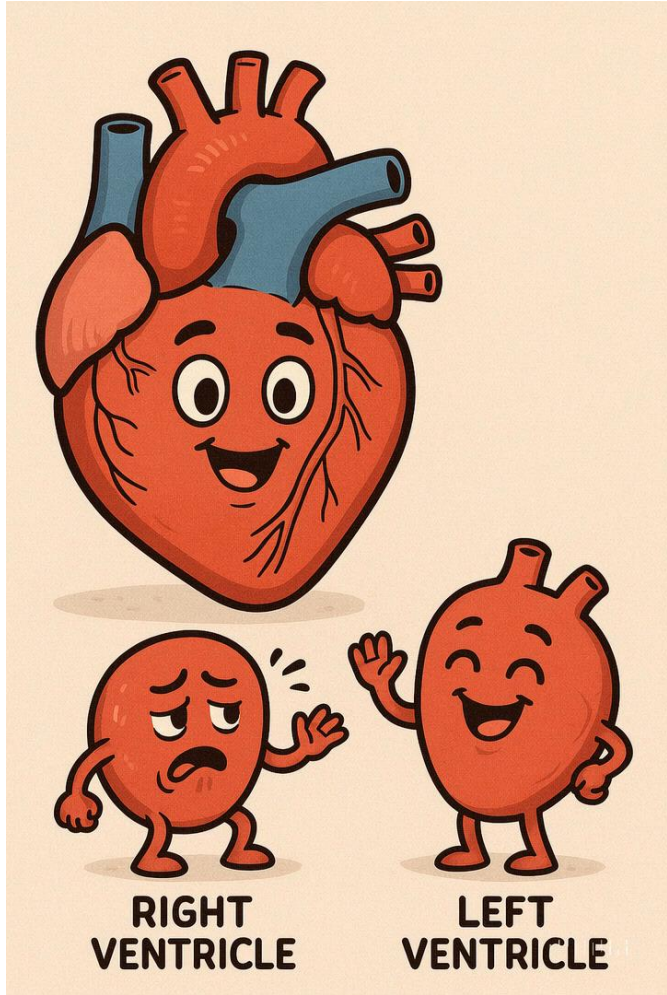


Torsten B. et al, J Card Surg 2006;21:292-295

PULMONOLOGIST

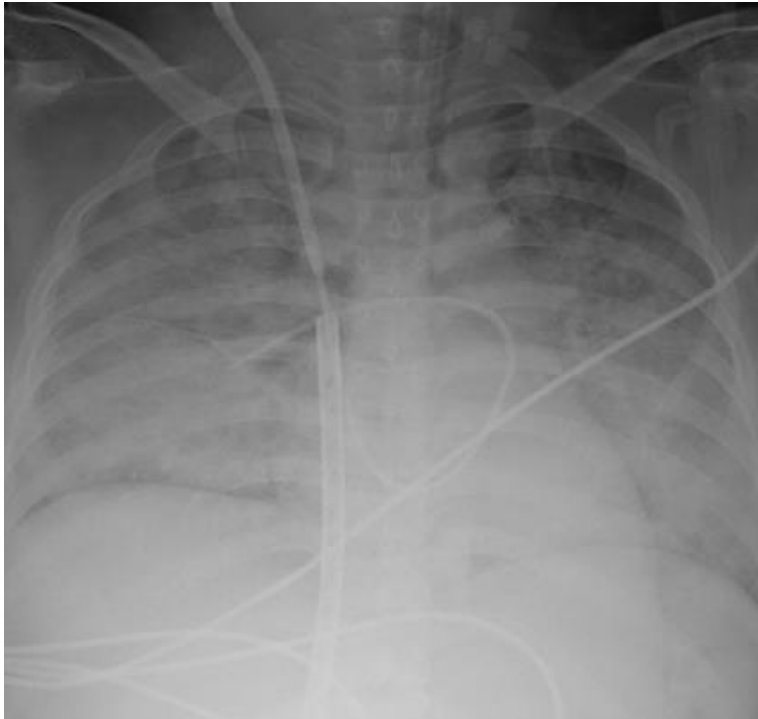
CARDIOLOGIST



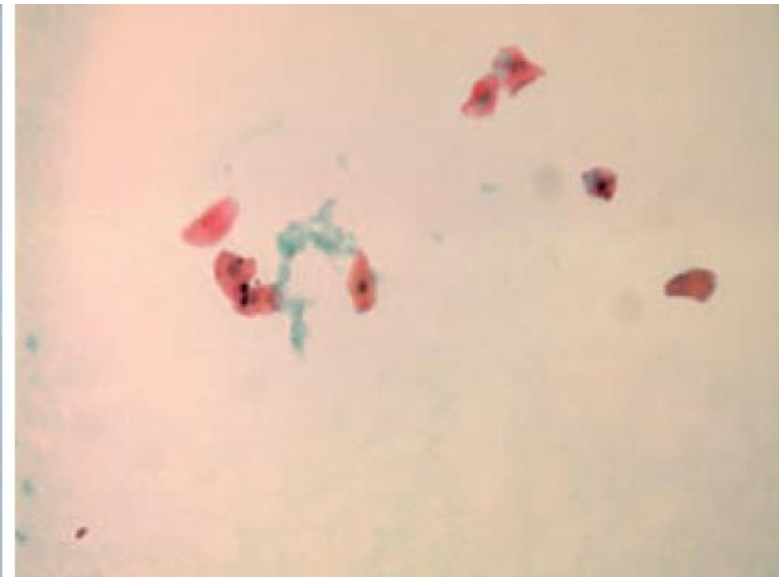
