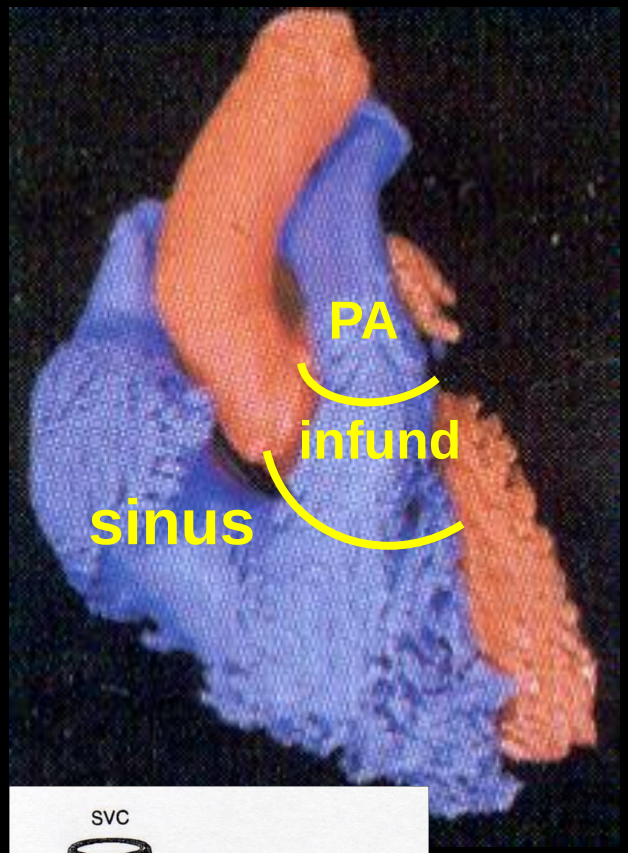
ECOCARDIOCHIRURGIA.it

Milano, 17 Ottobre 2012

La gestione dell'insufficienza ventricolare destra nel paziente sottoposto ad intervento cardiochirurgico

Emanuele Catena
Direttore SC Anestesia e Rianimazione

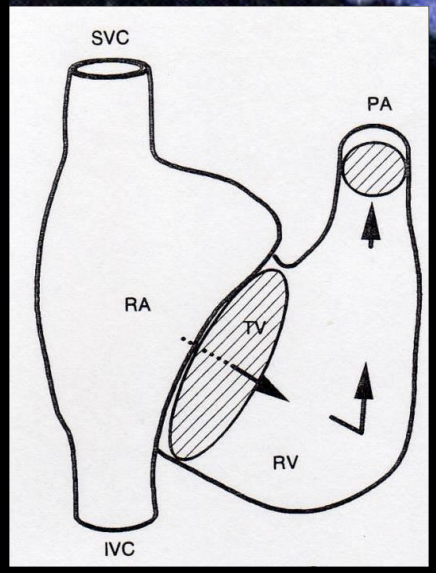
AZIENDA OSPEDALIERA CARLO POMA



Three components of the RV:

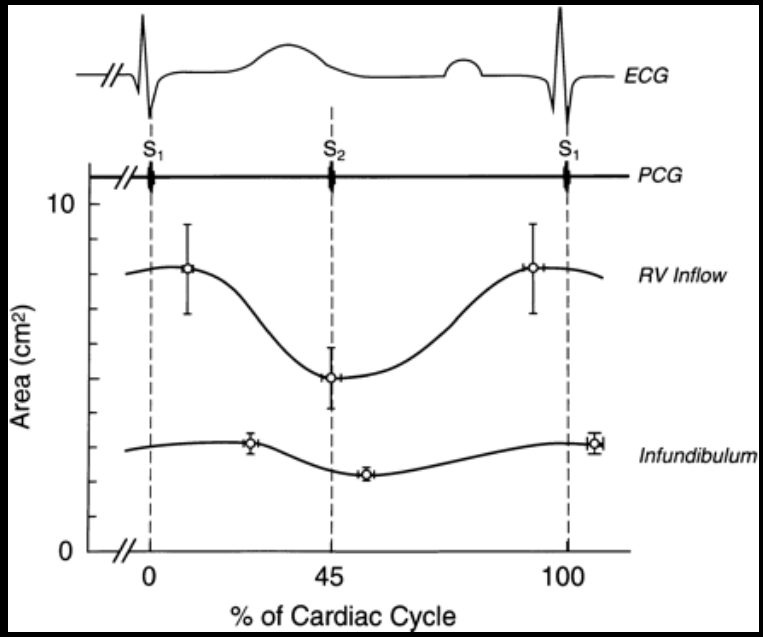
1) The inlet or sinus
2) (the trabeculated apex)
3) The infundibulum or conus

"U" shape

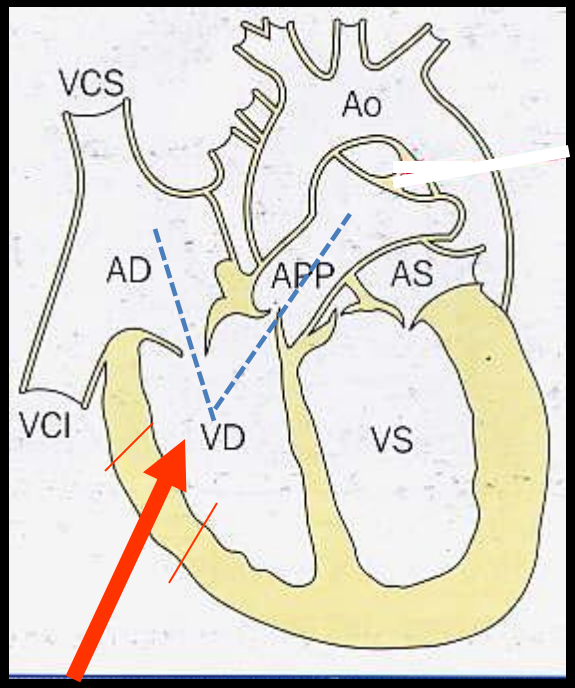


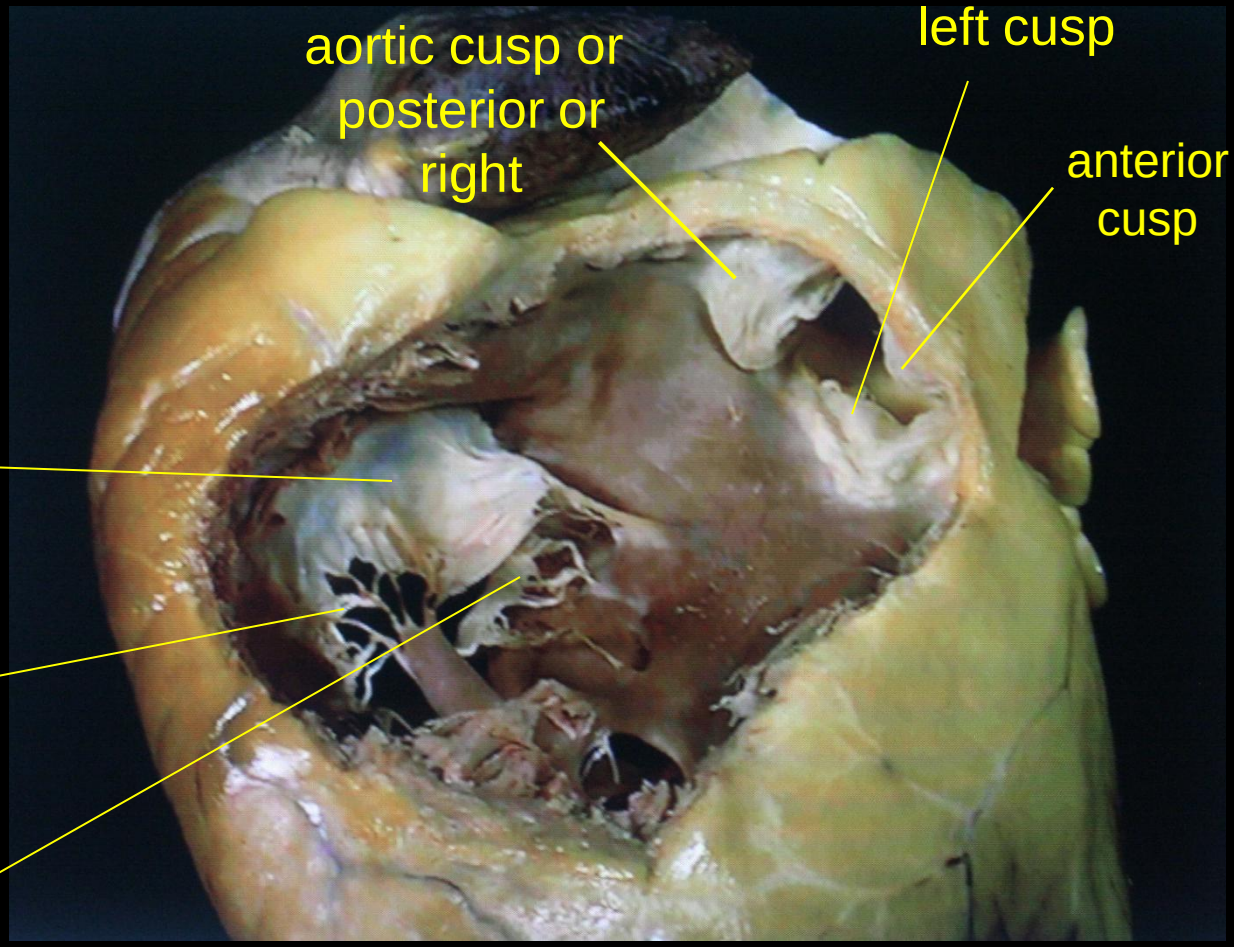
"peristaltic contraction"

Sinus supports 80-85% pump function



RV: complex geometry





RV coronary artery flow

- Right coronary artery supplies blood flow

  - to *RVOT* through the conus artery
  - to the *RV lateral wall* through the acute marginal branches
  - to the *posterior wall and IVS* through the posterior descending artery

- Left anterior descending artery supplies *RV anterior wall* through small branches

Extensive RV myocardial necrosis is associated with proximal RCA occlusions but many RCA occlusions do not result in significant RV dysfunction

