Right Heart Catheterization Overview

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Learning Objectives

- 1) Review Indications for RHC
- 2) Detail RHC procedure and interpretation of waveforms
- 3) Utilize hemodynamics to phenotype cardiogenic shock and classify PHTN

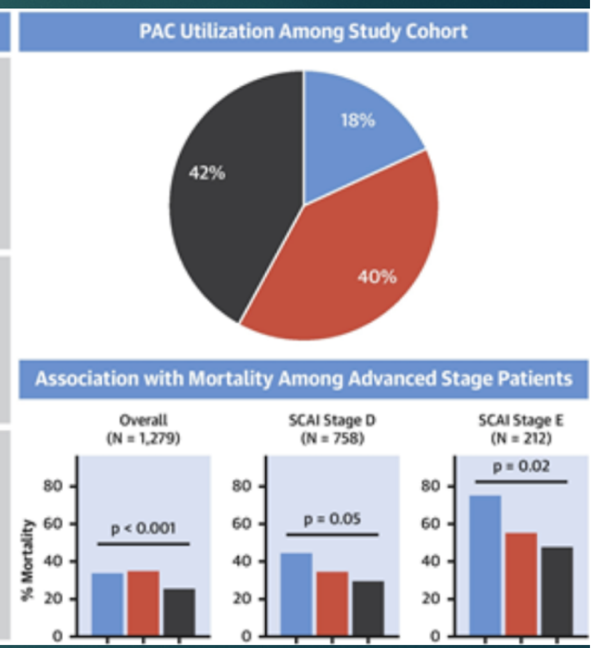
Indications

- Guiding medical therapy in decompensated HF and cardiogenic shock
- In patients being considered for advanced therapies
  - Class I indication for PVR measurement pre-listing***
  - Repeat every 3-6 months for listed patients
- Optimization of LVAD speed
- Confirm the diagnosis of PH and subsequently phenotype PH

"Escape ESCAPE"

- Prospective Trial (Rosello et al.) → short- and long-term mortality reduced with PAC in patients with CS
  - Mortality benefit in non-MI shock
- Garan et al. → complete PAC data prior to MCS resulted in significantly lower mortality.

PAC Utilization	Study Definition
None	Presence of **NONE** of the following invasive hemodynamics: • Pulmonary Artery Systolic Pressure • Pulmonary Artery Diastolic Pressure • Pulmonary Capillary Wedge Pressure • Pulmonary Artery Saturation • Right Atrial Pressure
Incomplete Assessment	Presence of **1-4** of the following invasive hemodynamics: • Pulmonary Artery Systolic Pressure • Pulmonary Artery Diastolic Pressure • Pulmonary Capillary Wedge Pressure • Pulmonary Artery Saturation
Complete Assessment	Presence of **ALL** of the following invasive hemodynamics: • Pulmonary Artery Systolic Pressure • Pulmonary Artery Diastolic Pressure • Pulmonary Capillary Wedge Pressure • Pulmonary Artery Saturation **AND** presence of Right Atrial Pressure