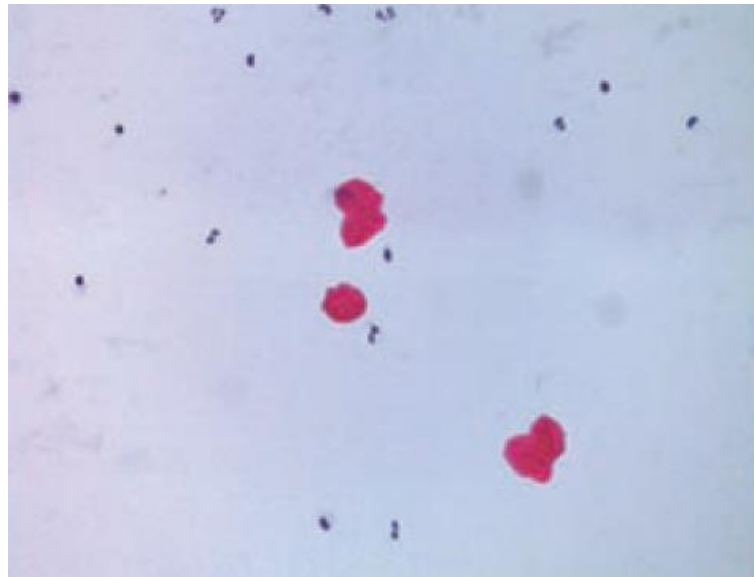


Suspected **amniotic fluid embolism**

-

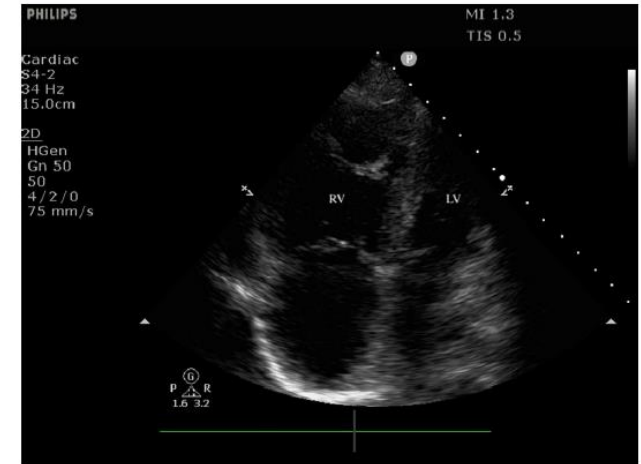
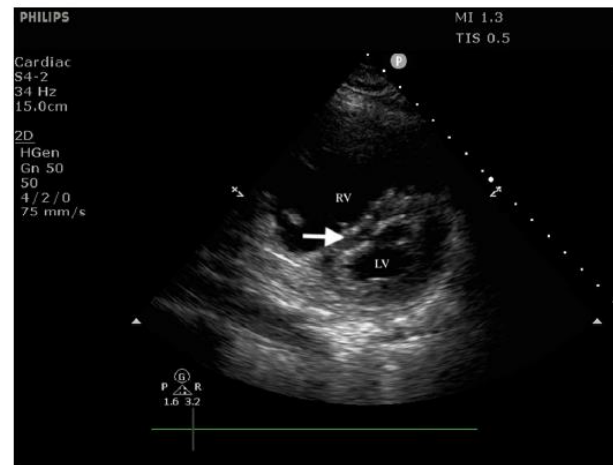