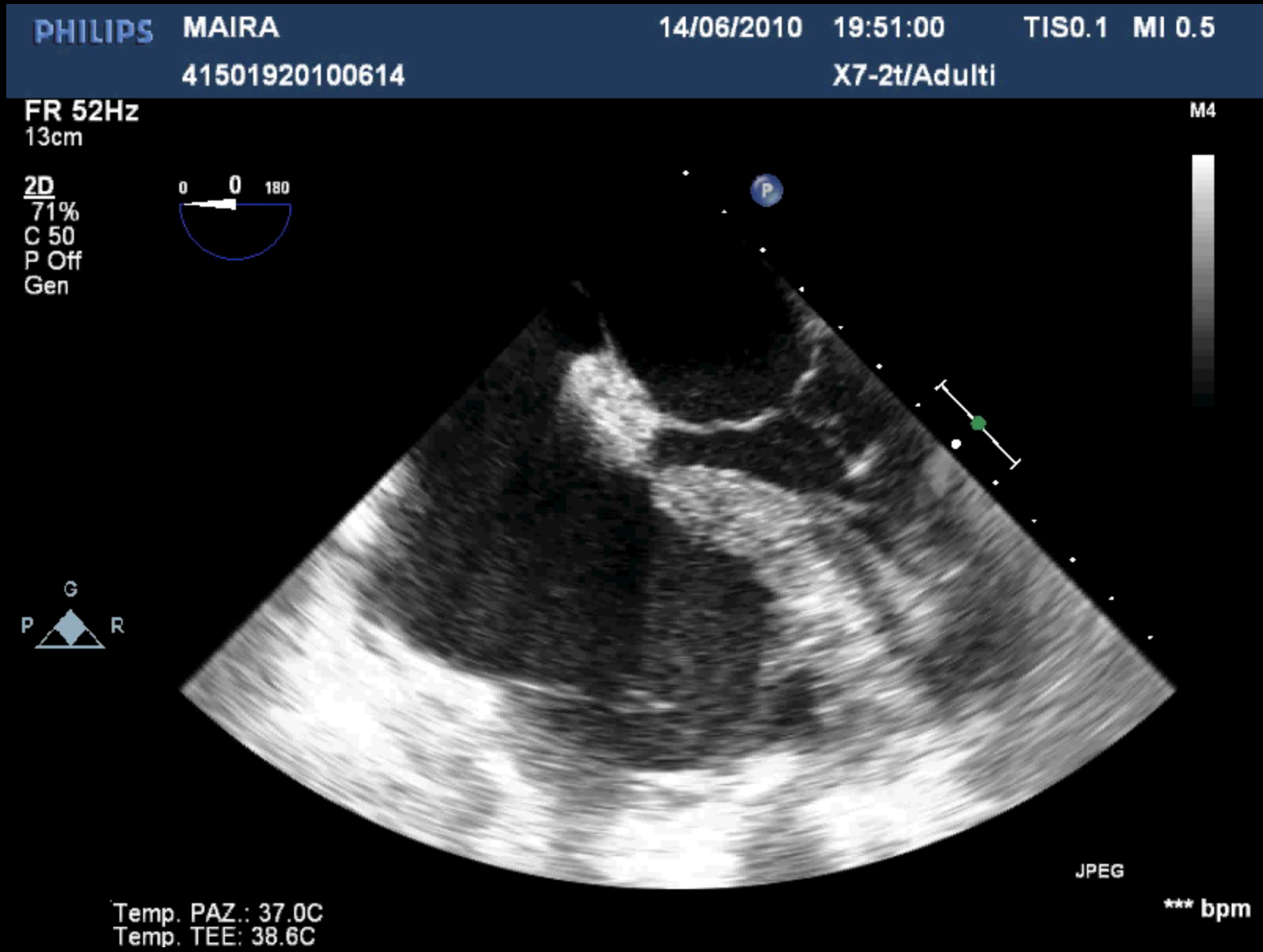
Protective factors

- Low myocardial O2 demand
- Greater systolic to diastolic flow ratio
- Ability to extract O2
- Extensive anatomic collaterals and Thebesian veins

Adverse factors

- RV hypertrophy. It increases O2 demand and may resulr in more ischemic injury during acute RCA occlusion

Guarracino F. et al. Minerva anestesiol 2005;71:307-12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41501920100614
X7-2t/Adulti
FR 52Hz
13cm
M4
2D
71%
C 50
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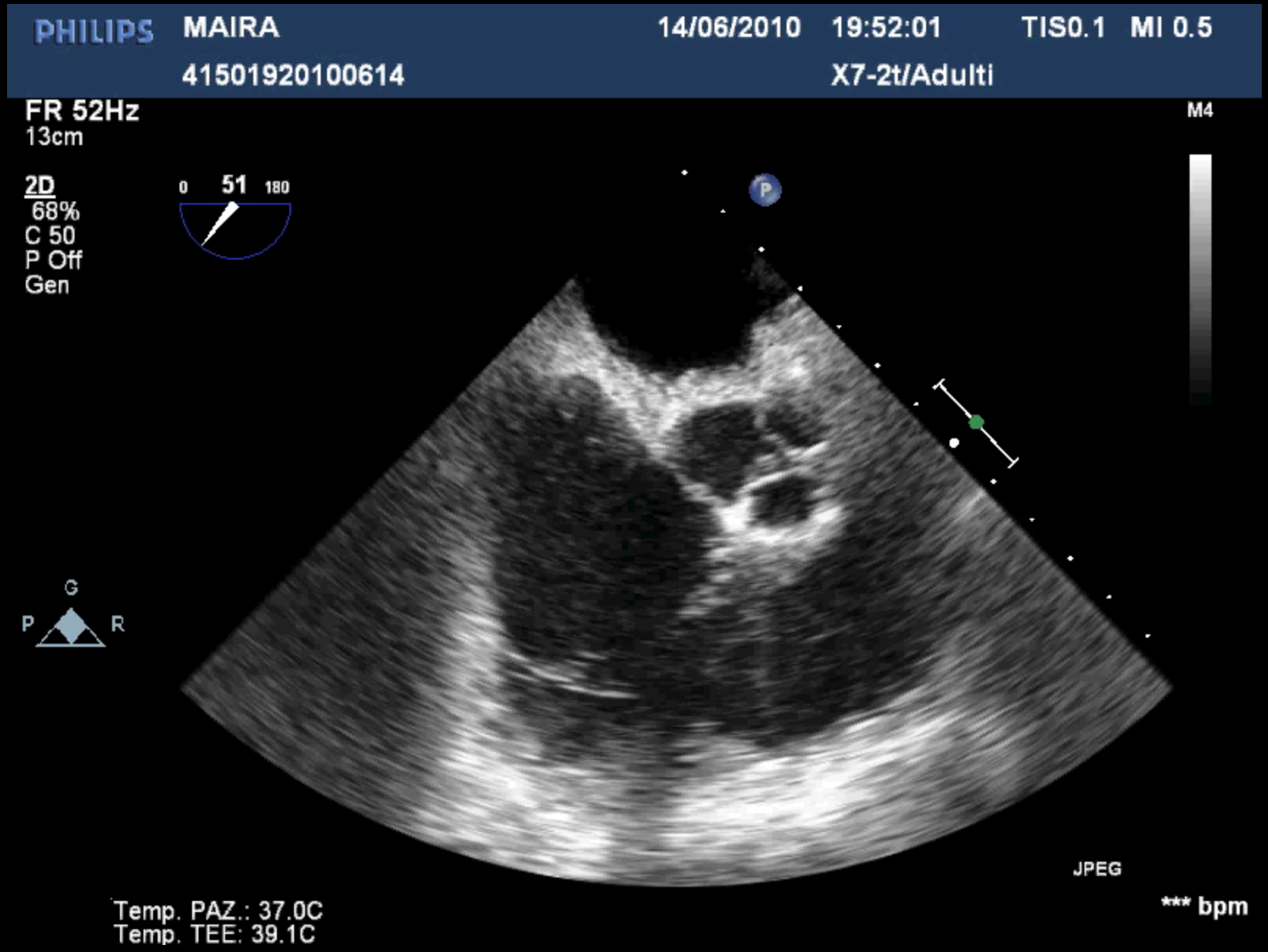
Temp. PAZ.: 37.0C
Temp. TEE: 38.6C
*** bpm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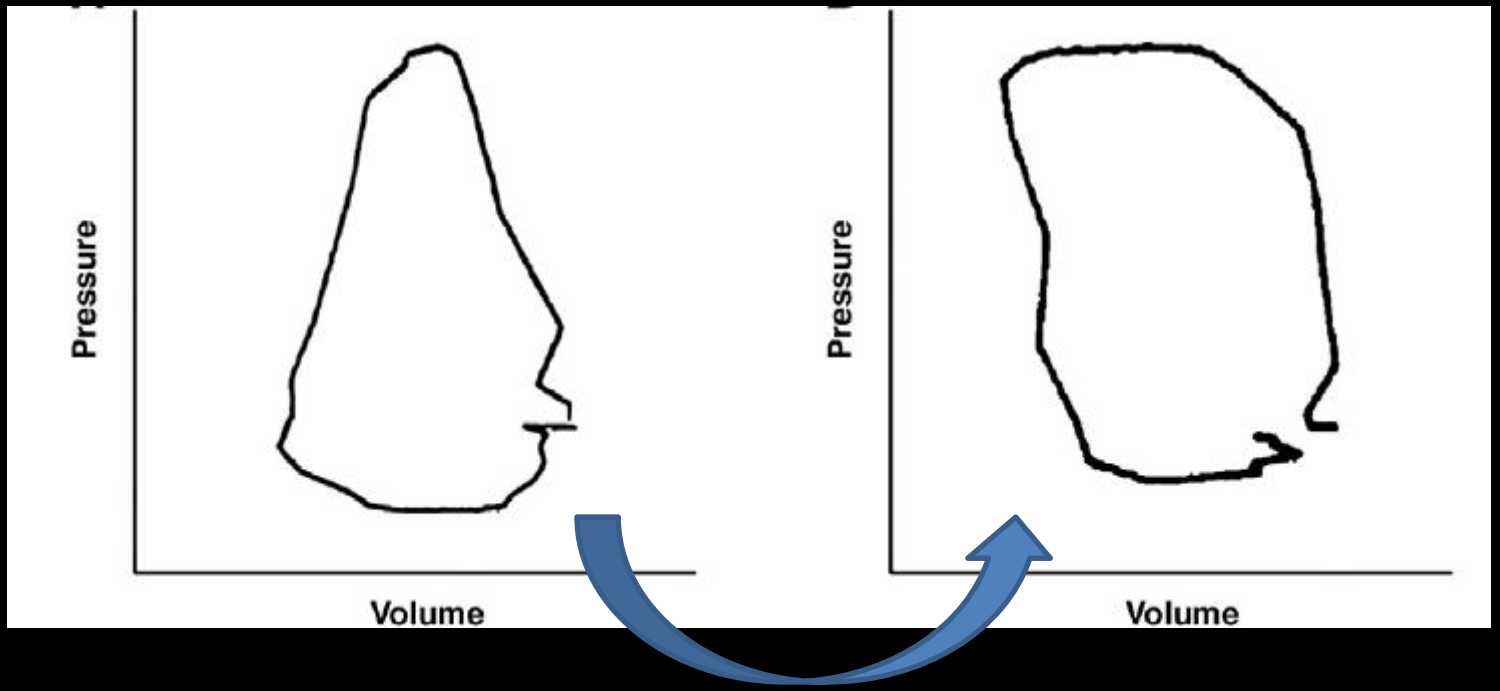
*** bpm