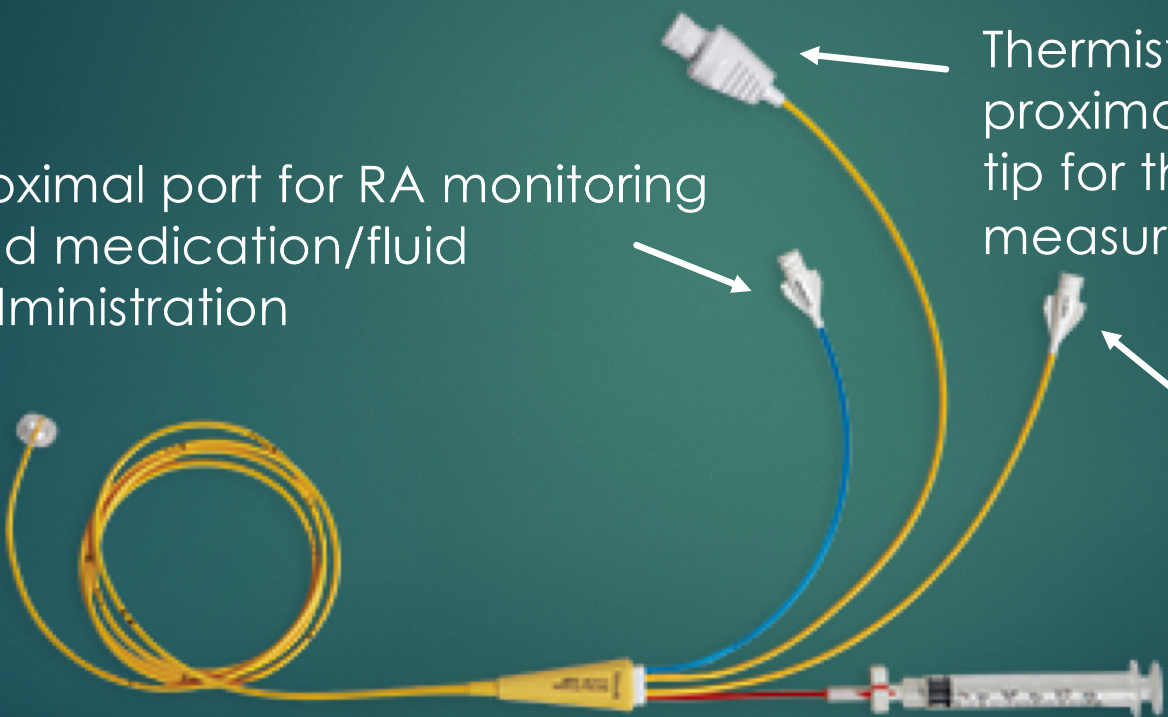


Key Point

- PAC is a DIAGNOSTIC tool that by itself cannot improve a patient's condition
- Therefore, focus should be on how to translate the HD information from PAC into appropriate interventions that lead to better outcomes
  - Earlier detection of clinical deterioration → expeditious escalation of support
  - Uncover RV failure that may need BiV support
  - Assessment of response/weaning from inotropes and MCS