Sudden cardiac arrest after delivery
on V-A ECMO
following ARDS
stress induced cardiomyopathy

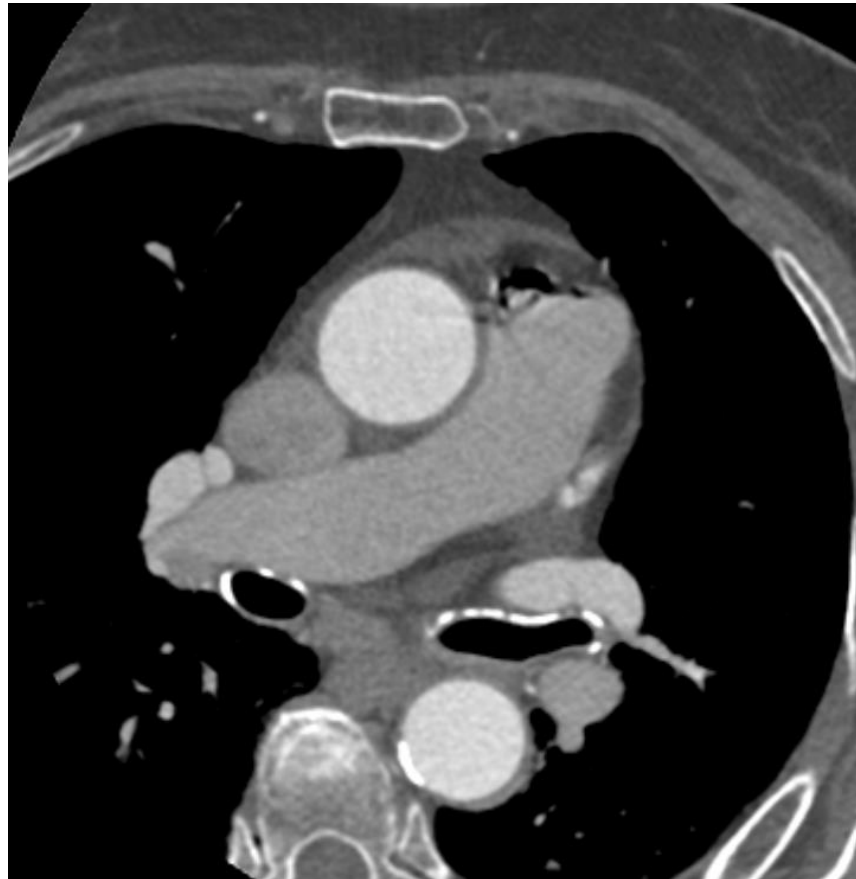


Hemodynamic response - **amniotic fluid embolism**

- Biphasic,
- initial severe pulmonary hypertension and right ventricular failure,
- → followed by LV failure