Physiology and pathophysiology of RV function

- Under normal conditions, the RV is coupled to the highly compliant pulmonary vascular system, which renders it a *volume pump* (low pressure) rather than a pressure pump.
- The RV is *more sensitive to an afterload change* compared to the LV. A similar increase in afterload to the RV (PAP) and LV (aortic pressure) leads to a significantly greater decrease in SV for the RV compared to the LV.
- In contrast, the *RV tolerates and adapts more easily to volume (diastolic) overload.*
- The RV has a greater end-diastolic volume than the LV, so the RV ejection fraction is less (RVEF 40–45%) than the LV (LVEF 50–55%).

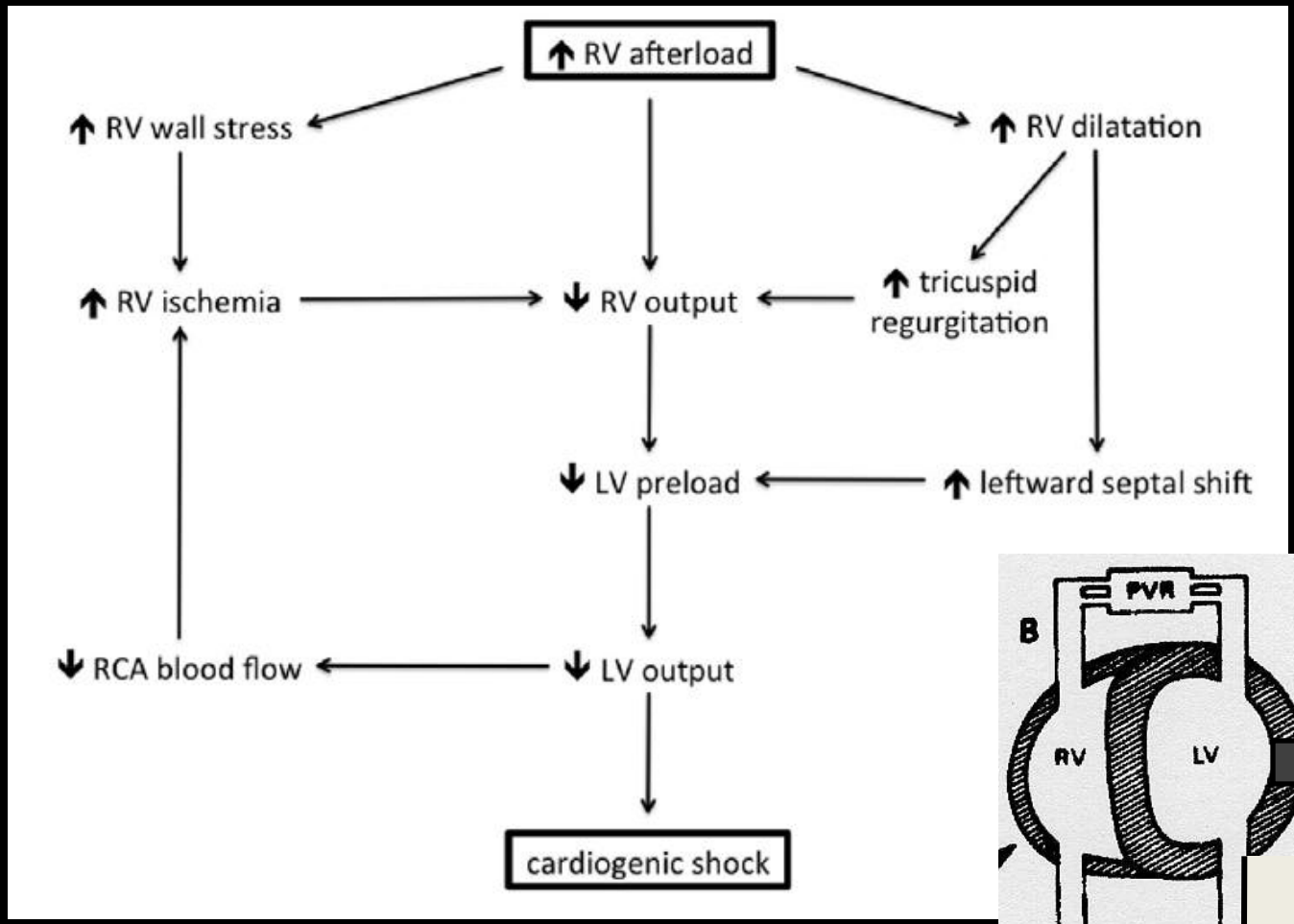
The RV has only brief periods of isovolumic contraction and relaxation



Increases in RV afterload lead to the development of prolonged periods of isovolumic contraction and relaxation, ultimately resulting in a decline in RV performance

Hooman D. Poor, et al Prog Cardiovasc Dis 2012;187-198

Pathogenesis of RV failure secondary to increased RV afterload:

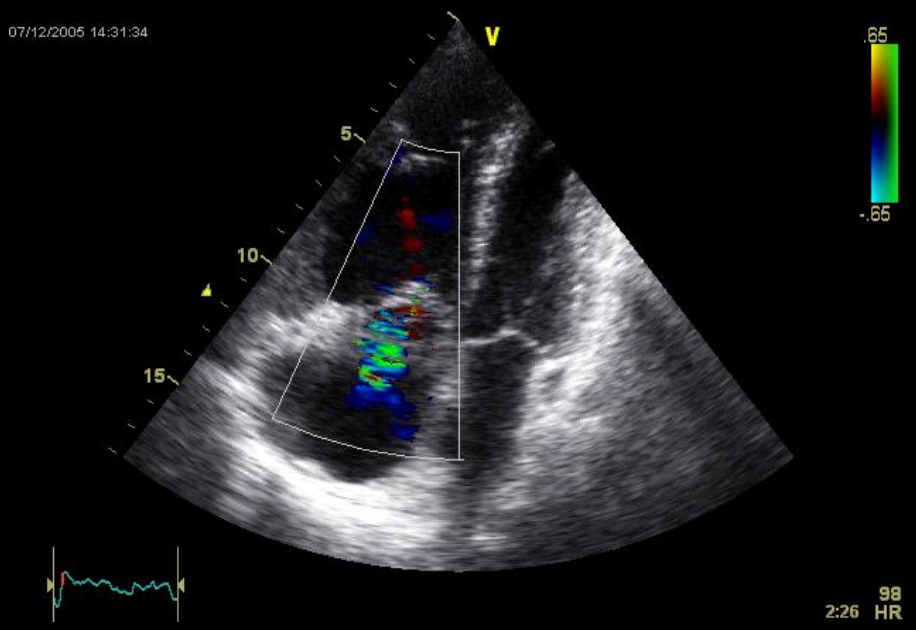


Hooman D. Poor, et al Prog Cardiovasc Dis 2012;187-198

Acute increase in afterload:

SEPTAL FLATTENING Or SHIFT

⬇ RVFAC
< 32%



RV DILATION
RVEDA/LVEDA>0.6-1

UNDEFILLED LV

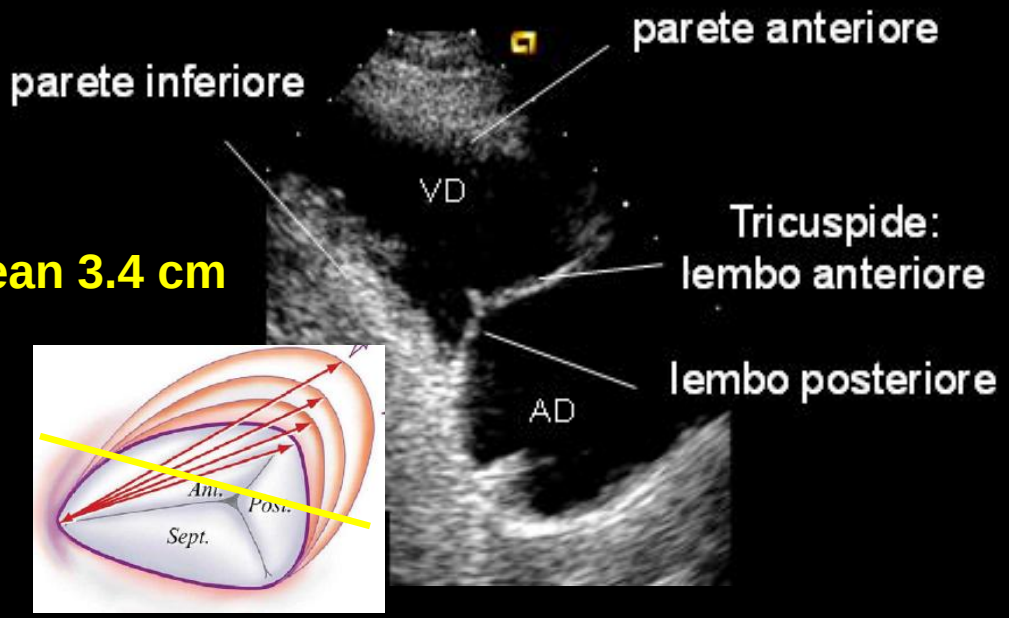
⬇ TAPSE
< 14 mm

Right-to-left SHUNT

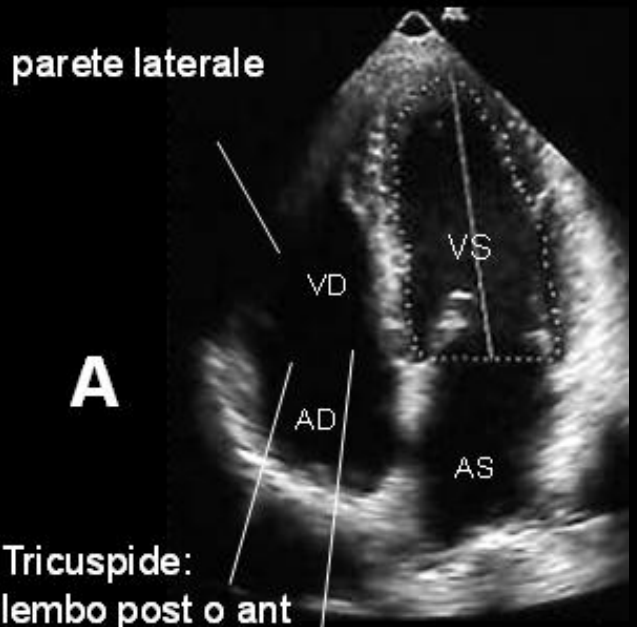
⬇ Sm
< 10 cm/s

Itr
+++/IV

Tricuspid annulus

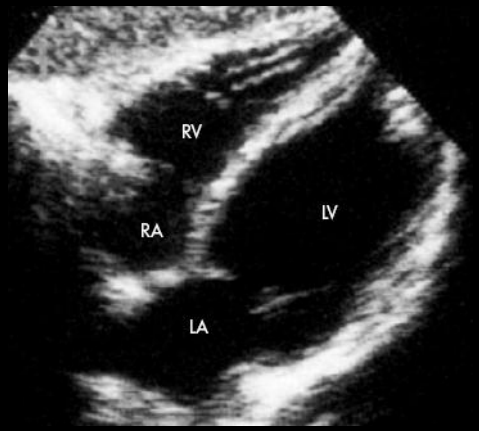


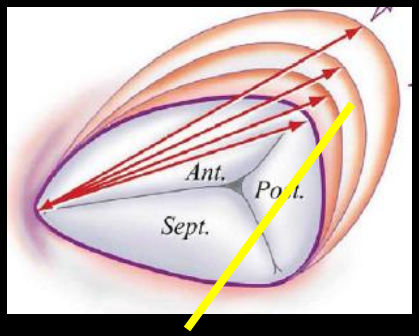
mean 3.4 cm

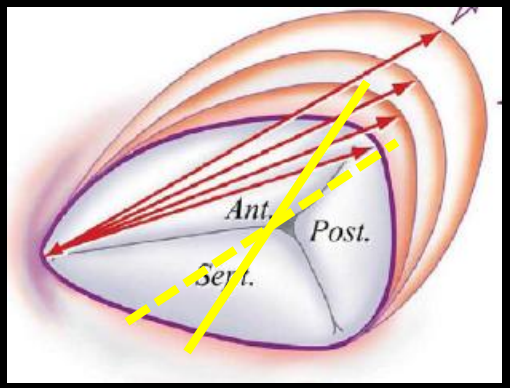


Mean 2.4 cm

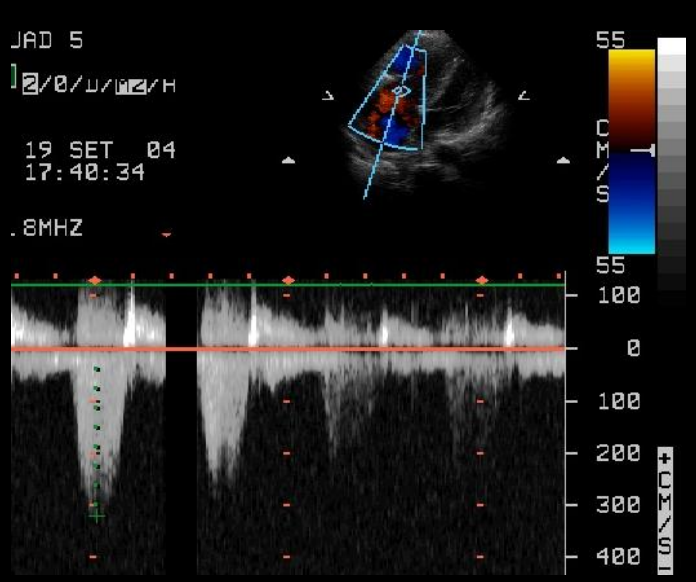
Tricuspide:
lembo settale





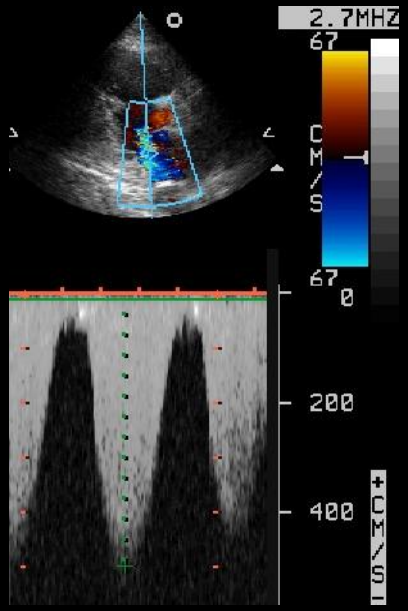


Acute or chronic disease?



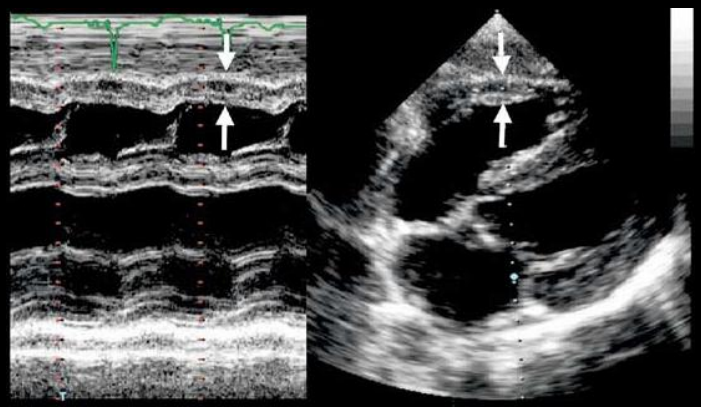
V max 3.2 m/s

P max 41 mmHg



V max 5 m/s

P max 100 mmHg



Patients may be predisposed to RV dysfunction because of pre-existing conditions:

- right coronary artery disease
- RV infarction
- Pulmonary hypertension (mitral/aortic disease, left-to-right shunts, diastolic LV dysfunction, embolism, heart transplantation, LVAD)

RV dysfunction may occur in patients with no known pre-existing problems:

- Poor myocardial protection
- Prolonged ischemic times/myocardial stunning
- Coronary embolism of air
- Systemic hypotension and RV ischemia
- Acute pulmonary hypertension (blood product tranfusions, CPB, LV dysfunction, protamine reaction, hypoxia, acidosis)
- RV pressure overload: intrinsec pulmonary disease
- Tricuspid valve surgery

Pulmonary hypertension

Pulmonary hypertension is defined as an elevated mean pulmonary artery pressure

> 25 mmHg

MILD: 25-40 mmHg

MODERATE: 41-55 mmHg

SEVERE: > 55 mmHg

Pulmonary hypertension results from an increase in:

1) Resistance to blood flow within the pulmonary arteries (pulmonary vascular resistace, PVR)

   "pre-capillary": PVR ≥ 3.0 WU, WP ≤ 15 mmHg , TPG ≥ 12-15 mmHg

2) Pulmonary venous pressure from left heart disease

   "post-capillary": WP > 15 mmHg, TPG < 12 mmHg

3) "mixed": pulmonary hypertension is out of proportion to the degree of WP elevation, due to arterial vasoconstriction and vascular remodeling. WP > 15 mmHg, TPG ≥ 12-15 mmHg, PVR ≥ 3.0 WU

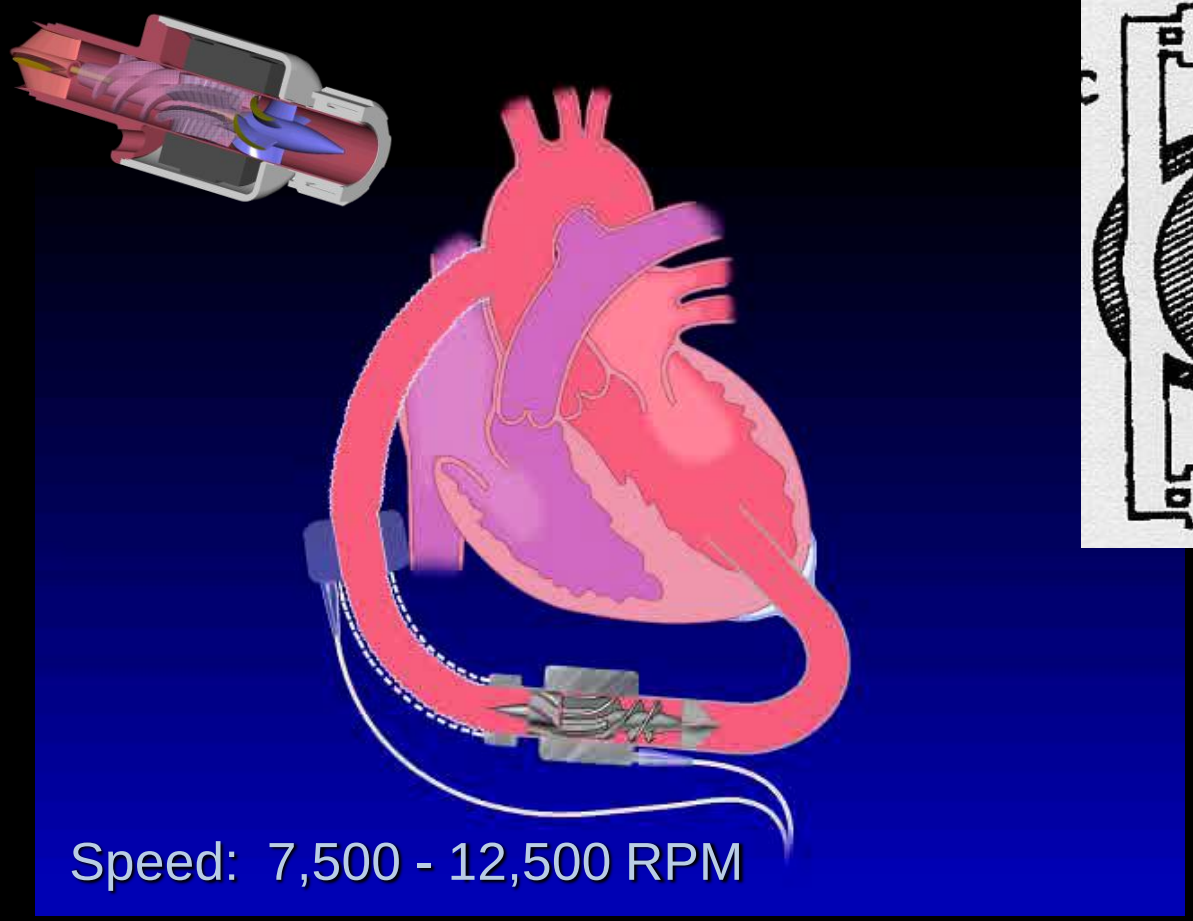
4) Pulmonary blood flow: sepsis, anemia, …