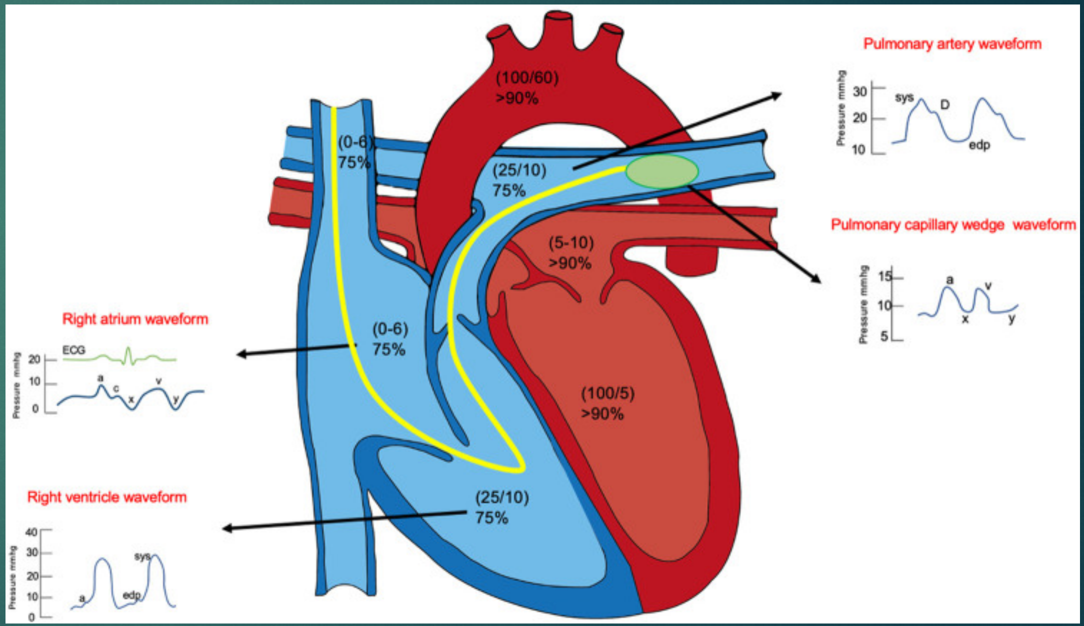
Mechanics

Anatomy of a Pulmonary Artery Catheter



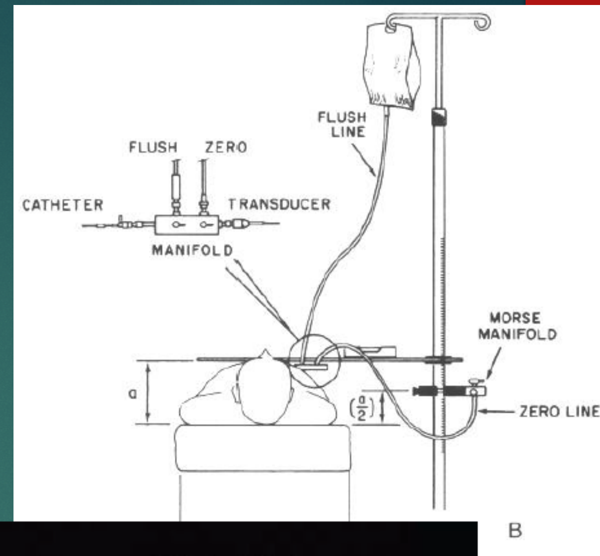
Placing the PA Catheter

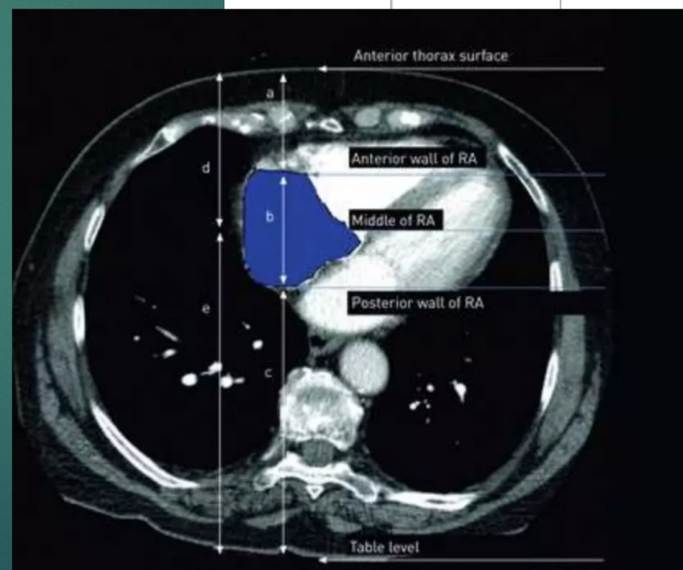
- Common routes of access include the IJ, femoral, or basilic veins
- Always inflate balloon while advancing and deflate while retracting
- Catheter, itself, is often curved to help guide placement



Taking Measurements

- The transducer must be leveled at about the mid chest level, about 5cm below the sternal border
- Pressure transducer should be zeroed to atmospheric pressure at the level of the LA.
  - LA is at midthorax in 97% of patients



