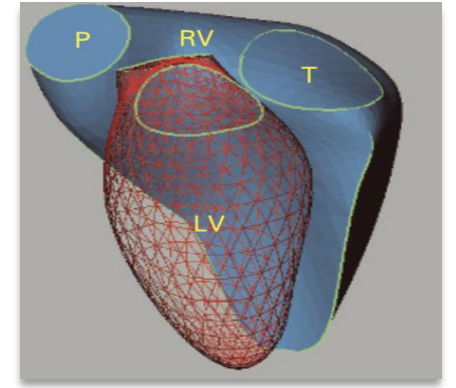
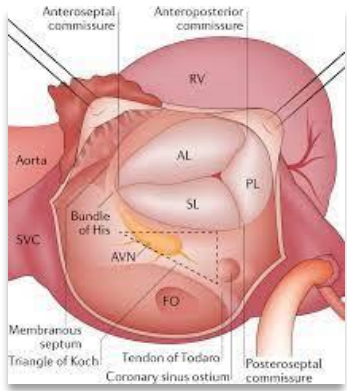




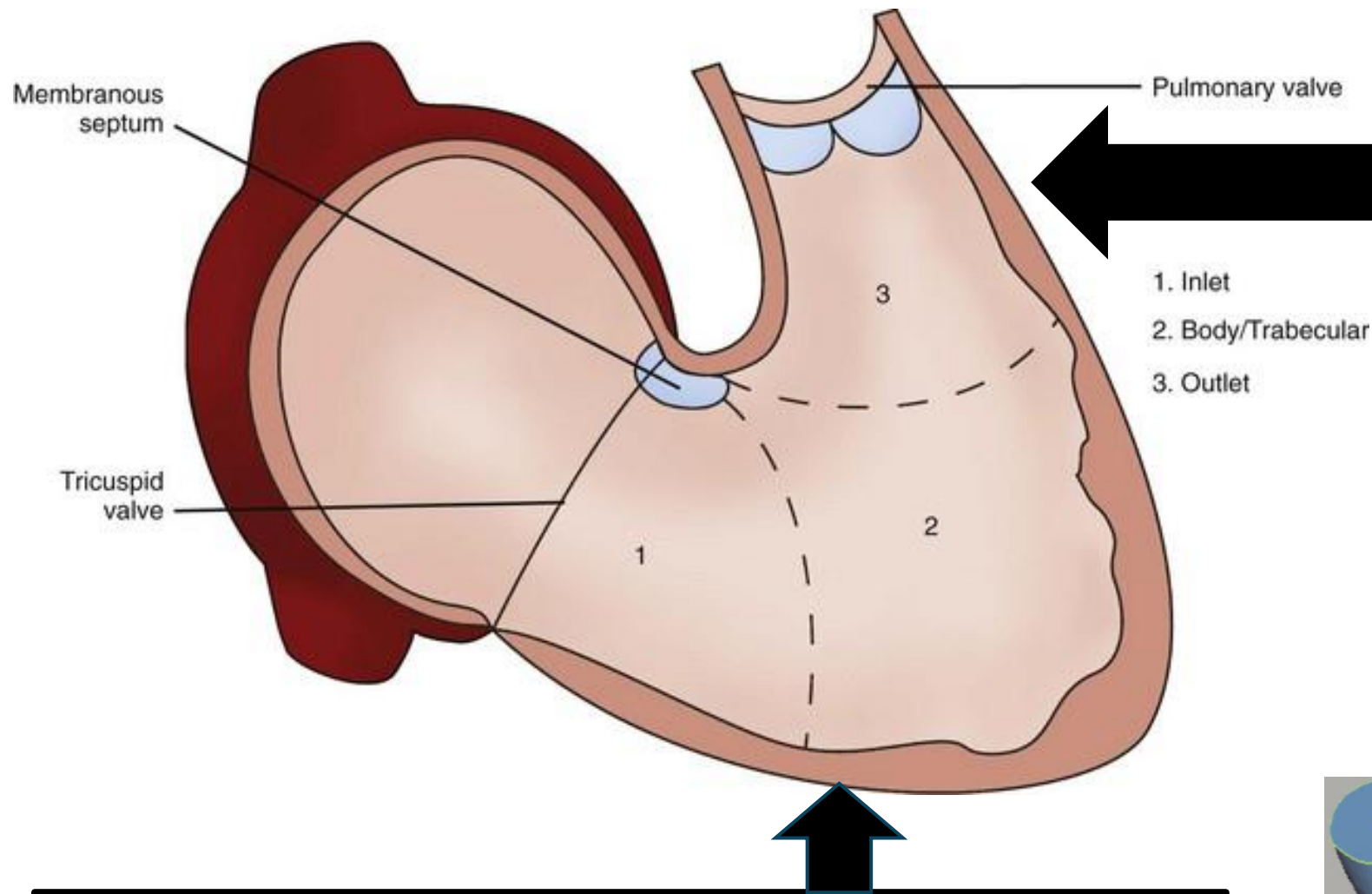
Is it time to update the echo report and put some more focus on the right ventricle?

M. Vaturi

Rabin Medical Center, Beilinson Hospital



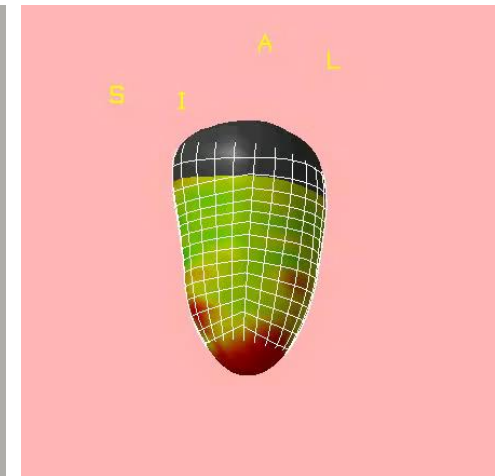
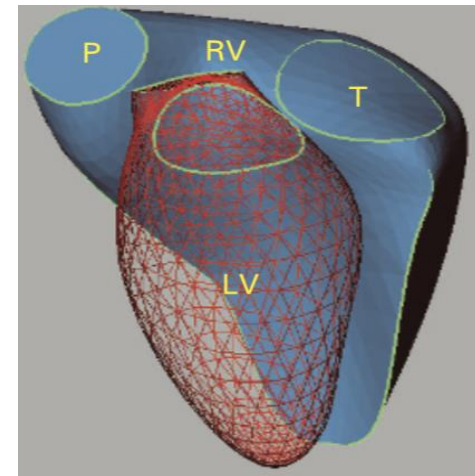
The Foundations of RV function



The RVOT is anatomically distinct from the body of the RV.