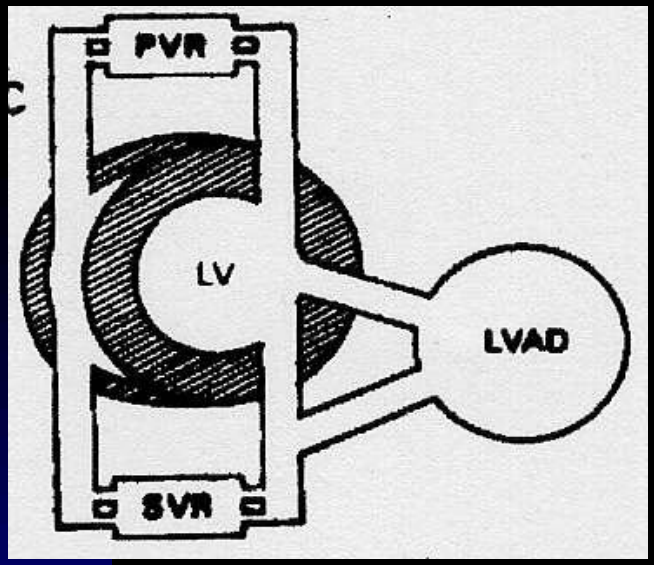
"vasoreactive"
or
"fixed"

Dana McGlothlin et al. Prog Cardiovasc Dis 2012;55:199-217

Left ventricular assist device (LVAD)

the axial-flow pump

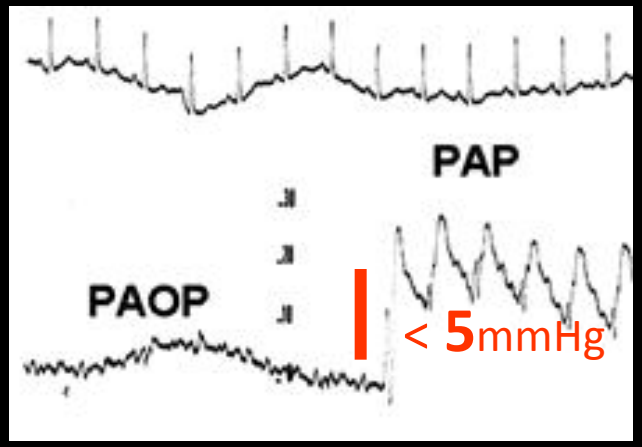




Adequate LVAD function has to be warranted by adequate transpulmonary blood flow and RV function.

Catena E. et al. Best Practice & Research Clinical Anaesthesiology 2012; 26

Factors predictive of no severe RV failure after LVAD activation

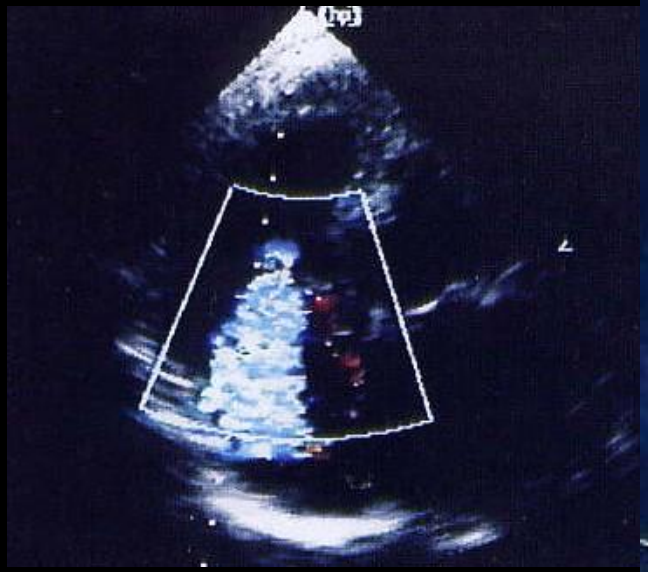


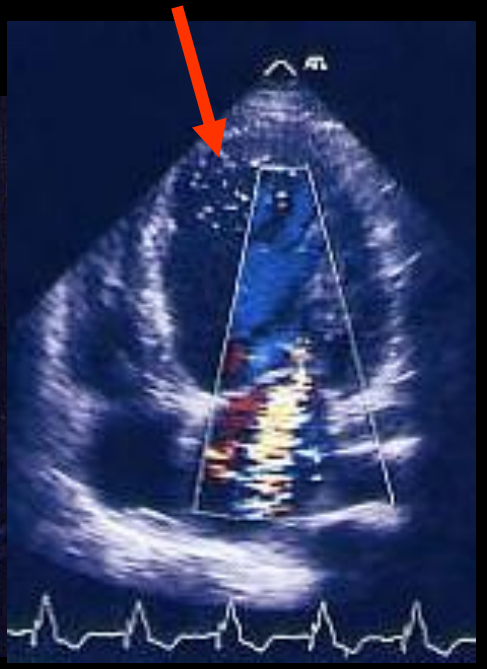
Transpulmonary pressure gradient:

PAP m – wedge < 5 mmHg

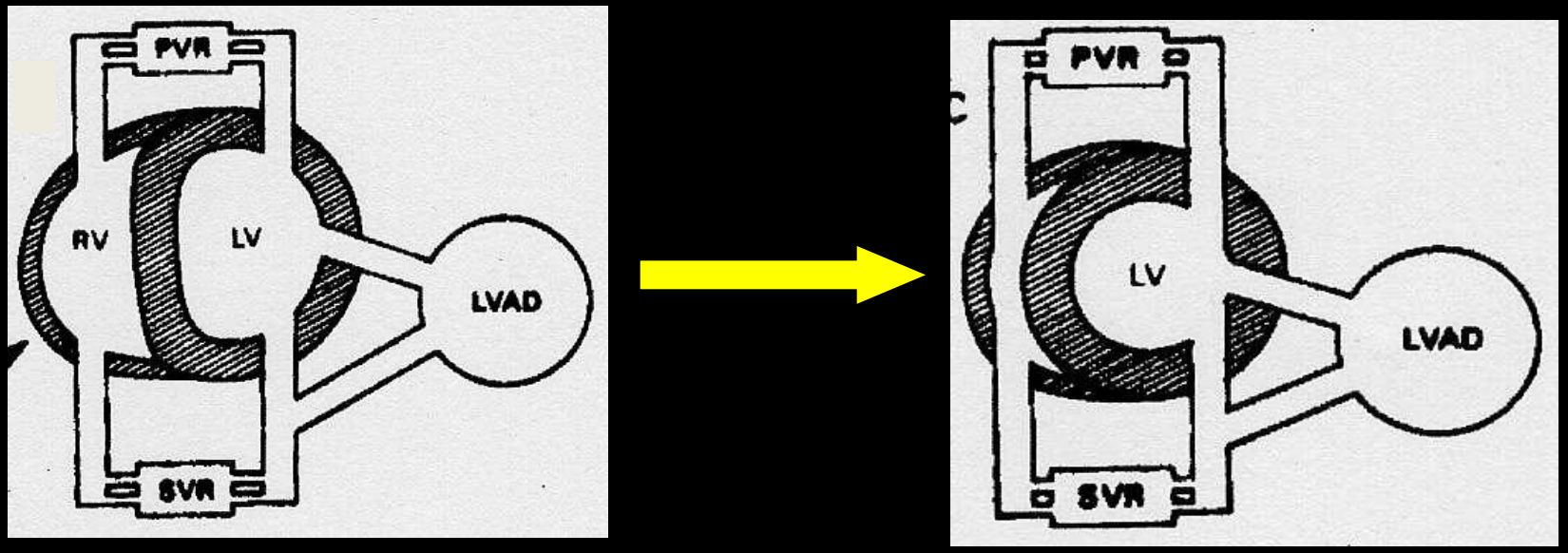
suggests that tricuspid regurgitation is secondary to increased RV afterload,due to LV failure.