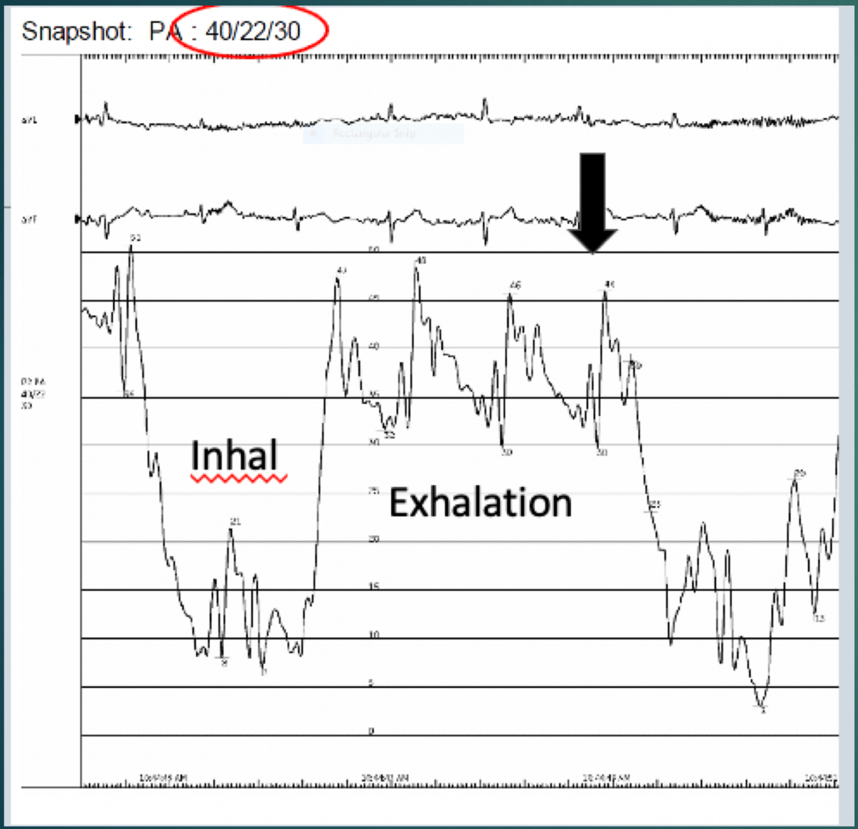


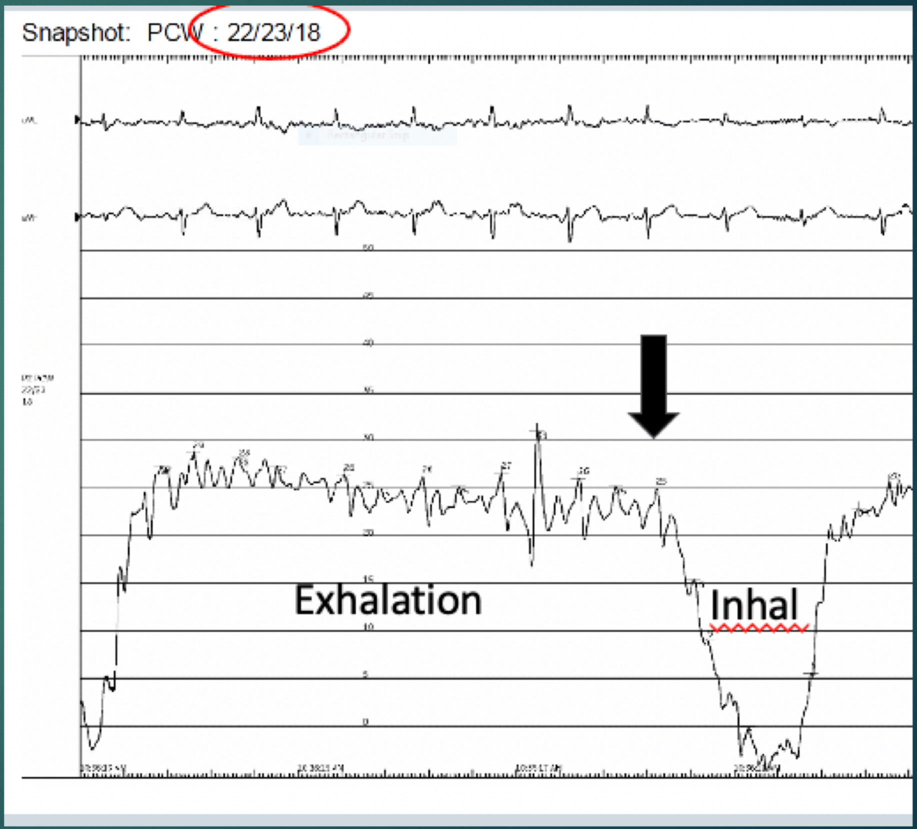
Baim DS and Grossman W. Cardiac Catheterization, Angiography, and Intervention. 5th Edition.

General Rules

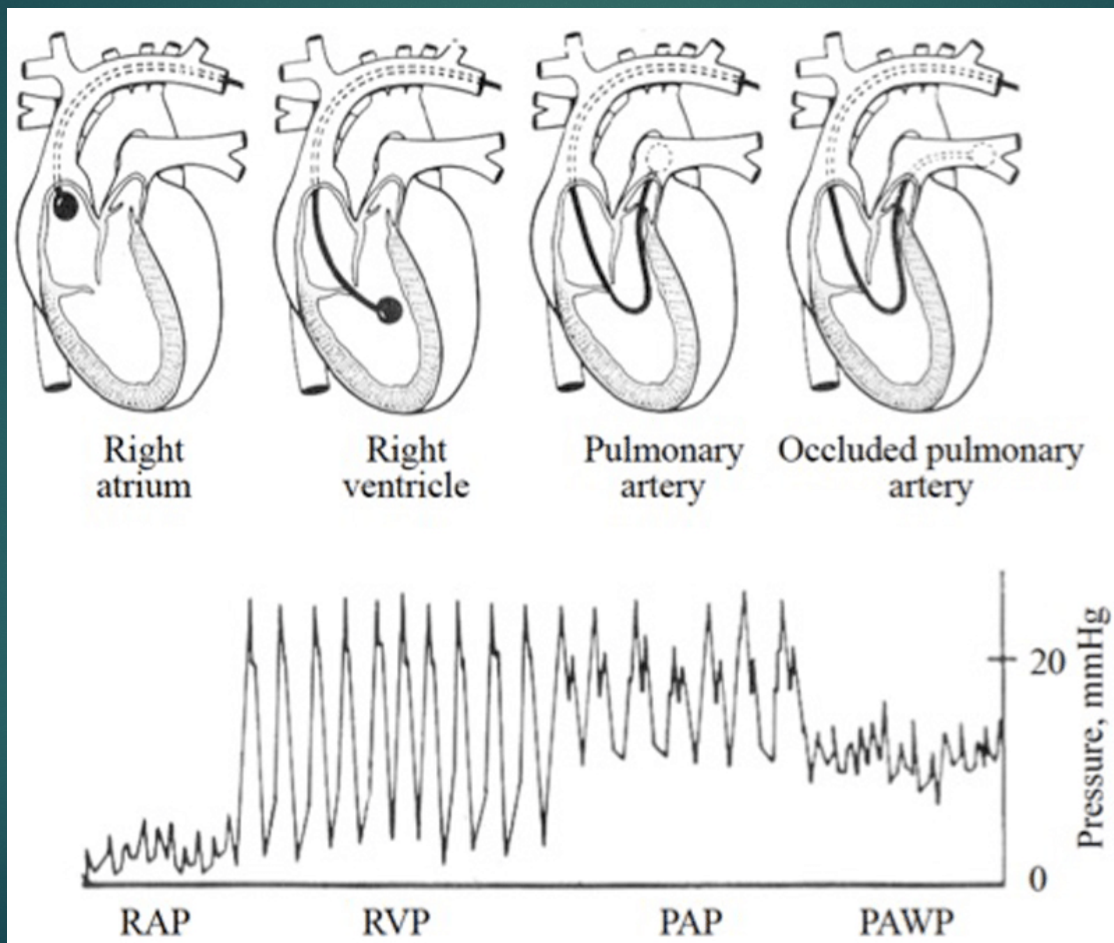
- Inhalation → drop in intrathoracic pressures → drop in pressure measurements
- All pressure measurements should be taken at end-exhalation
- Significant respiratory variation can be seen in obesity, COPD, OSA
- Always confirm computer measurements with personal review
- Downstream pressures can't be higher than upstream pressures (ie PCWP shouldn't be higher than PADP)