Transient RV dysfunction – Venous Air embolism



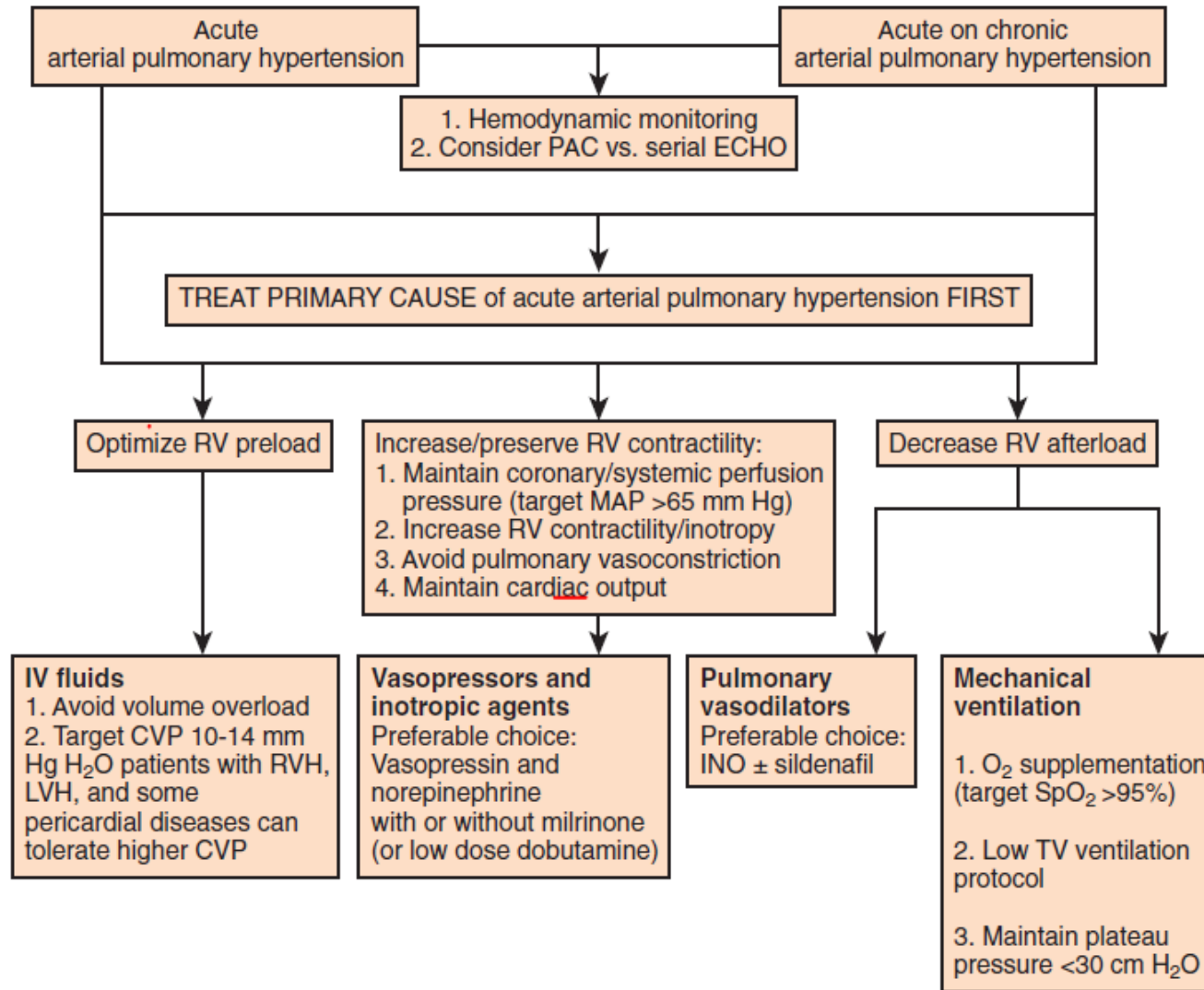


Figure 45.3 Therapeutic approach to pulmonary hypertension in the intensive care unit. CVP, central venous pressure; ECHO, echocardiogram; INO, inhaled nitric oxide; IV, intravenous; LVH, left ventricular hypertrophy; MAP, mean arterial pressure; PAC, pulmonary artery catheter; RV, right ventricle; RVH, right ventricular hypertrophy; SpO₂, oxygen saturation in the blood; TV, tidal volume. (From Tsapenko MV: Arterial pulmonary hypertension in noncardiac intensive care unit. Vasc Health Risk Manage 2008;4:1043-1060.)