- ITr ++ o +++/IV
  PAPs increased
- RVFAC (30-50%)
- TAPSE (14-18 mm)
- Sm (9-13 cm/s)
- RVOT fs% (20-40%)
- T acc 60-80 msec





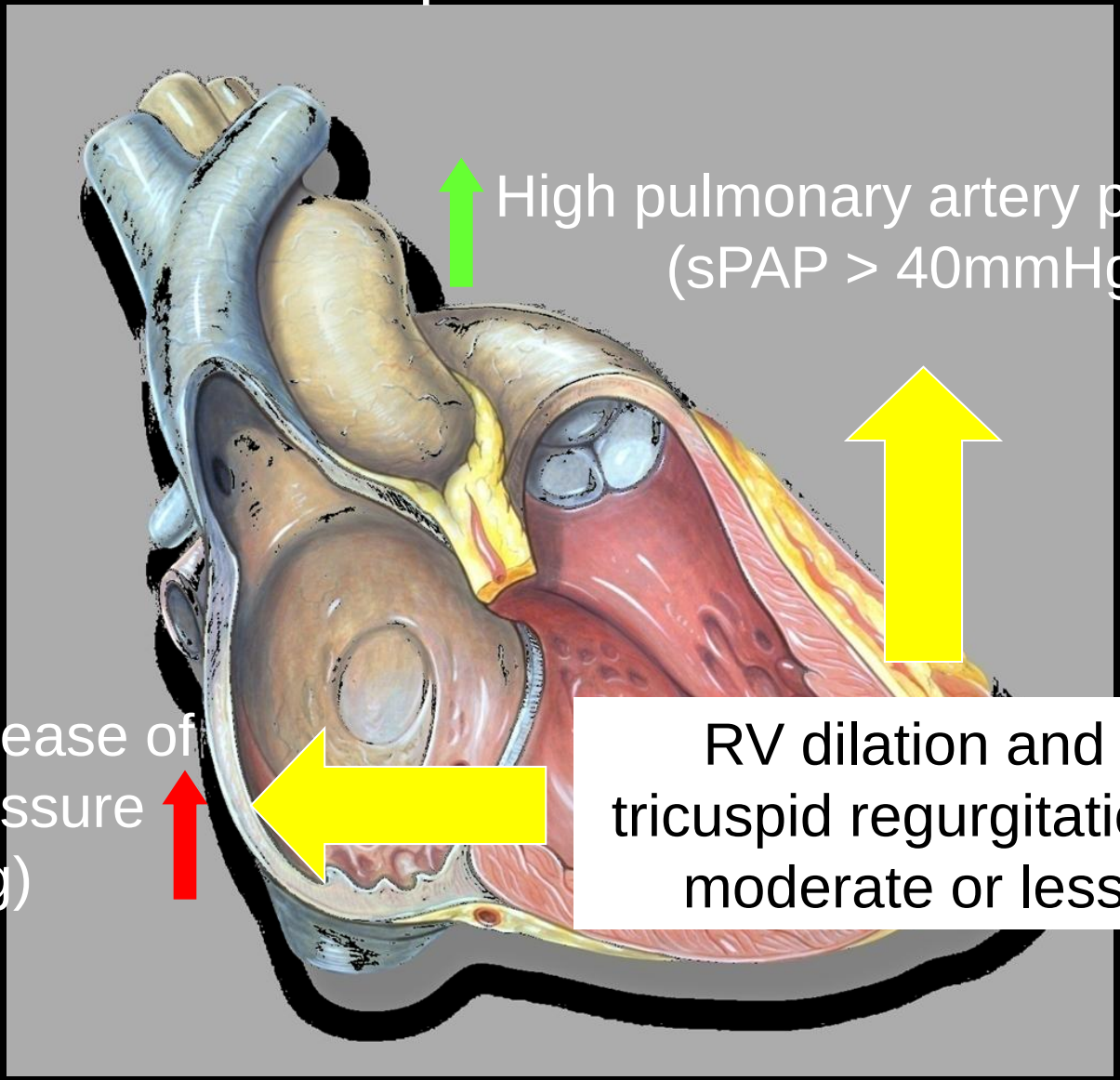
Factors predictive of no severe RV failure after LVAD activation



In patients with normal transpulmonary pressure gradient and normal pulmonary vascular resistances, LVAD support alone is able to improve right ventricular afterload by reducing left atrial pressure.

Patients with high PAP and high wedge rarely are a problem!

LVAD activation: preserved RV function



Factors predictive of severe RV failure after LVAD activation

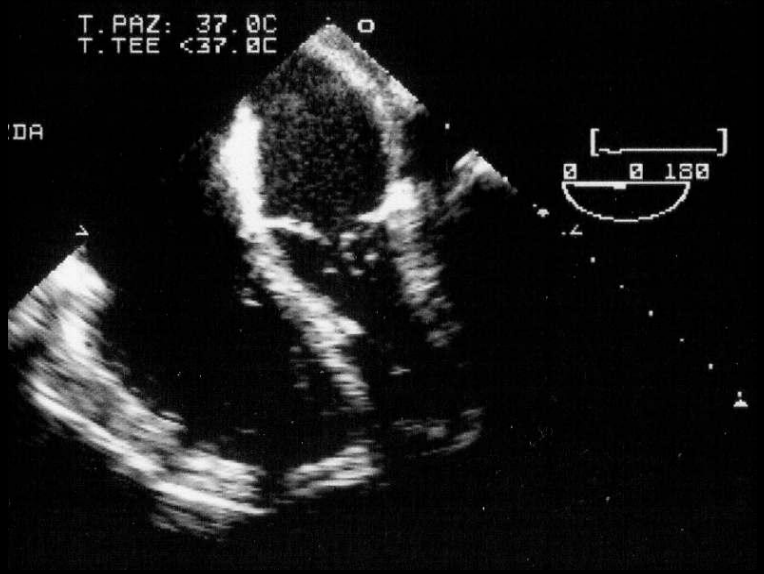
- ↑ RAP ↓ PAP

(mPAP-mRAP <4)