Breathing and Waveforms



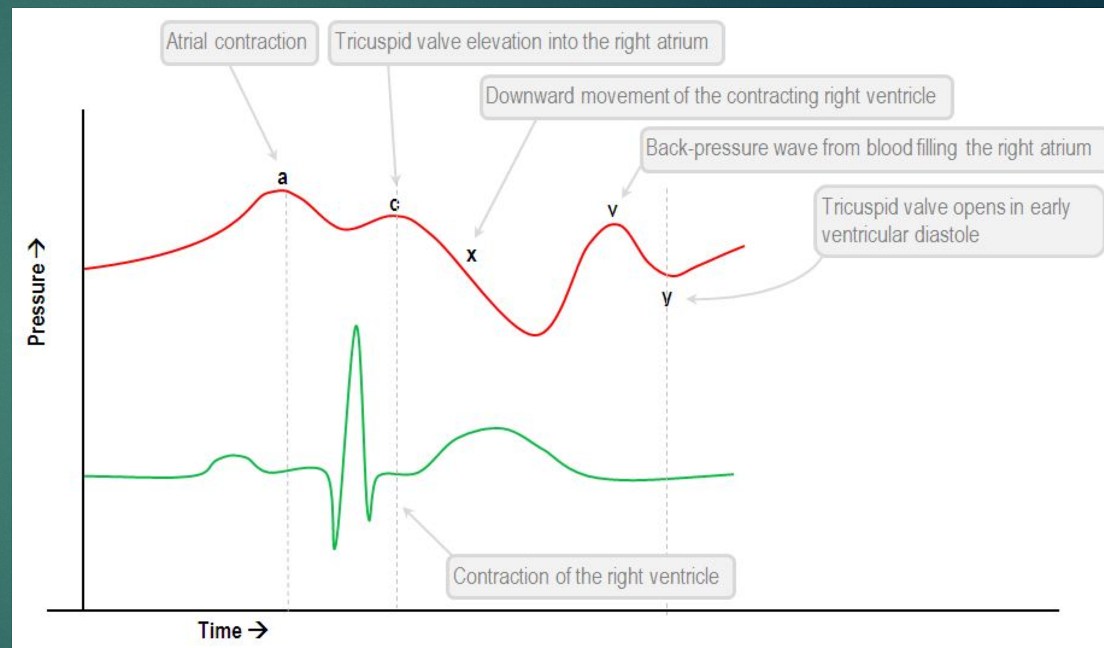


"As the Swan Swims"

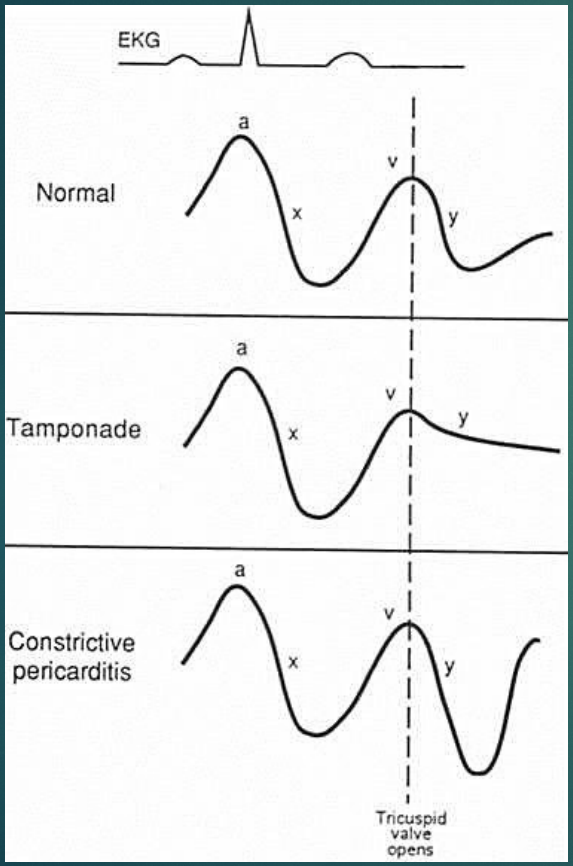


Right Atrial Tracing

- A wave: atrial systole
- X descent: RA relaxation and RV contraction
- V wave: RA filling with closed TV
- Y descent: TV opening and passive filling of the RV
- Normal RAP 7 mmHg or less
- Document presence/absence of Kussmaul sign