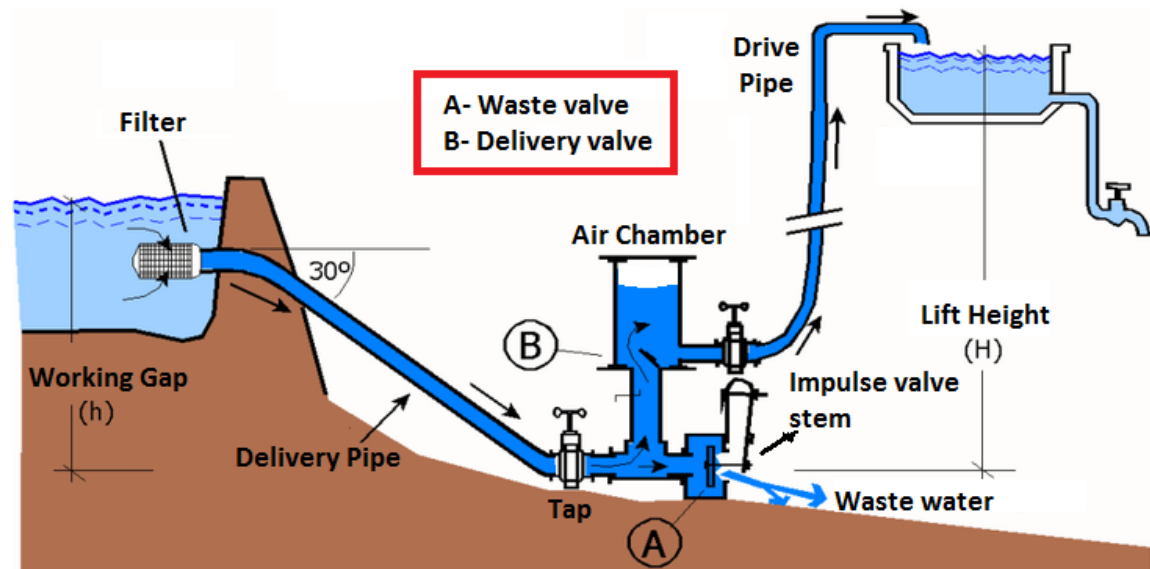
The muscle fibers in the conus run in a longitudinal alignment **from the epicardium to the endocardium**, contributing actively to RV forward flow.

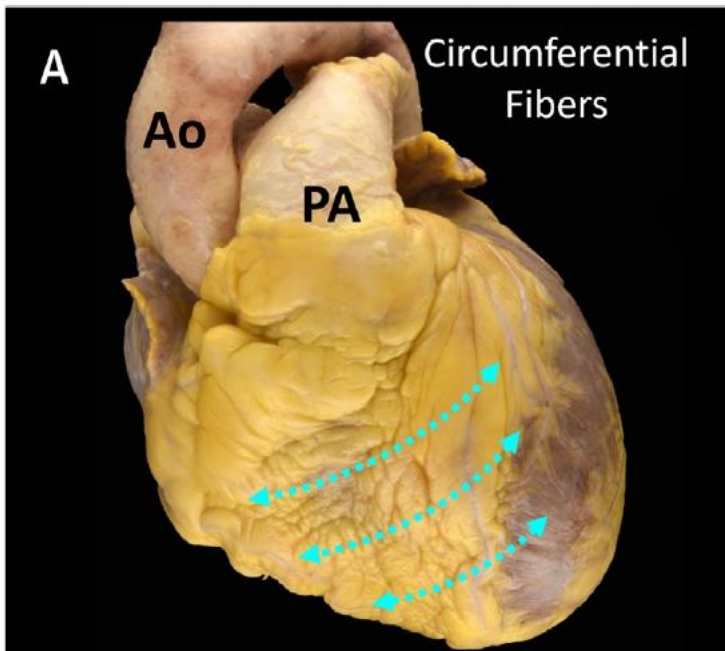
Flow studies show less vortex formation in the RV compared with the LV but rather more of a helical flow toward the outflow facilitated by the trabeculated apex.



RV functions as a hydraulic ram

- Fluid enters the ram at a lower pressure (*IVC,SVC*) and height and exits at substantially higher pressure (*PA*) and height through redirection of the fluid flow.
- Transition from continuous flow to a pulsatile flow





The RV has 2 dominant layers:
A. a subepicardial circumferential layer, continuous with the LV, encircling both ventricles (red dotted lines)

From histology to the echo machine

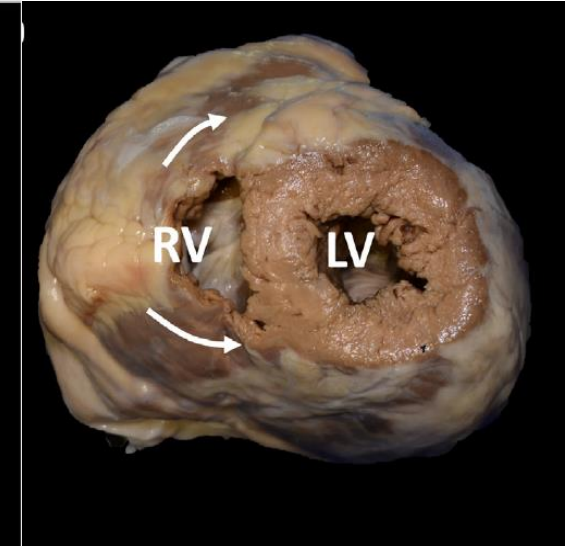
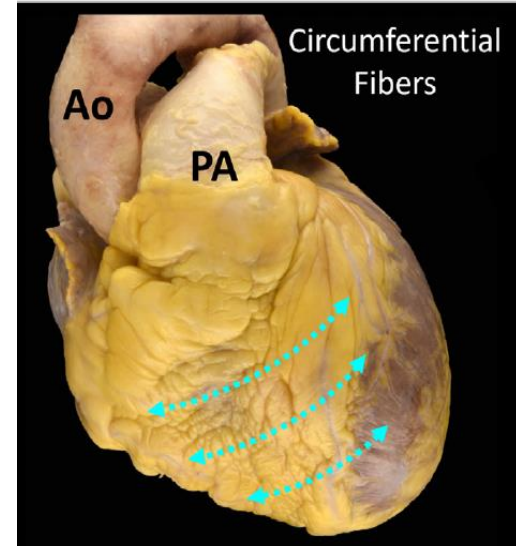
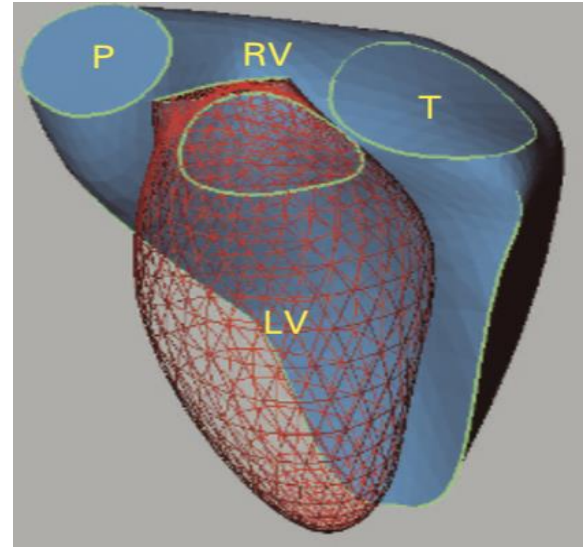
- TAPSE, RV S', RV strain (longitudinal fibers)
- RV FAC (circumferential fibers)

The Right Ventricular Unique Capacity for Conformational Adaptation

- The normal RV fills **at or below** its unstressed volume; hence, the RVEDV changes occur **without any change** in the RV diastolic wall stretch.
- The conformational changes in RV shape rather than stretch allow these volume changes to occur **without measurable changes in transmural RA pressure*** (RV distending pressure).

From histology to the echo machine

- Monitor the IVC during fluid resuscitation.
- Is dilated RV with “preserved” contraction still a normal ventricle?