Fluid overload
Shunt (ASD)

Ischemia
Sepsis
acidosis

Embolism
LV failure
Mechanical
ventilation

Contractility
↓

Preload ↑

Afterload ↑

**RV
distension**

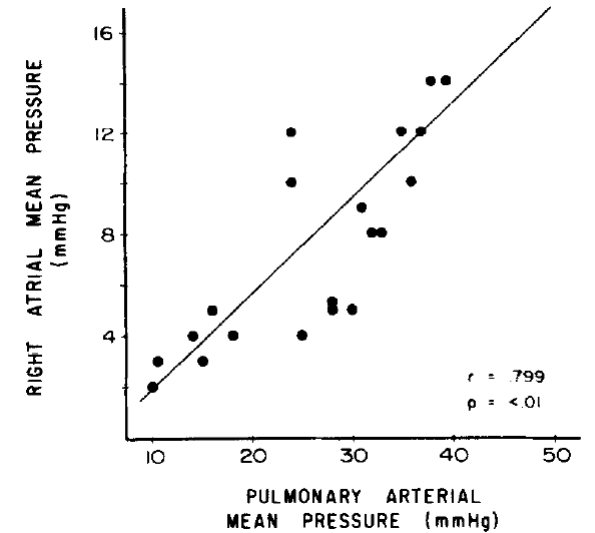
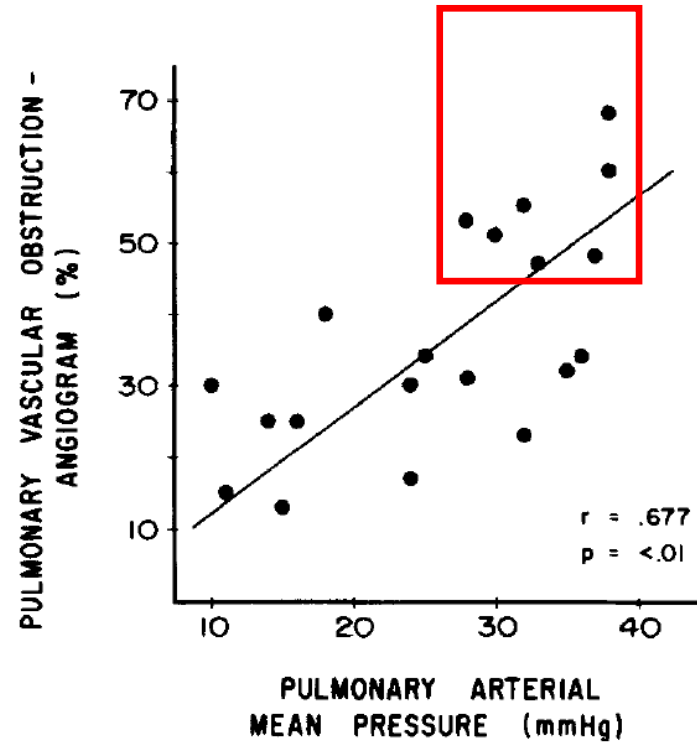
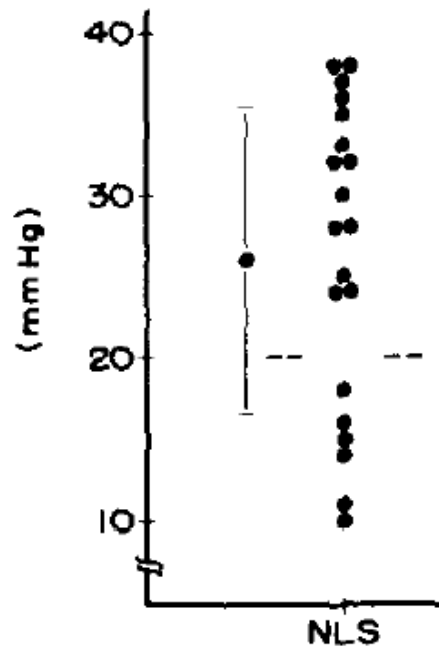
Pulmonary Hypertension in **acute pulmonary embolism**

- Angiographic obstruction of PA **>50%** → PAP ↑
- “Acute PE never resulted in **mean PAP > 40 mm Hg** in previously healthy patients”
- maximum upper limit that a non-adapted right ventricle can generate when exposed to an acutely increased afterload.