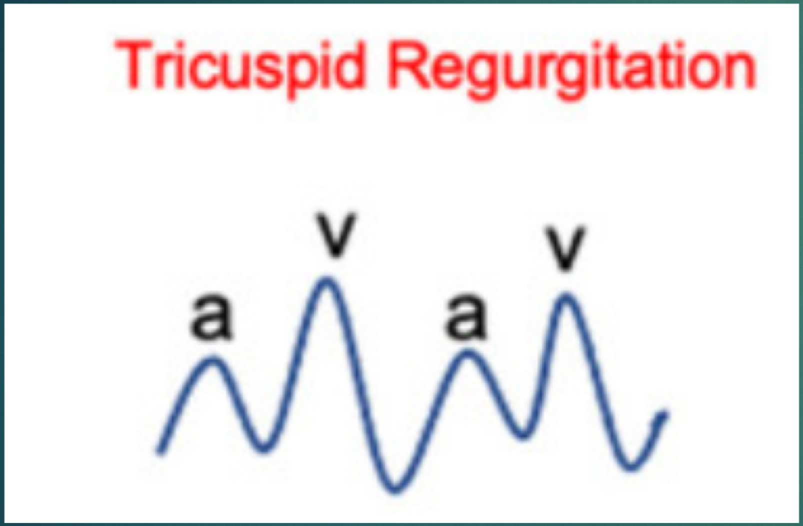


RAP Abnormalities



- Deep X and Y descents seen in constrictive pericarditis
  - Constriction limits total volume of blood that can be accommodated by heart during diastole
  - Accentuated early rapid ventricular filling 2/2 high atrial driving pressures/unimpeded ventricular relaxation → rapid Y descent
  - Prominent X due to preserved atrial relaxation and exaggerated ventricular longitudinal contraction
- Deep X (systole), shallow Y (diastole) in tamponade → flow to ventricles is impeded throughout all diastole, and subsequently little/no Y descent
  - "Flat Y Tamponade – FYT"

RAP Abnormalities



- Severe TR features a prominent V wave
  - V > 1.5X A wave or larger than mean RAP by >5 mmHg
  - RA fills from venous return AND regurgitant volume during systole
  - Other cause of prominent V wave?
    - Decompensated RV failure → RA volume overload surpasses RA compliance
    - Generally speaking, V wave size correlates inversely with atrial compliance

Other RAP Abnormalities

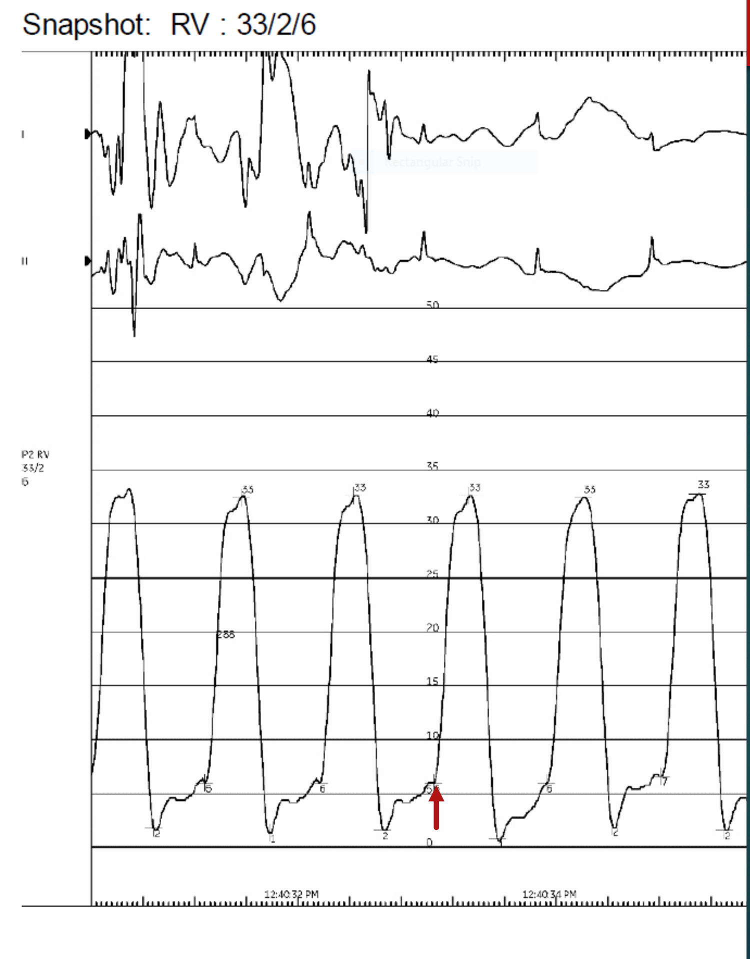
- Prominent A wave?
  - RVH, TS, AV dyssynchrony
  - Reflects reduced ventricular compliance → A wave correlates inversely with RV compliance (V wave correlates inversely with RA compliance)

Summary of RAP Abnormalities

- Deep X and deep Y → constrictive pericarditis
- Deep X and flat Y (diastolic flow blunting) → tamponade
- Large V wave → severe TR and/or RV failure
- Large A wave → impaired RV compliance, AV dysynchrony, TS