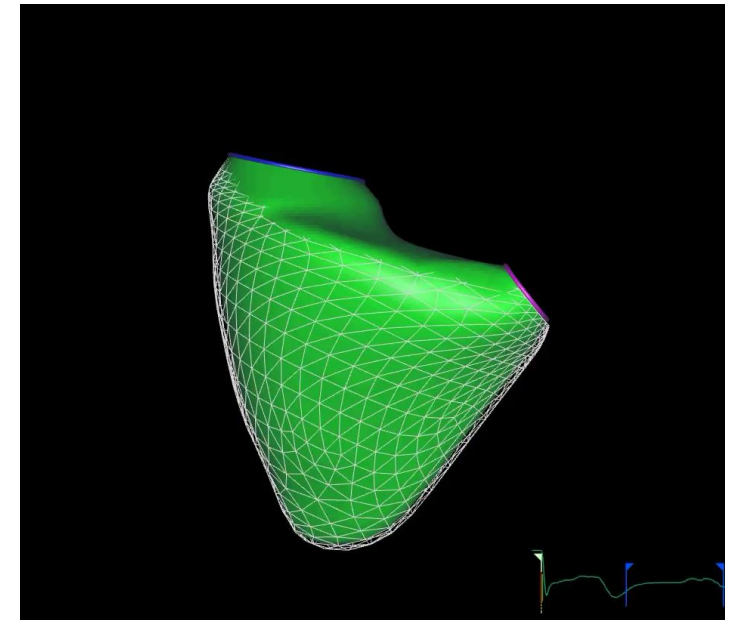
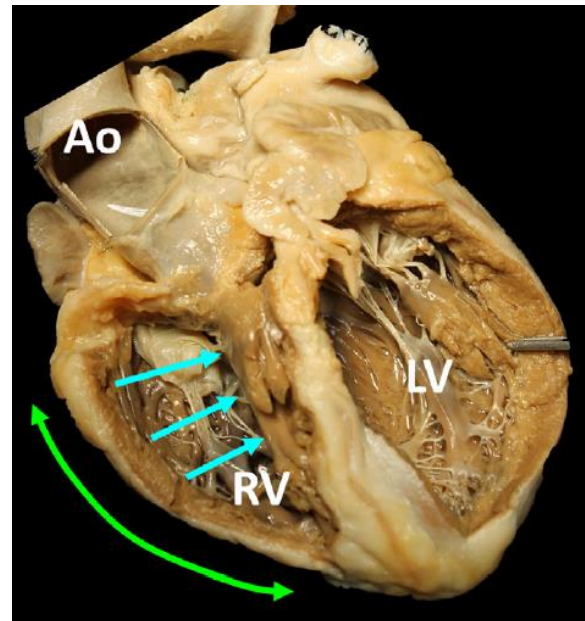
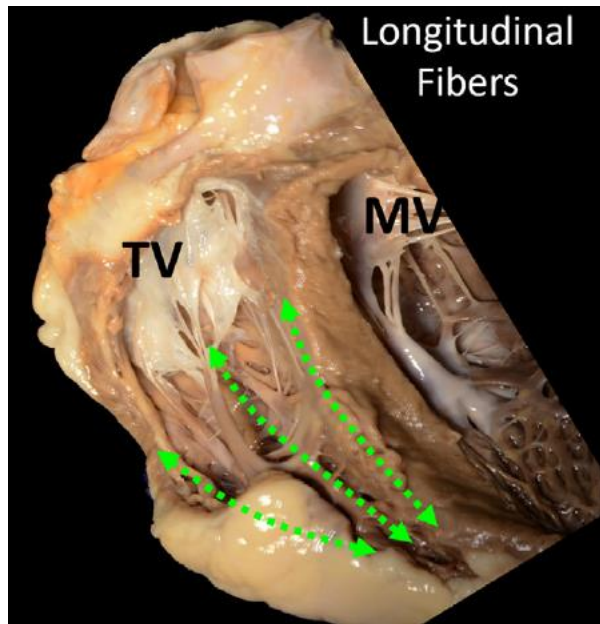
More than **half** of RV systolic pressure originates from LV contraction.

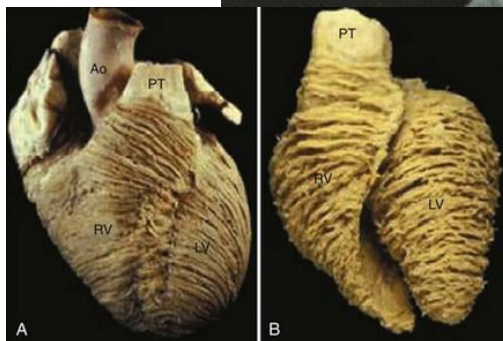
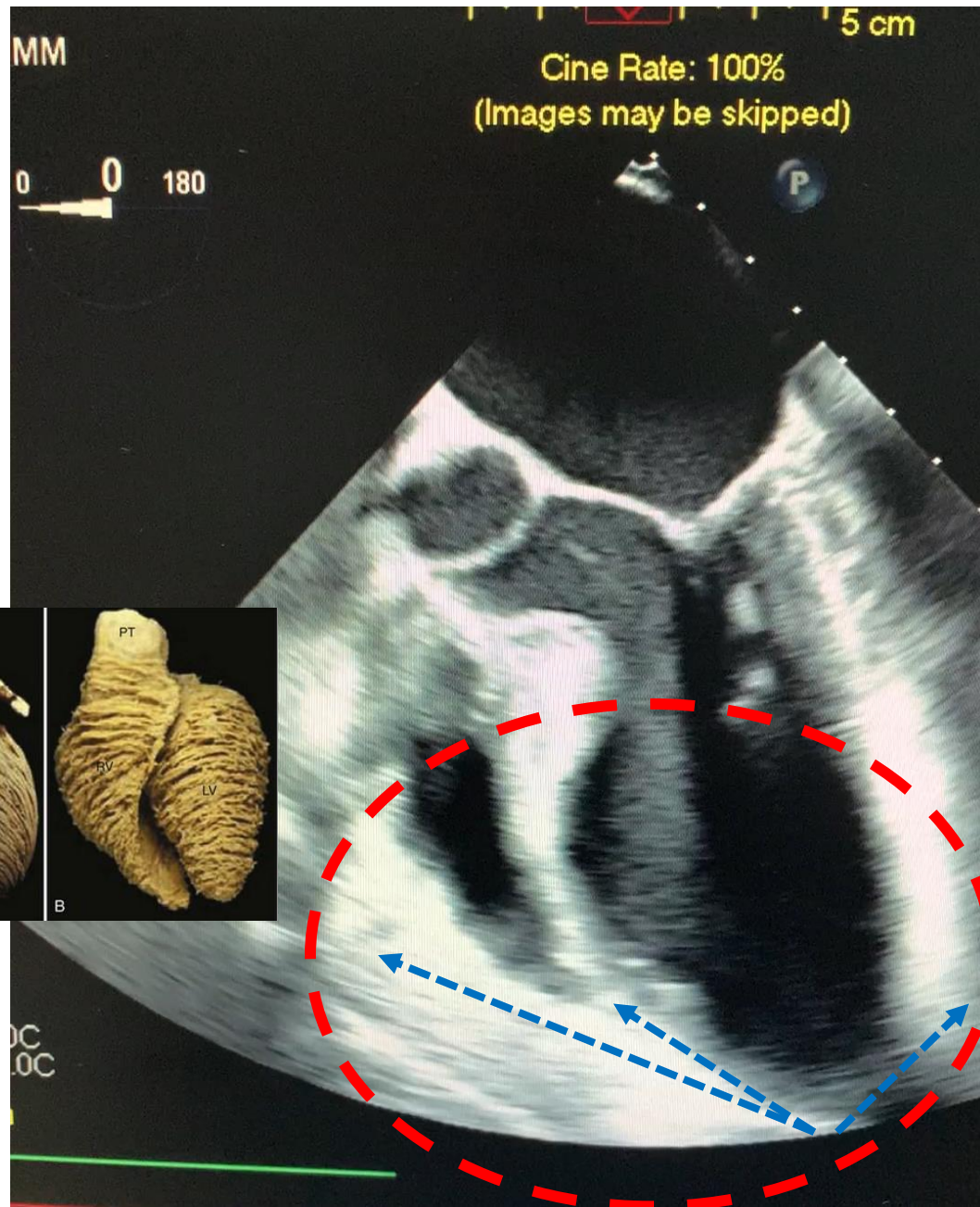
LV dysfunction impacts on the adjacent chamber (both directly and indirectly via increased pulmonary afterload). Hence, when the LV function is compromised, the RV's contraction cannot remain unaffected.