El-Banayosy et al. Ann Thorac Surg 2001;71:S98 –102

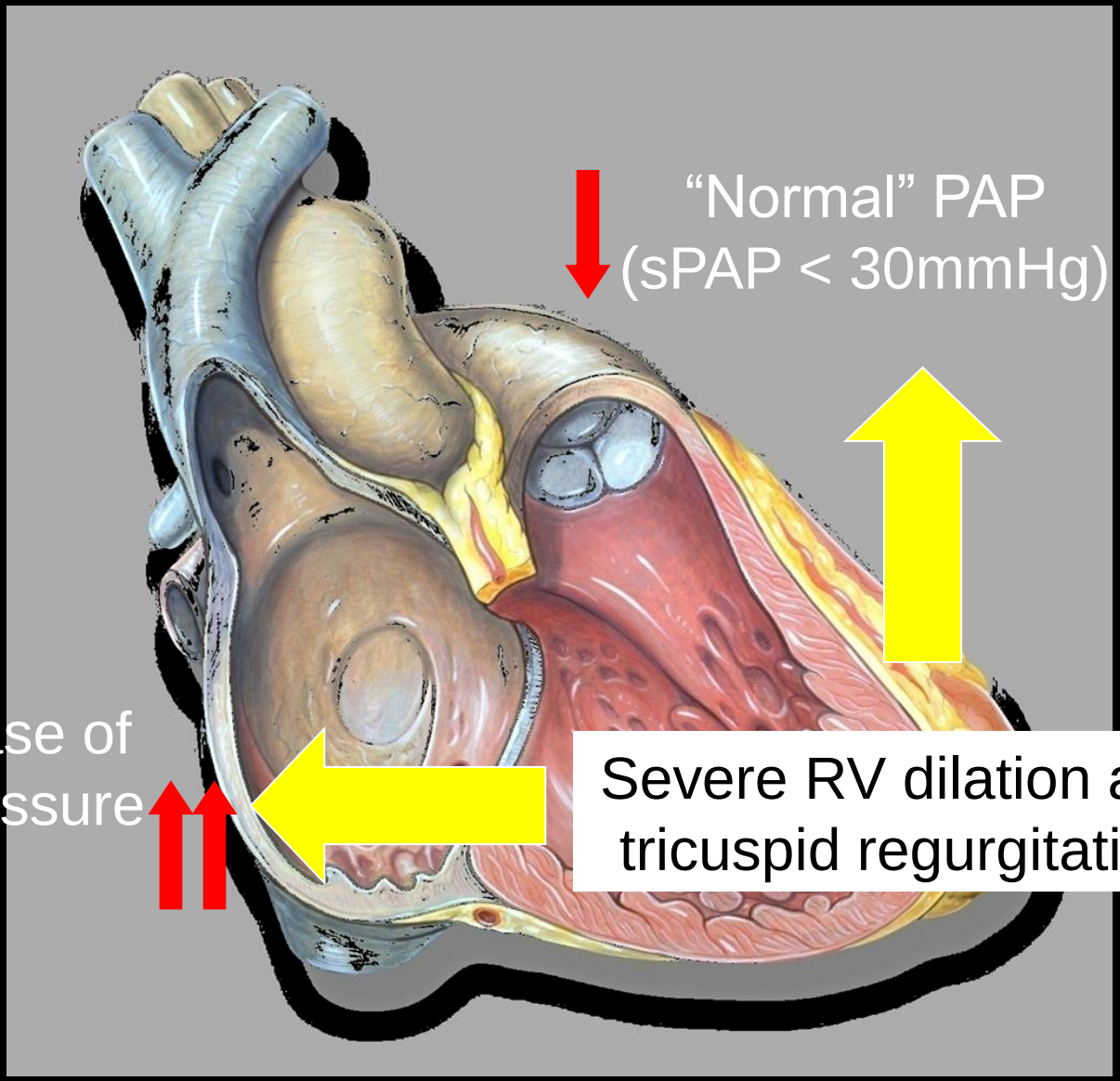
- ↓ RVSWI: mPAP-mRAP. $\frac{CI}{HR}$

K Fukamachi, et al. Ann Thorac Surg 1999;68:2181-4



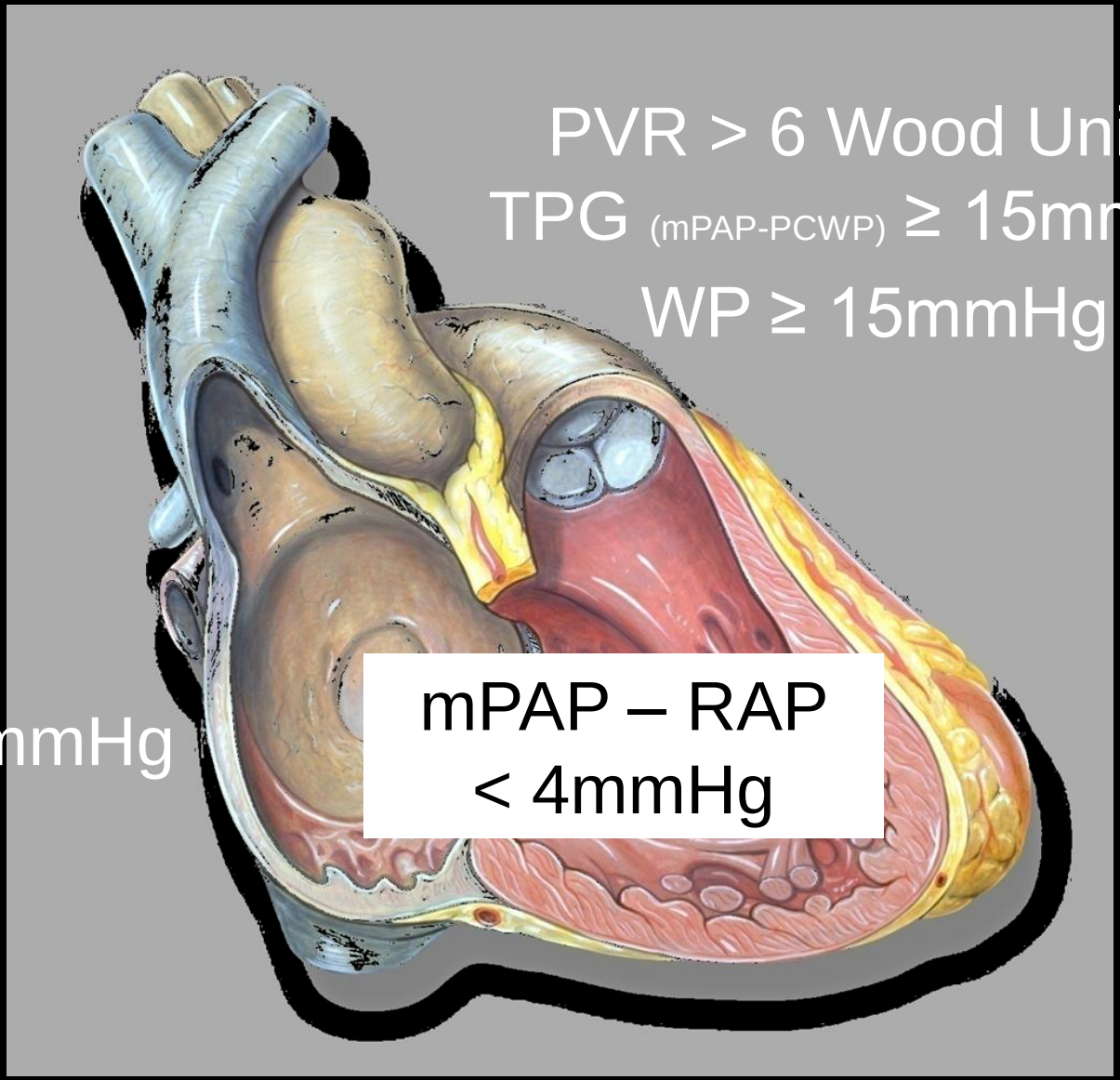
- **Enlarged RV chamber:**
  RV volume >> LV volume
- **RVFAC < 20%**
- **TAPSE < 10 mm**
- **Sm < 5-8 cm/s**
- **RVOT fs% 10-15 mm**
- **Light or absent tricuspid regurgitation**

LVAD activation: RV failure



Severe increase of
right atrial pressure
(> 20 mmHg)

Risk factors for RV failure on LVAD



PVR > 6 Wood Units

TPG (mPAP-PCWP) ≥ 15mmHg

WP ≥ 15mmHg

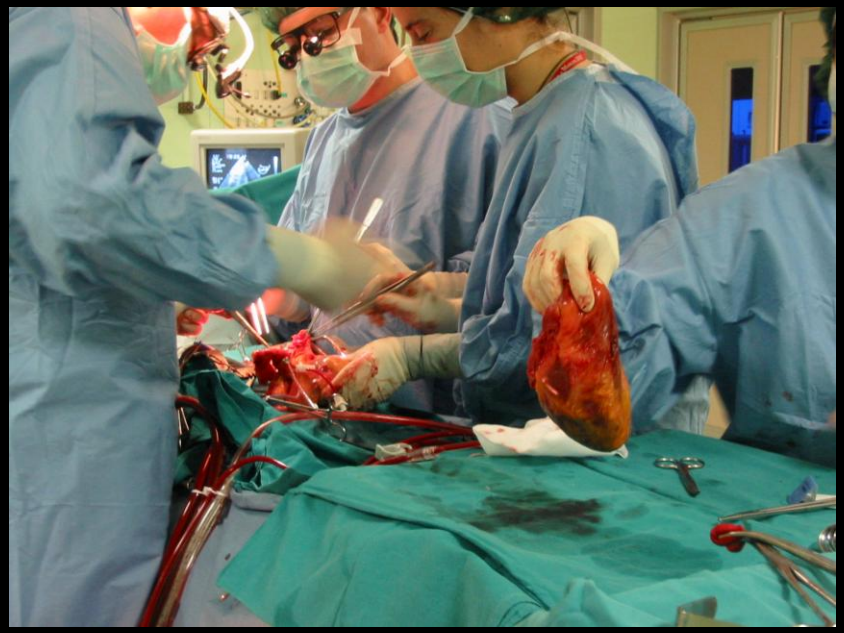
RAP > 20mmHg

mPAP – RAP
< 4mmHg

What about pulmonary hypertension?

Patients showing pulmonary hypertension with increased and fixed pulmonary vascular resistances (>2.5 UW), with high transpulmonary pressure gradient (PAPm – WP > 12-15 mmHg), unresponsive to vasodilators, are at high risk of RV failure after heart transplantation.

Reversibility is required for heart transplantation (RVP <2.5, TPG <12)



Has “unresponsive” pulmonary hypertension to be considered a contraindication for LVAD support?

It is a risk factor for RV failure,
it is an indication for LVAD support!

…LVAD support and continuous nonpulsatile mechanical unloading of the left ventricle can reverse medically unresponsive pulmonary hypertension and render patients eligible for orthotopic heart transplantation.

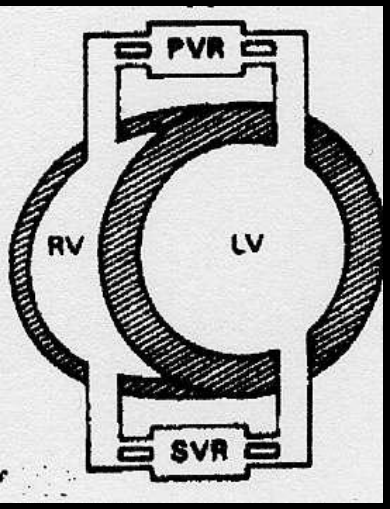
Etz CD et al. Ann Thorac Surg 2007;83:1697

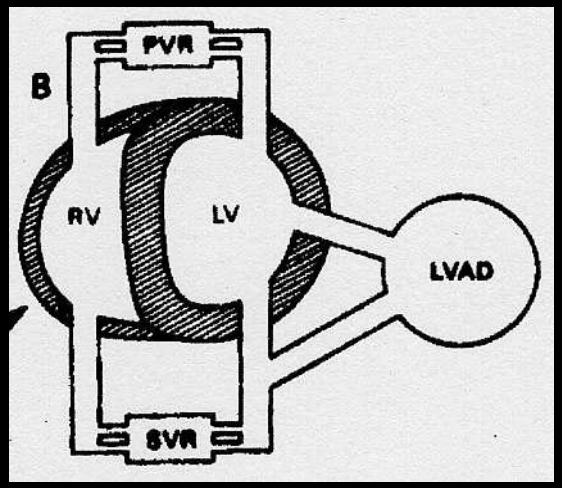
…left ventricular assist devices should be considered in all cardiac transplant candidates with fixed pulmonary hypertension.

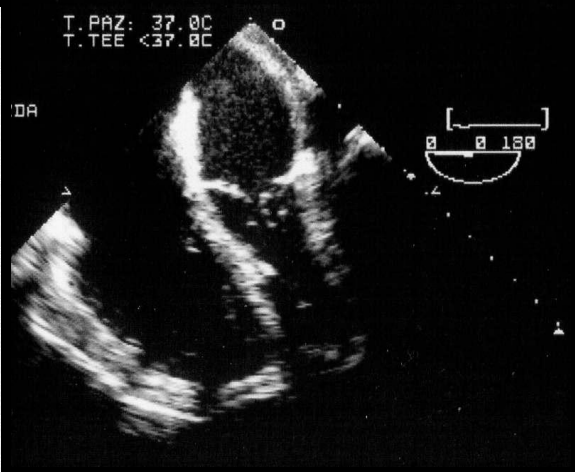
Zimpfer D, et al. J Thorac Cardiovasc Surg. 2007 Mar;133(3):689-95.

RIGHT VENTRICULAR FUNCTION

"the output of one ventricle is the input of the other"



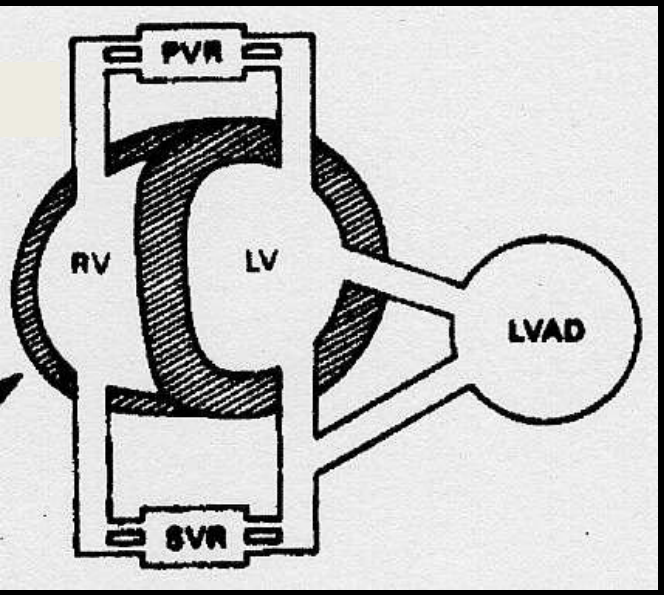




Adequate LVAD function has to be warranted by adequate transpulmonary blood flow and RV function.

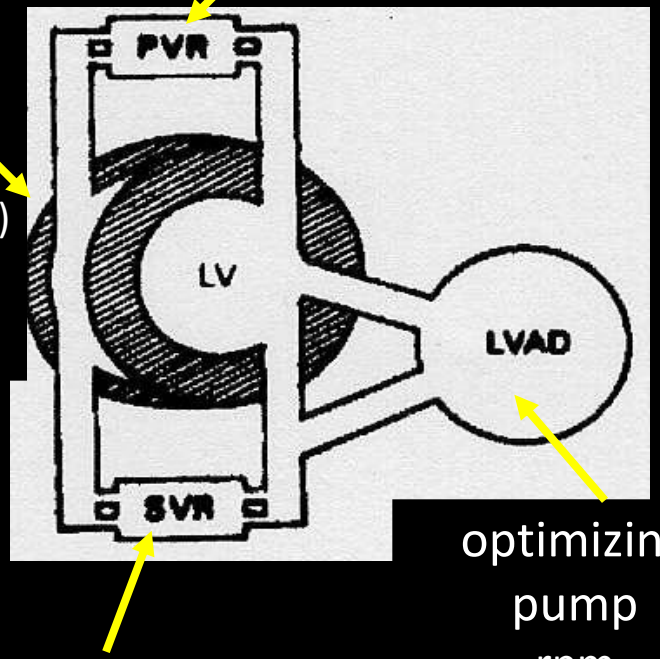
How can you improve right ventricular function?