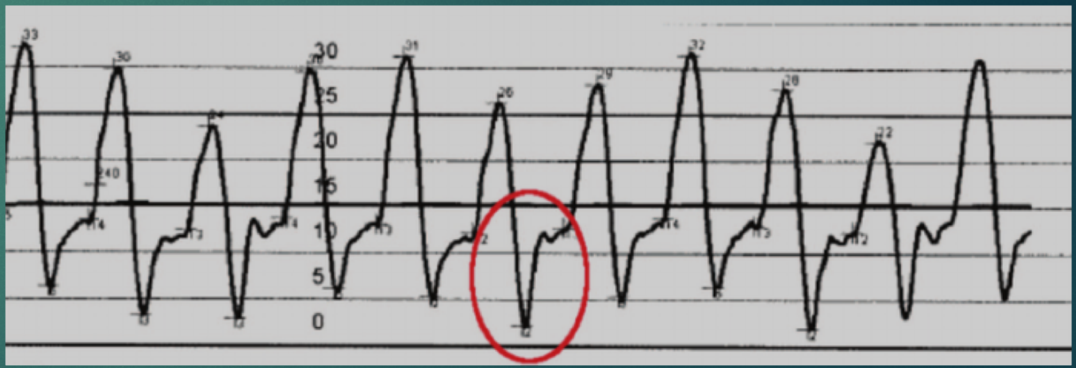
RV Pressure Tracing

- Step up in systolic pressure
- Normal RVSP is 35 mmHg or less
- Normal RVEDP is 8 mmHg or less



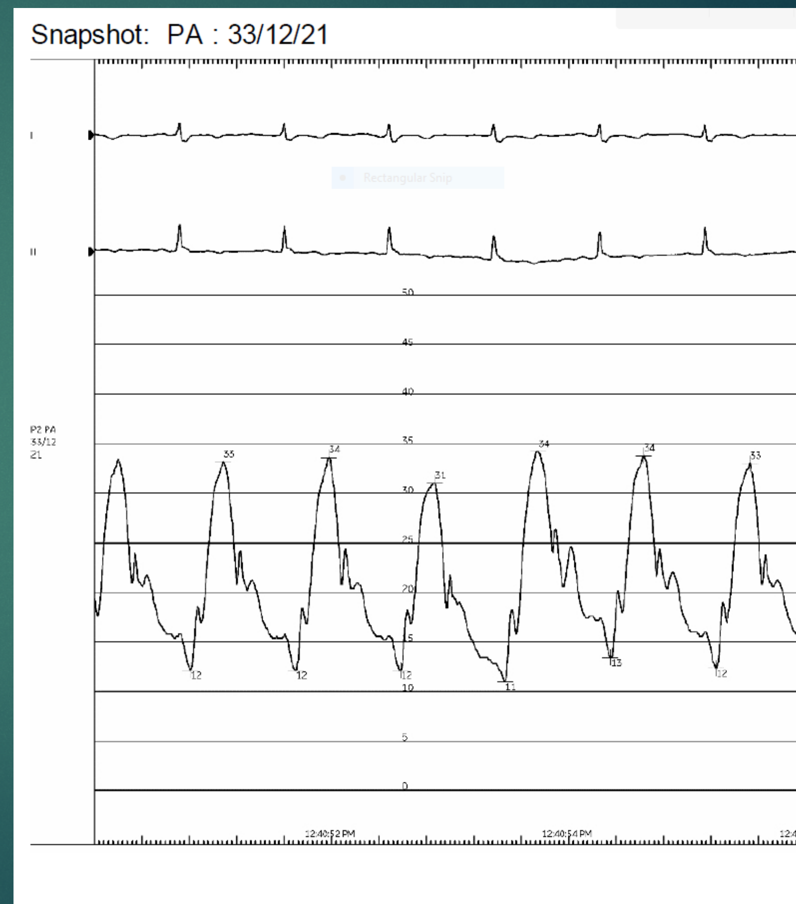
Dip and Plateau Sign

- Seen in RCM, CP, and RV failure. Sometimes in bradycardia as well
- Early diastolic dip
- Followed by "plateaued" high diastolic pressure → late diastolic filling is abbreviated and halts abruptly