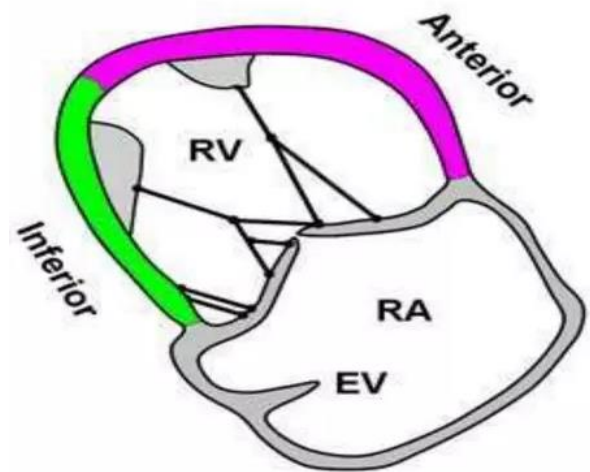
If the LV is dysfunctional, carefully examine
the RV for contraction flaws

RV's contraction is similar to a piston pump

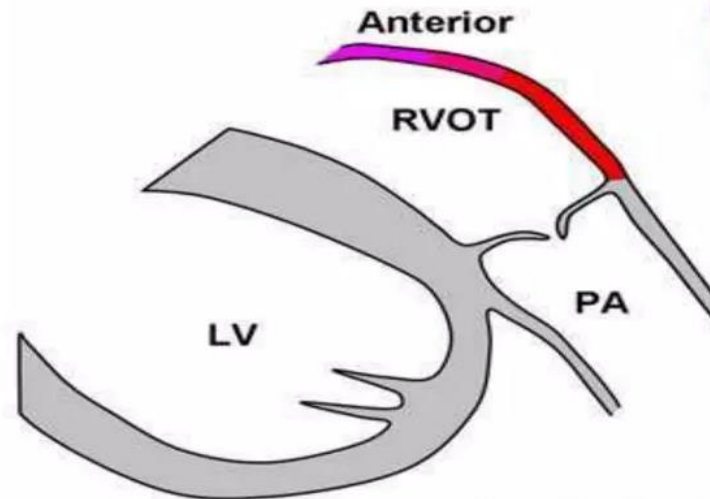
- Longitudinal shortening and base-to-apex piston-like motion of the atrioventricular plane contribute more to RV stroke volume than short-axis or circumferential shortening.
- The impact of hypo/akinetic interventricular septum on RV's function