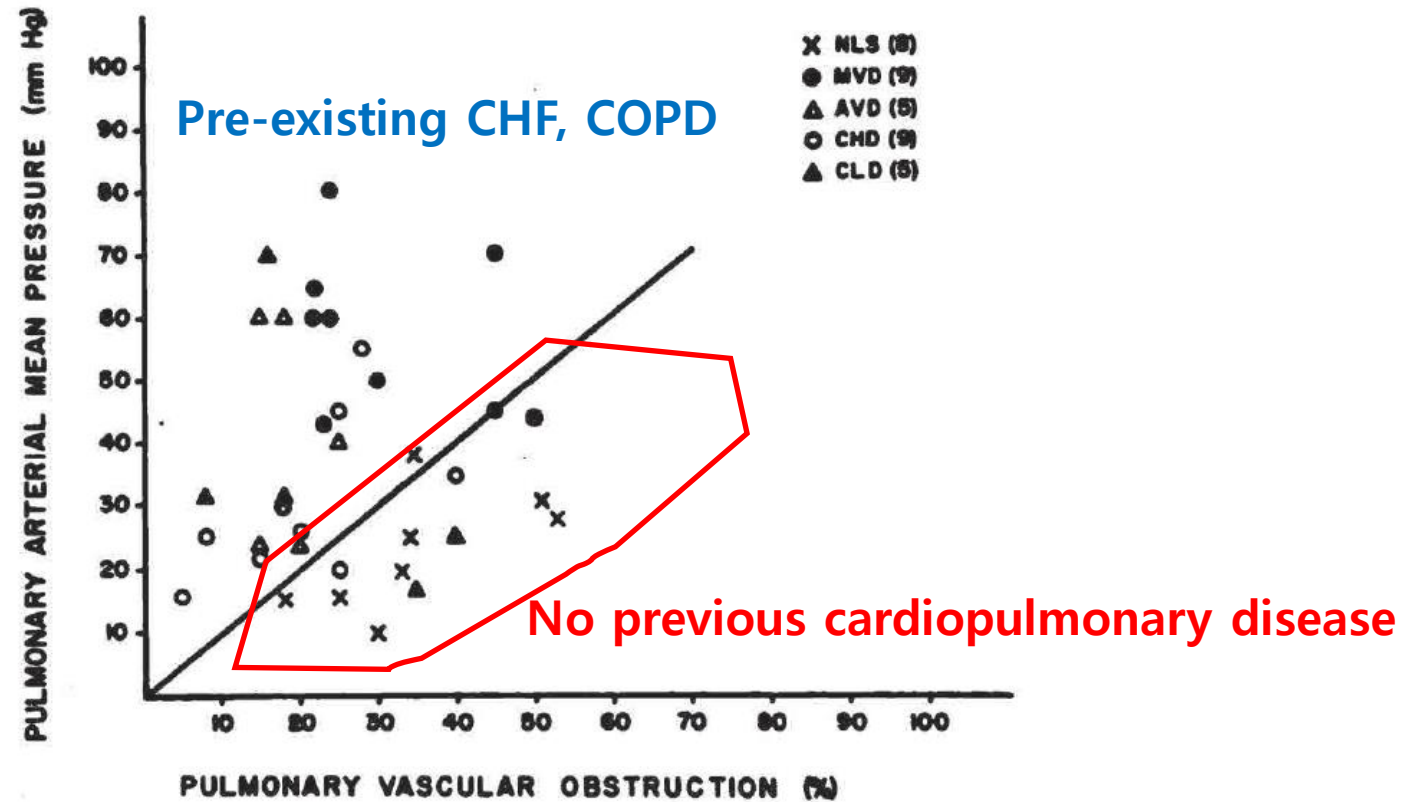
Hemodynamic response to pulmonary embolism