SELECTIVE PULMONARY VASODILATOR: NITRIC OXIDE



INOTROPIC DRUGS:

EPINEPHRINE
(0.06-0.12 mcg/Kg/min)
MILRINONE
(0.3-0.8 mcg/Kg/min)



optimizing pump rpm

optimizing vasodilators dosage:
NITROPRUSSIDE

Any increase in flow to the systemic circulation from the LVAD will result in an increase in venous return to the right ventricle. The right ventricle has to have a “reserve” to be able of increasing its cardiac output to at least the amount being pumped by the LVAD

Inotropes

EPINEPHRINE: a potent α and ß receptor agonist that causes vasoconstriction, increased inotropy, increased CO, without altering the PVR/SVR ratio.

MILRINONE: a selective phosphodiesterase-3 inhibitor has inotropic and direct acting pulmonary vasodilatory properties.

DOPAMINE: in doses ranging from 2 – 10 mcg/Kg/min increases CO and heart rate but not PVR.

It fails to improve RV ejection fraction

DOBUTAMINE: has inotropic effects through ß-1 receptor stimulation and vasodilatory effects through ß-2 receptor agonism

Effects of selective pulmonary vasodilator:

Nitric oxide

↓

Increased RV output

↓

Increased LV preload

→

LV failure

↓

Increased pulmonary capillary pressure

↓

pulmonary edema

Bocchi EA et al. Am J Cardiol 1994; 74: 70-72

Effects of systemically administered pulmonary vasodilator:

systemically administered pulmonary vasodilators when given to hypoxic patients with parenchymal lung disease can increase perfusion to poorly ventilated lung regions, resulting in worsened hypoxemia via V/Q mismatching and hypotension.

Phosphodiesterase type 5 inhibitors are showing some promise in this group

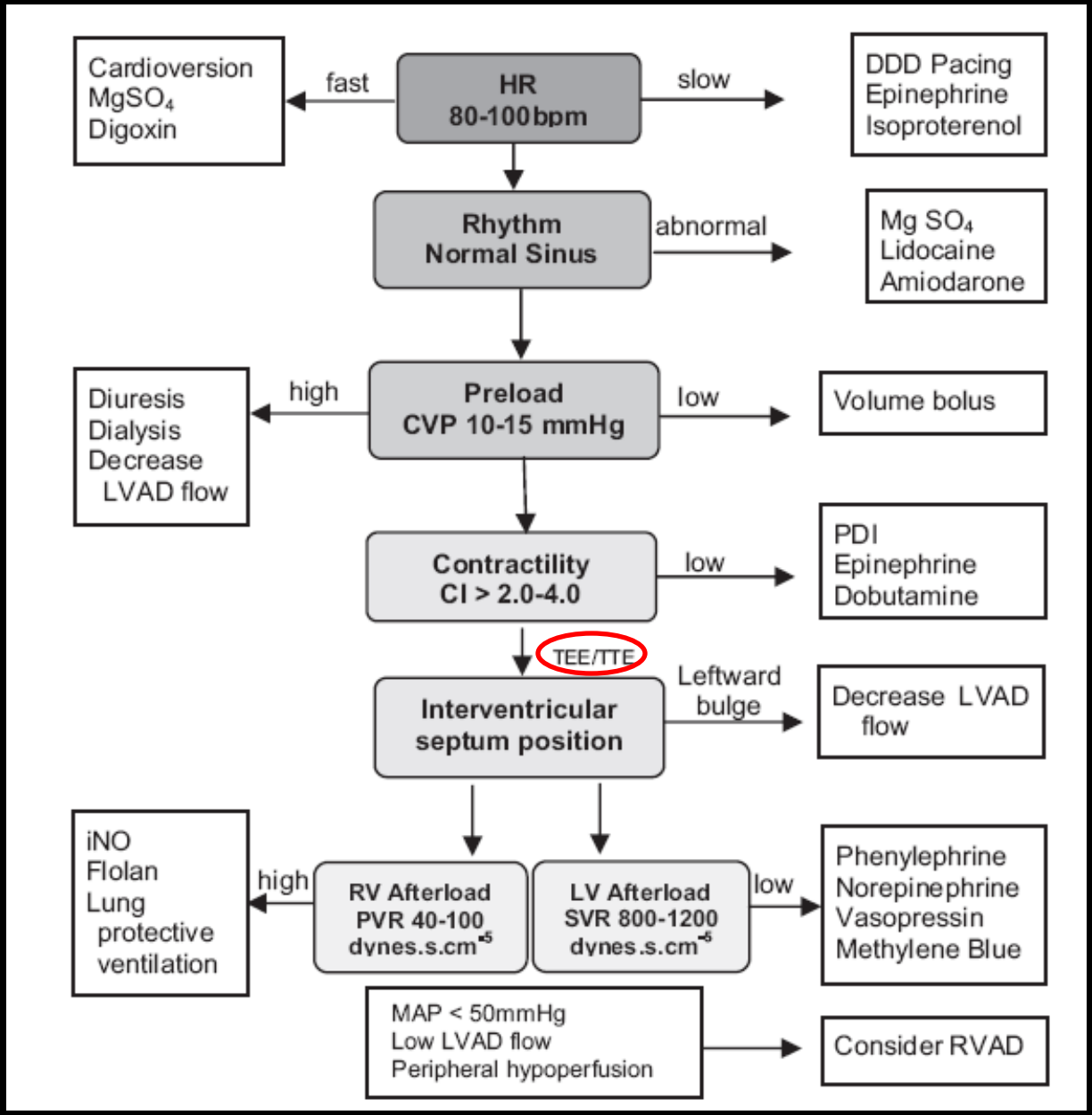
Ventilatory conditions to avoid and promote

AVOID PULMONARY VASOCONSTRICTORS:

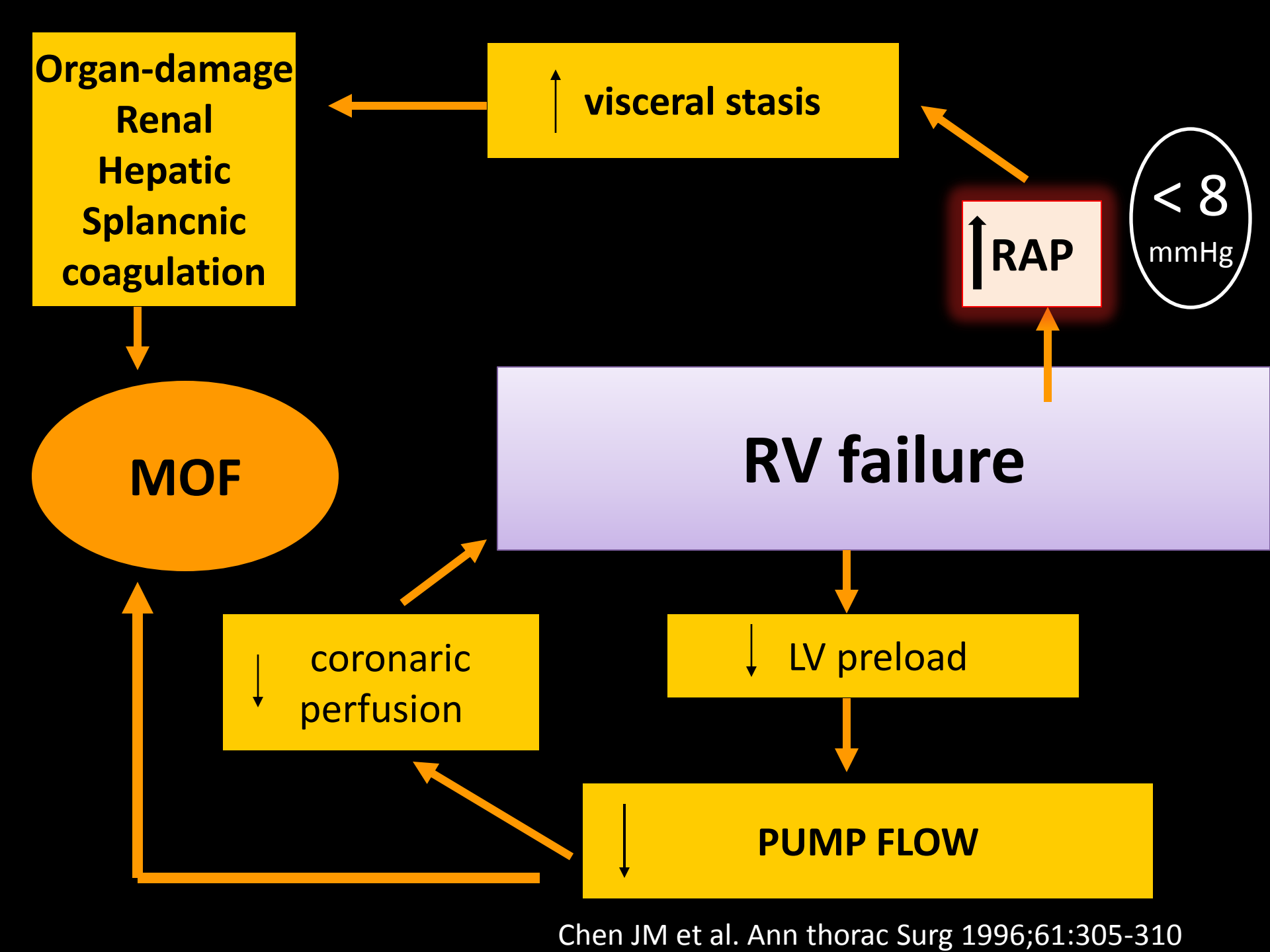
- Hypoxemia
- Inspiratory pressure > 30 mmHg
- High PEEP (> 15 mmHg)
- Hypercapnia and acidosis

PROMOTE PULMONARY VASODILATION

- Improve oxygenation (FiO2 1)
- Permissive hypocapnia (pCO2 < 30-35 mmHg)
- Optimal ventilatory volume



Maineri M et al. Best Practice & Research Clinical Anaesthesiology 26 (2012) 217–229



Chen JM et al. Ann thorac Surg 1996;61:305-310

Thank you for your attention