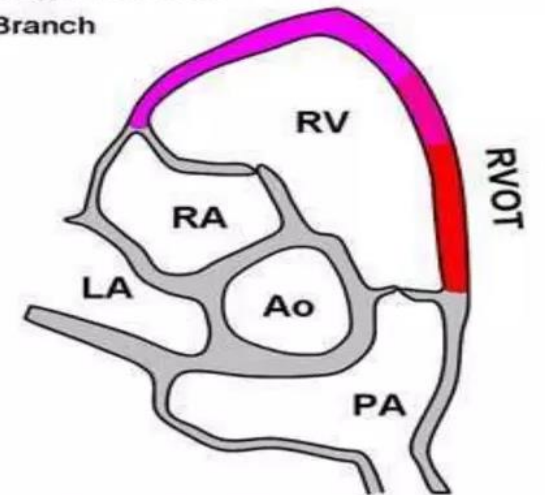




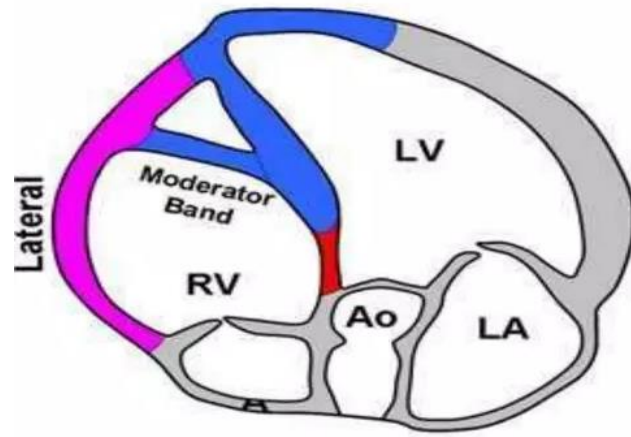
Parasternal view of RV inflow



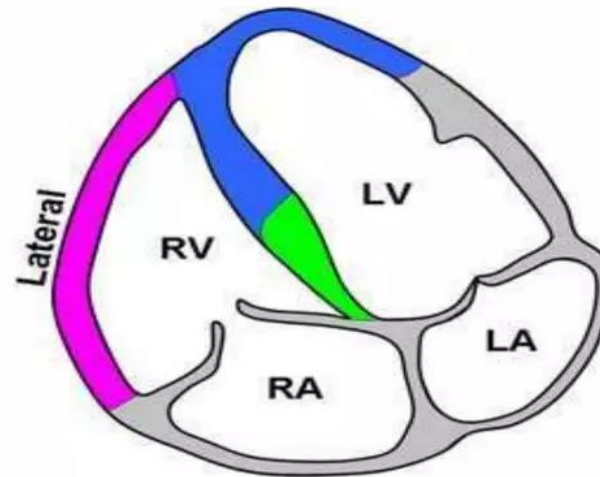
Parasternal long-axis of RVOT



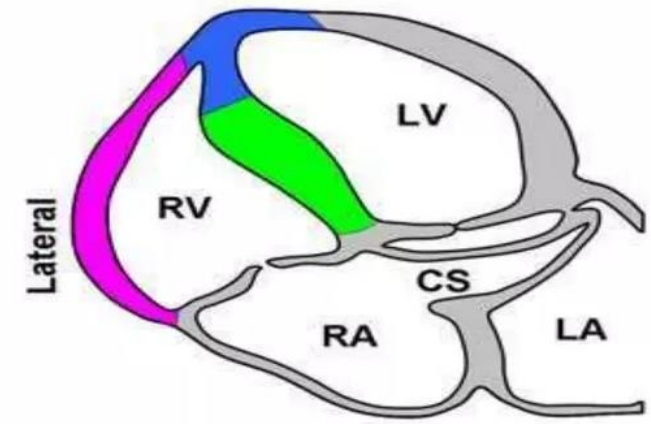
Subcostal short-axis of basal RV



RV apical 5-chamber view



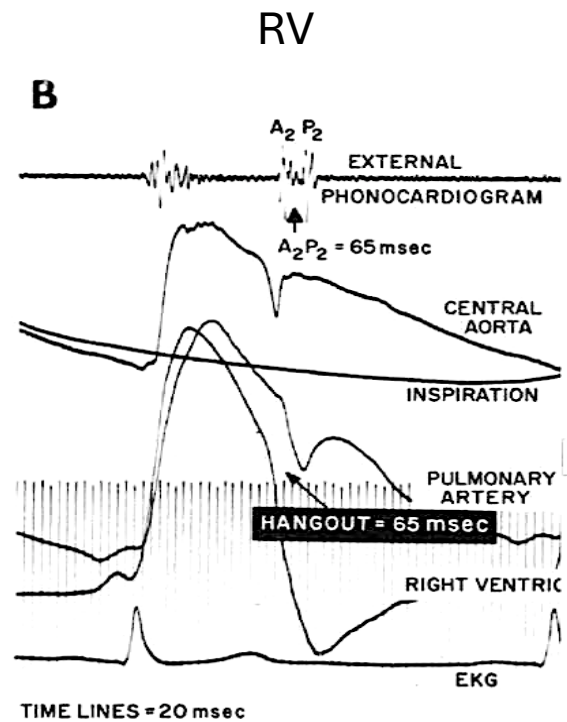
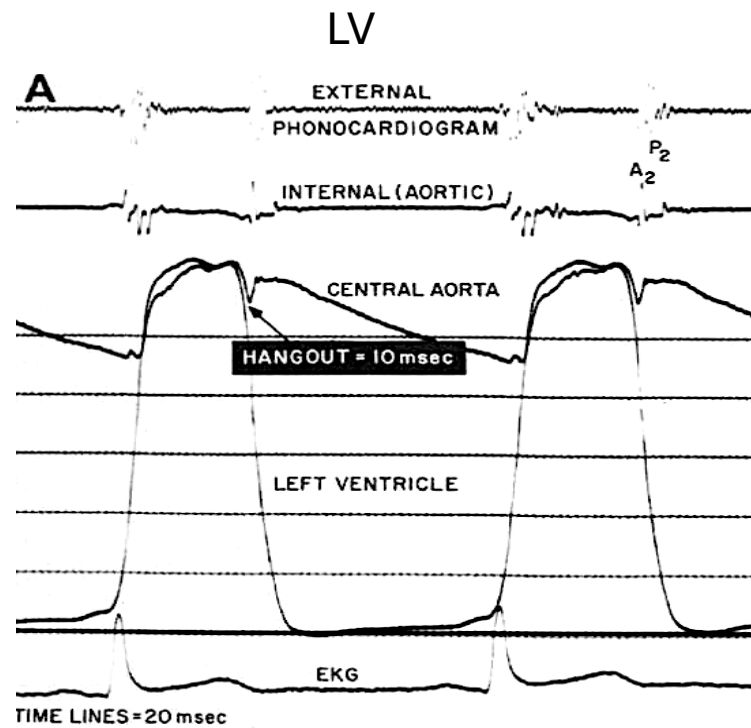
RV apical 4-chamber



RV apical coronary sinus view

- RCA: Posterior Descending Artery
- RCA: Acute Marginal Branch
- RCA: Conus Branch
- LAD





The LV ejection ends within 10 ms (practically immediately) after the LV reaches its peak pressure.

On the other hand, RV ejection continues for 65 ms after peak systolic pressure (on account of the isovolumic relaxation time).

The disappearance of the 'hangout' period due to elevated PA pressure marks the loss of a fundamental aspect of RV well-being.

Estimation of the PVR- not only at the cath. lab.

Abbas formula ($PVR \leq 6$ WU)

$$TRV/TVI_{RVOT} \times 10$$

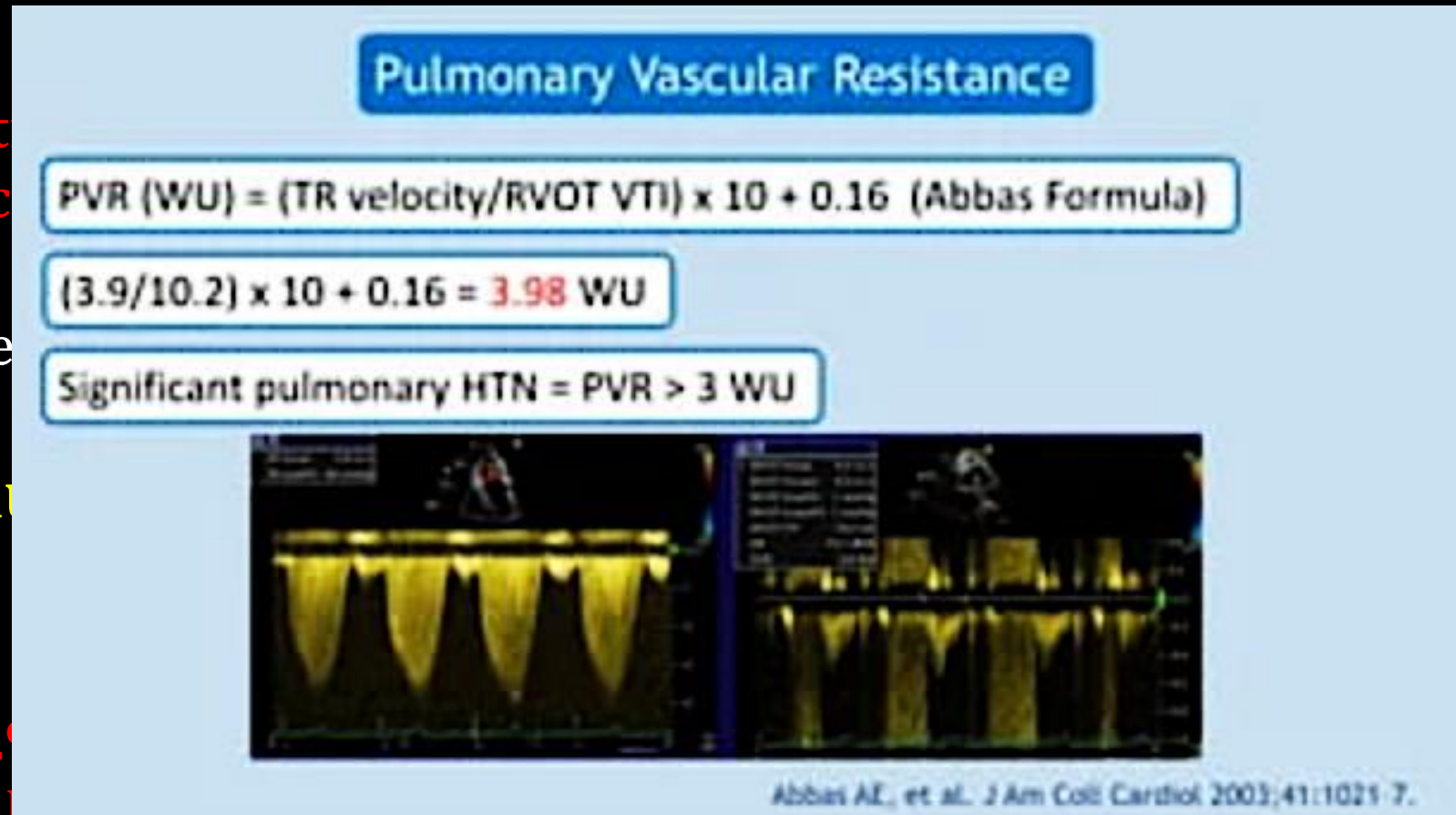
- $TRV/TVI_{RVOT} > 0.175$ dist sensitivity and 81% spec