- Included 20 patients previously healthy

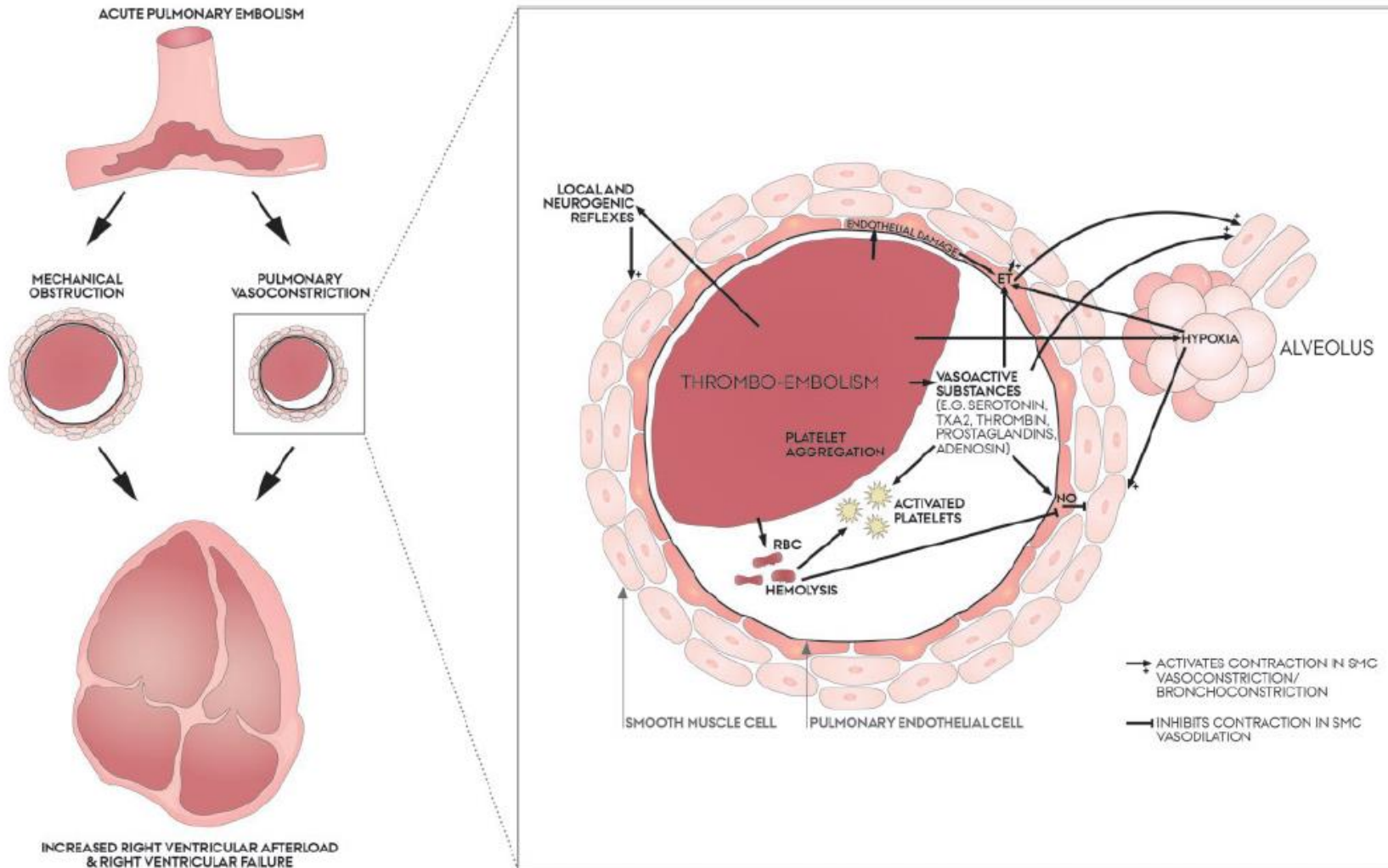


The ratio of PAP to pulmonary vascular obstruction

: With or without previous cardio-pulmonary diseases



Pulmonary Hypertension in acute pulmonary embolism



Pulmonary vasodilation in acute pulmonary embolism systemic review

- **Inhaled Nitric oxide** → may improve the hemodynamic status

Summary of Outcomes From Analyzed Patients			
Improvement in MAP/CO	Improvement in PAP/PVR	Improvement in PaO ² / SpO ²	Survival From ICU
14/14 who reported (100%)	6/7 who reported (86%)	16/18 who reported (89%)	14/18 (78%)

CO, cardiac output; ICU, intensive care unit; MAP, mean arterial pressure; PaO²: partial pressure of oxygen; PAP, pulmonary arterial pressure; PVR, pulmonary vascular resistance; SpO²: percutaneous oxygen saturation.

- PDE-5 inhibitors
- sCG stimulators/activators
- Prostacycline
- ERA



No recommendation