$TRV/TVI_{RVOT} > 0.275$ suggest

Modified Abbas formula

$$TRV^2/TVI_{RVOT} \times 5$$

- TRV^2/TVI_{RVOT} cutoff of 0.9 determine $PVR_{cath} > 6$ WU



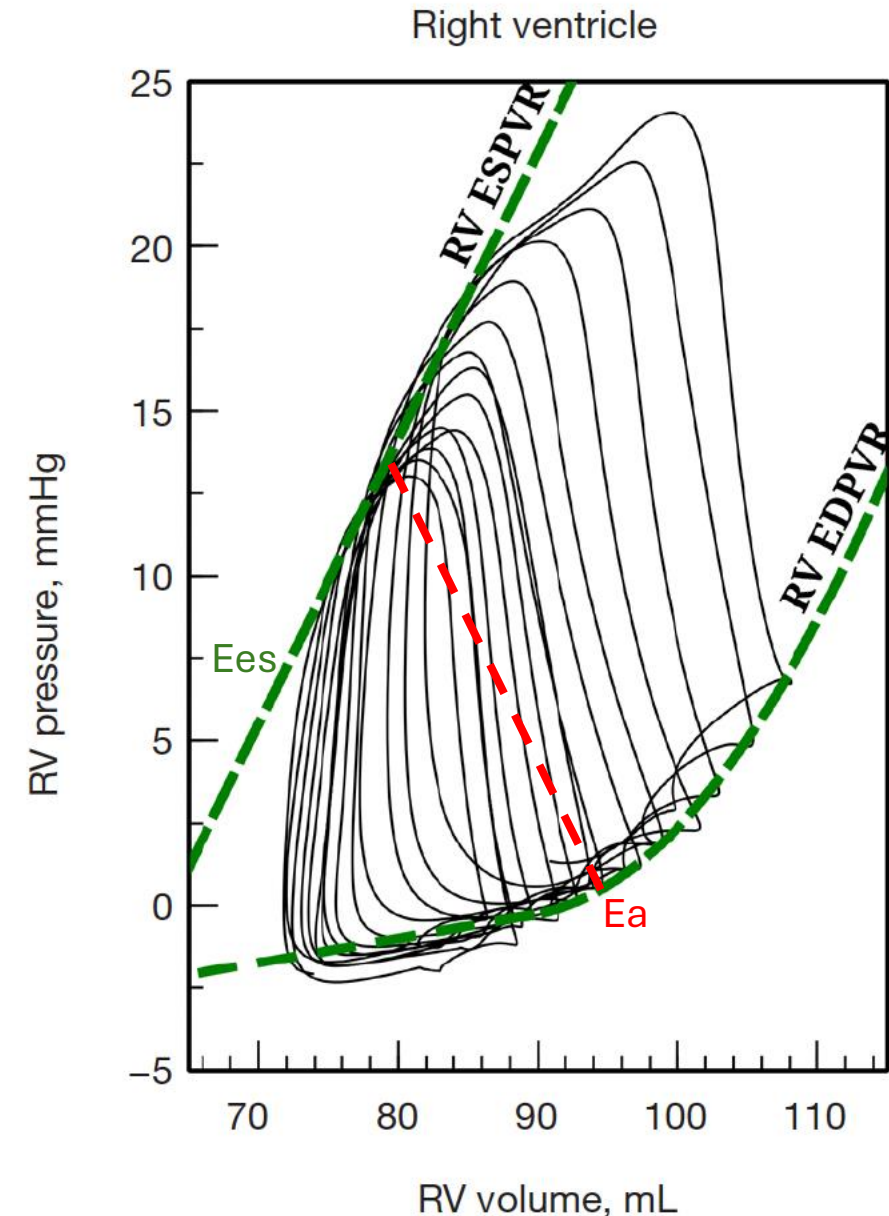
Invasive assessment is preferable
(Abbas formulas are not reliable)

- Extremely high PVR (>12 WU)
- Marked variation in HR

RV-PA coupling

The ratio between contractility and afterload

- The **area within the loop** defines the **RV Stroke Work**
- The **width of the loop** represents the **stroke volume**
- **End-systolic elastance (Ees)** is a load-independent measure of **contractility**
- RV afterload is determined by dividing the RVESP by the stroke volume providing the **effective arterial elastance (Ea)**
- **Ees/Ea ratio = ventriculo-arterial coupling** (contractility in the context of afterload); the optimal mechanical coupling is ratio 1.0, with uncoupling occurring with a ratio < 0.6
- **When PASP increases acutely**, RV SV decreases significantly, and Ea increases disproportionately compared to Ees. Consequently, the **RV function becomes inefficient**, requiring **more energy** to maintain adequate RV output.



The Assessment of RV Function by Echo-Doppler

Table 10 Normal values for parameters of RV function

Parameter	Mean $\pm$ SD	Abnormality threshold
TAPSE (mm)	24 $\pm$ 3.5	< 17
Pulsed Doppler S wave (cm/sec)	14.1 $\pm$ 2.3	< 9.5
Color Doppler S wave (cm/sec)	9.7 $\pm$ 1.85	< 6.0
RV fractional area change (%)	49 $\pm$ 7	< 35
RV free wall 2D strain* (%)	-29 $\pm$ 4.5	> -20 (<20 in magnitude with the negative sign)
RV 3D EF (%)	58 $\pm$ 6.5	< 45
Pulsed Doppler MPI	0.26 $\pm$ 0.085	> 0.43
Tissue Doppler MPI	0.38 $\pm$ 0.08	> 0.54
E wave deceleration time (msec)	180 $\pm$ 31	< 119 or > 242
E/A	1.4 $\pm$ 0.3	< 0.8 or > 2.0
e'/a'	1.18 $\pm$ 0.33	< 0.52
e'	14.0 $\pm$ 3.1	< 7.8
E/e'	4.0 $\pm$ 1.0	> 6.0

MPI, Myocardial performance index.

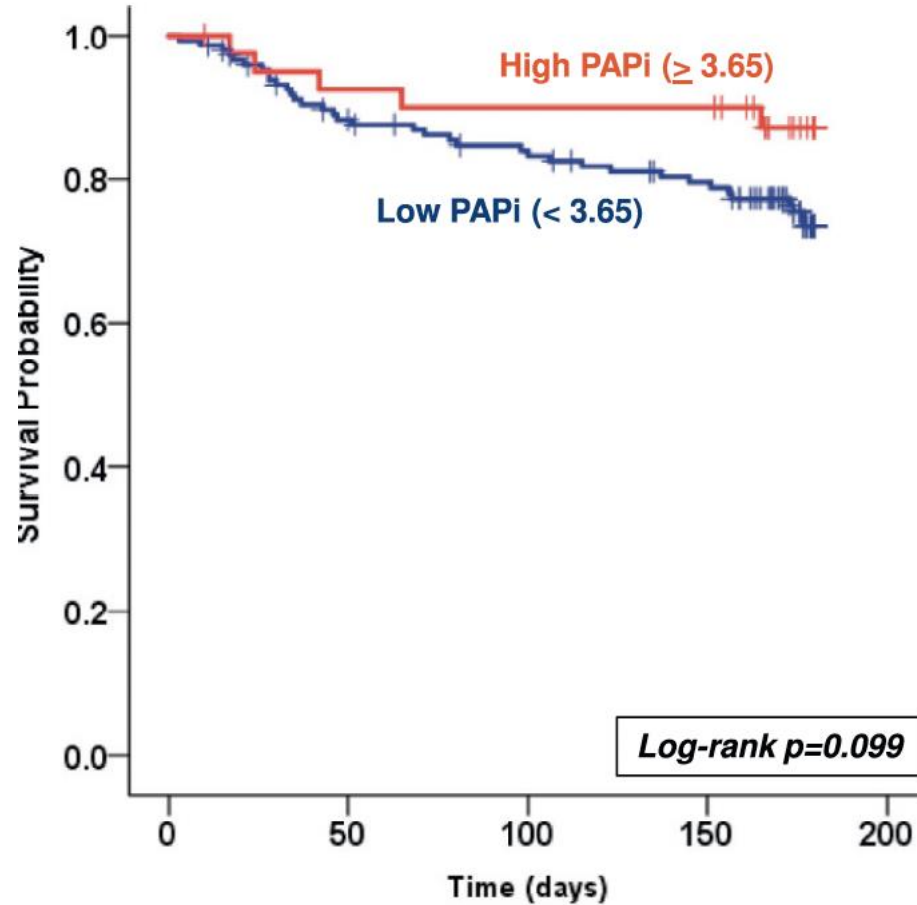
* Limited data; values may vary depending on vendor and software version.

- Estimate the RV's afterload (PASP, PVR)
- Estimate the RV contraction
- RV contraction seems to be o.k...estimate the RV-PA coupling
 - TAPSE/PASP (RV strain/PASP)
 - The ratio becomes lower when either the contractility declines or the RV afterload increases (...or when both take place)
- PAPI

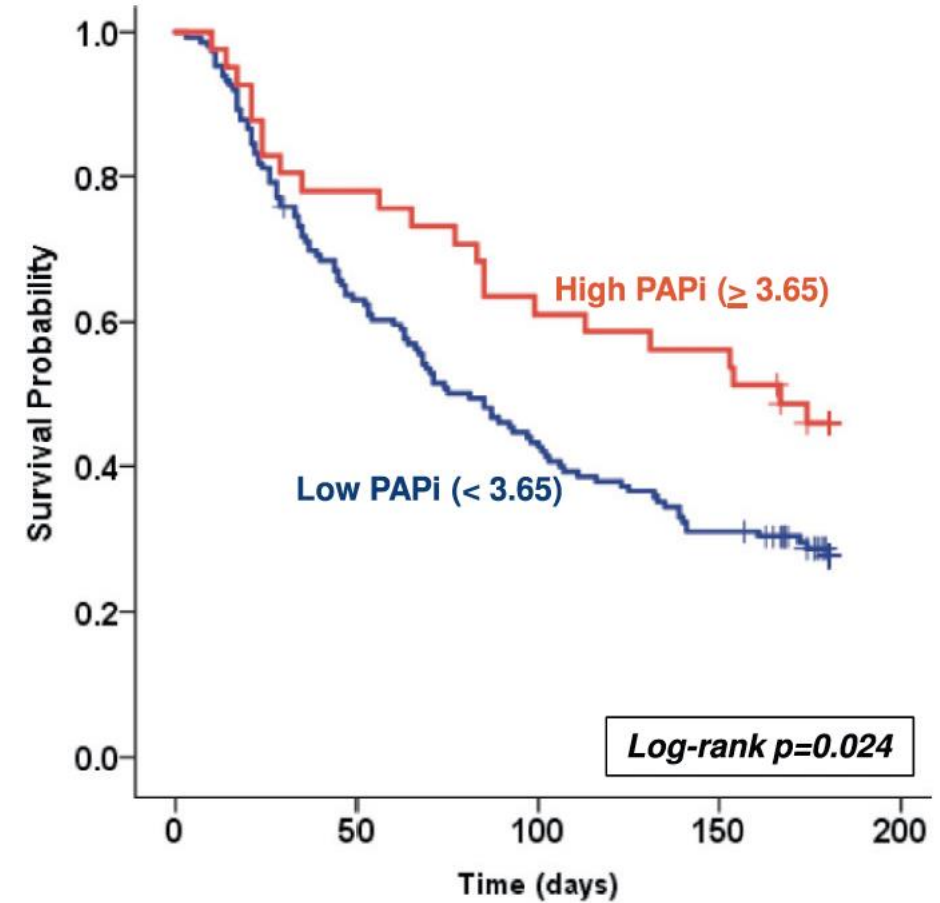
Traditional hemodynamic indices of RV function

- RAP
- RAP/PCWP
- RV stroke work index (RVSWI)
- PAPI
 - Higher PAPI is associated with lower PASP and RA pressure but not PADP.
 - Correlates significantly with indices of RV failure
 - Superior to the RAP/PCWP
 - Predicts in-hospital mortality and/or RVAD placement requirements.
 - Predicts RV failure and need for RVAD support following LVAD implantation
- Not only the RV: PAPI also correlates with LVEF, PCWP, and cardiac index.

Freedom from Death



Freedom from Death or Rehospitalization



Summarizing the Detrimental Factors Affecting the RV

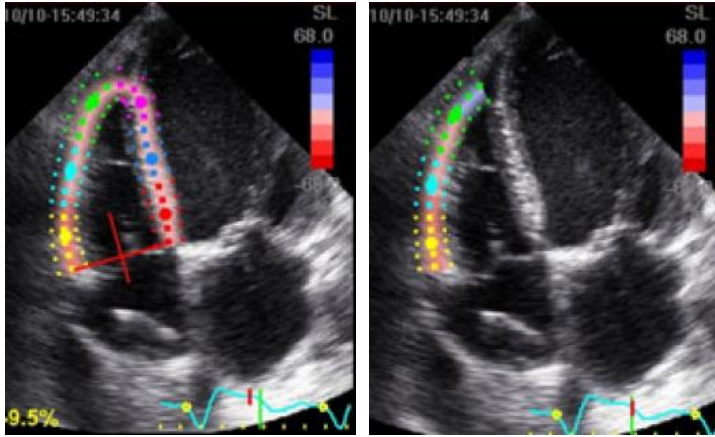
- RV-PA uncoupling (increased afterload)
- Altering the inter-ventricular dependence
- Impairment of the RCA flow (pulmonary blood pressure exceeding systemic blood pressure)
- Unregulated RV preload

Conventional RV systolic parameters such as TAPSE and S' have some limitations, such as **load and angle dependence**, and they represent only the **displacement or function of a single RV segment**.

RV-PA coupling

RV strain

RV FAC



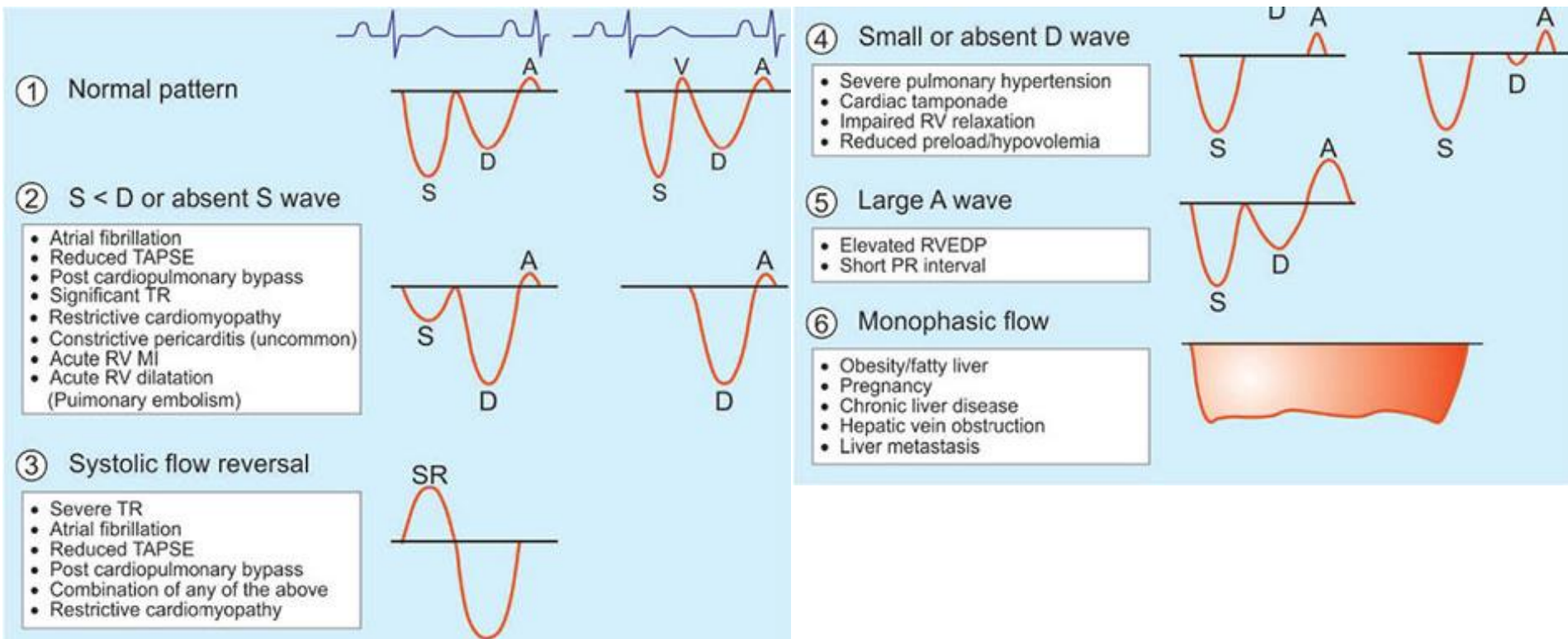
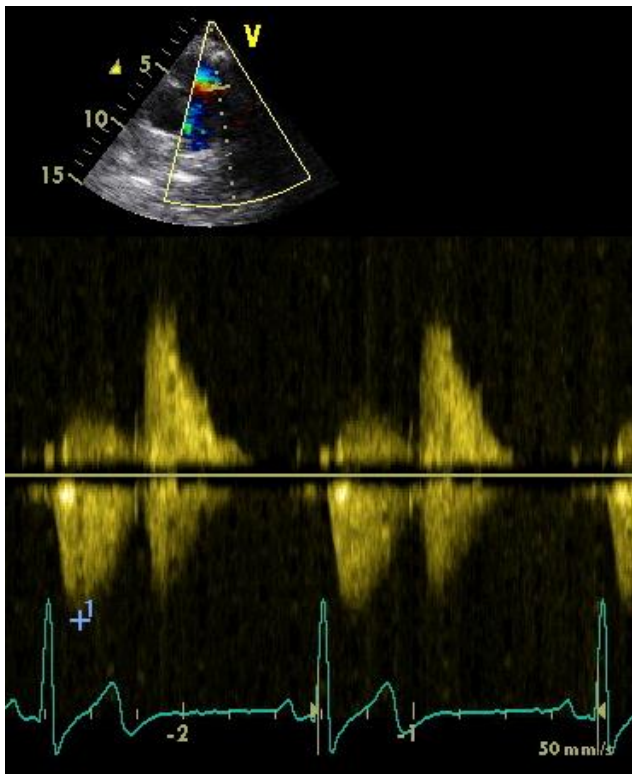
The EACVI suggested that a cut-off of RV-free wall systolic strain > -20%

Table 2 Myocardial RV systolic parameters using 2DSTE

	Healthy subjects (n = 238)	Asymptomatic patients (n = 216)	HFpEF patients (n = 218)	HFrEF patients (n = 208)
RV global (septal and free wall) systolic strain, %				
All patients	-24.5 ± 3.8	-22.4 ± 3.5	-20.7 ± 4.0	-15.3 ± 4.7
Lowest expected value	-17.0	n/a	n/a	n/a
Women	-25.0 ± 4.0	-22.9 ± 3.6	-21.0 ± 4.0	-15.5 ± 5.4
Lowest expected value	-17.1	n/a	n/a	n/a
Men	-23.9 ± 3.5 ^a	-22.0 ± 3.5	-20.5 ± 4.1	-15.2 ± 4.5
Lowest expected value	-17.0	n/a	n/a	n/a
Younger than 50 years	-24.3 ± 3.7	-22.2 ± 3.9	-20.6 ± 3.5	-15.9 ± 5.1
Lowest expected value	-17.0	n/a	n/a	n/a
Older than 50 years	-24.8 ± 3.9	-22.4 ± 3.4	-20.7 ± 4.1	-15.2 ± 4.6
Lowest expected value	-17.1	n/a	n/a	n/a
RV free wall systolic strain, %				
All patients	-28.5 ± 4.8	-26.7 ± 5.1	-24.6 ± 5.1	-19.0 ± 5.8
Lowest expected value	-19.0	n/a	n/a	n/a
Women	-29.0 ± 5.0	-27.3 ± 5.1	-24.6 ± 5.1	-19.6 ± 6.7
Lowest expected value	-19.2	n/a	n/a	n/a
Men	-27.9 ± 4.7	-26.3 ± 5.1	-24.7 ± 5.1	-18.9 ± 5.6
Lowest expected value	-18.7	n/a	n/a	n/a
Younger than 50 years	-28.4 ± 4.9	-26.8 ± 6.0	-25.0 ± 4.4	-20.6 ± 7.0
Lowest expected value	-18.8	n/a	n/a	n/a
Older than 50 years	-28.8 ± 4.5	-26.7 ± 4.8	-24.6 ± 5.1	-18.9 ± 5.7
Lowest expected value	-19.9	n/a	n/a	n/a

RV Diastolic function: the dark side of the study

- Look for the PR Doppler signal (slope, timing of flow termination)
- The wave pattern in the hepatic vein (S/D ratio, A amplitude)



Take Home Message

- RV is a complex chamber. Assessing its size and function requires integrating data from multiple perspectives and variables.
- Eyeballing estimation of the RV contraction is essential to the quantity assessment.
- Embrace the RV-PA coupling and RV strain.
- Diastolic function
- Investigate the etiology of RV dysfunction.
- Pursuit for (subtle) signs of RV dysfunction when LV dysfunction is present.

Thank You for Your Attention