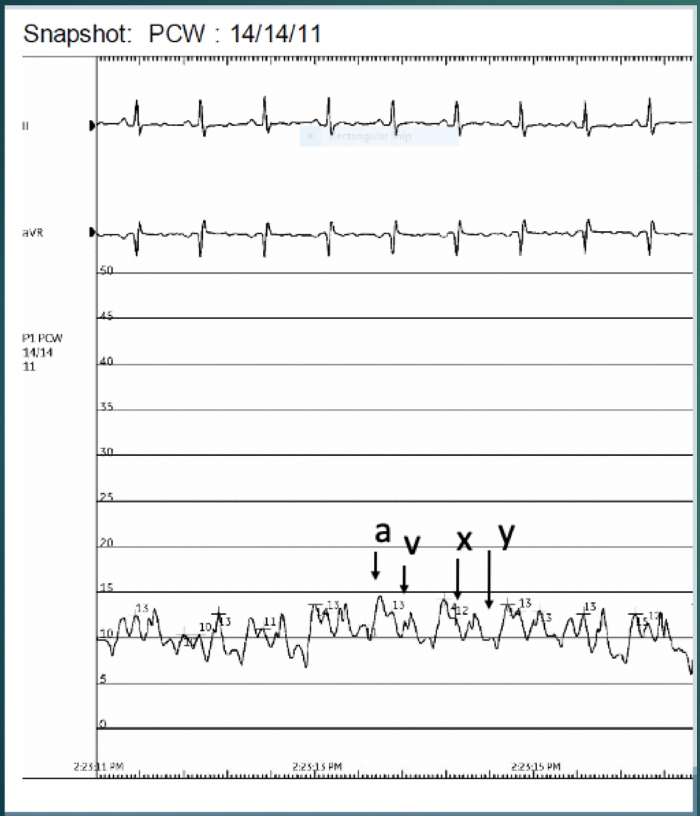


Pulmonary Artery Pressure Tracing

- Report systolic, diastolic, and mean pressures
  - What mPAP defines PHTN?
    - 20 mmHg.
  - Moderate PHTN >35 mmHG. Severe >45 mmHg
- Note dichrotic notch from closing of the PV
- Pressure falls during diastole (rises in RV due to filling)



Pulmonary Capillary Wedge Tracing

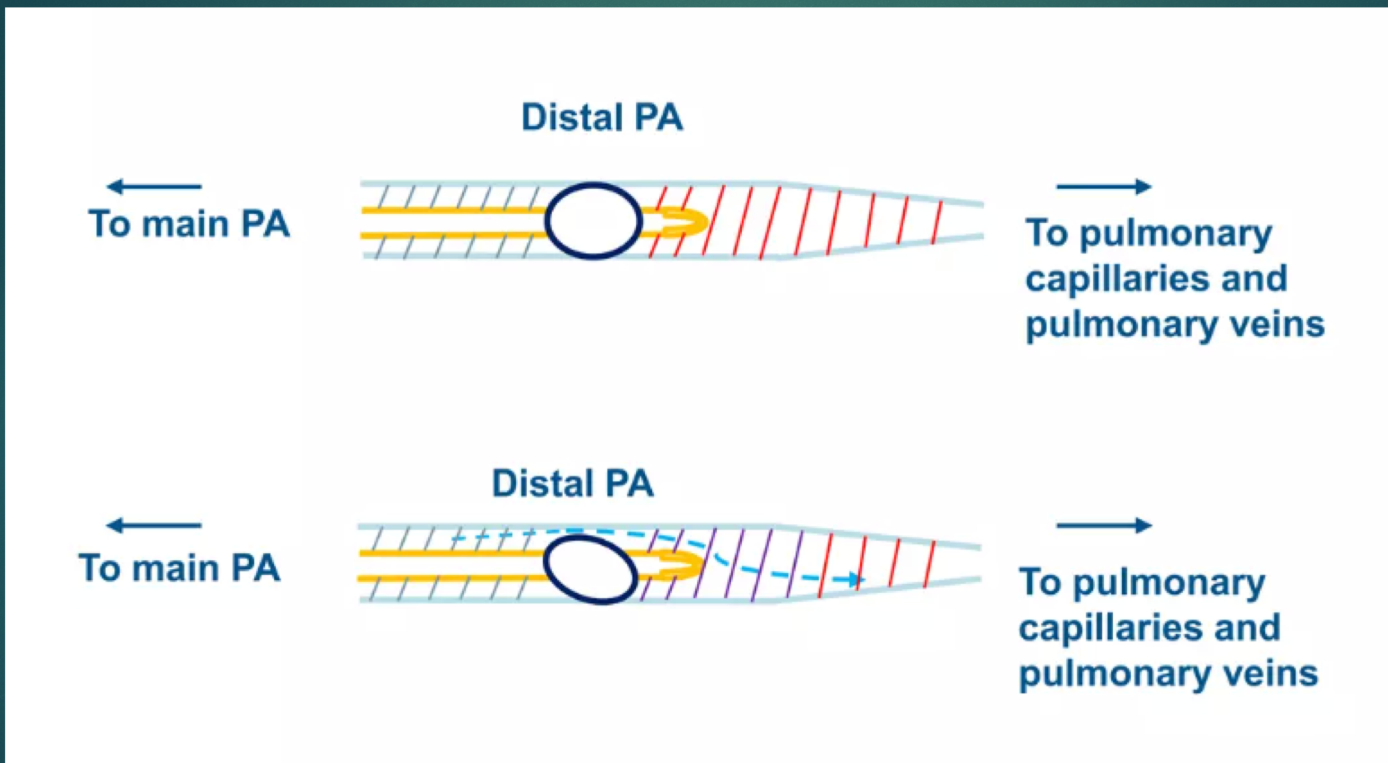


- A wave: left atrial systole
- X descent: LA relaxation and LV contraction
- V wave: LA filling with closed MV
- Y descent: MV opening and passive filling of the LV

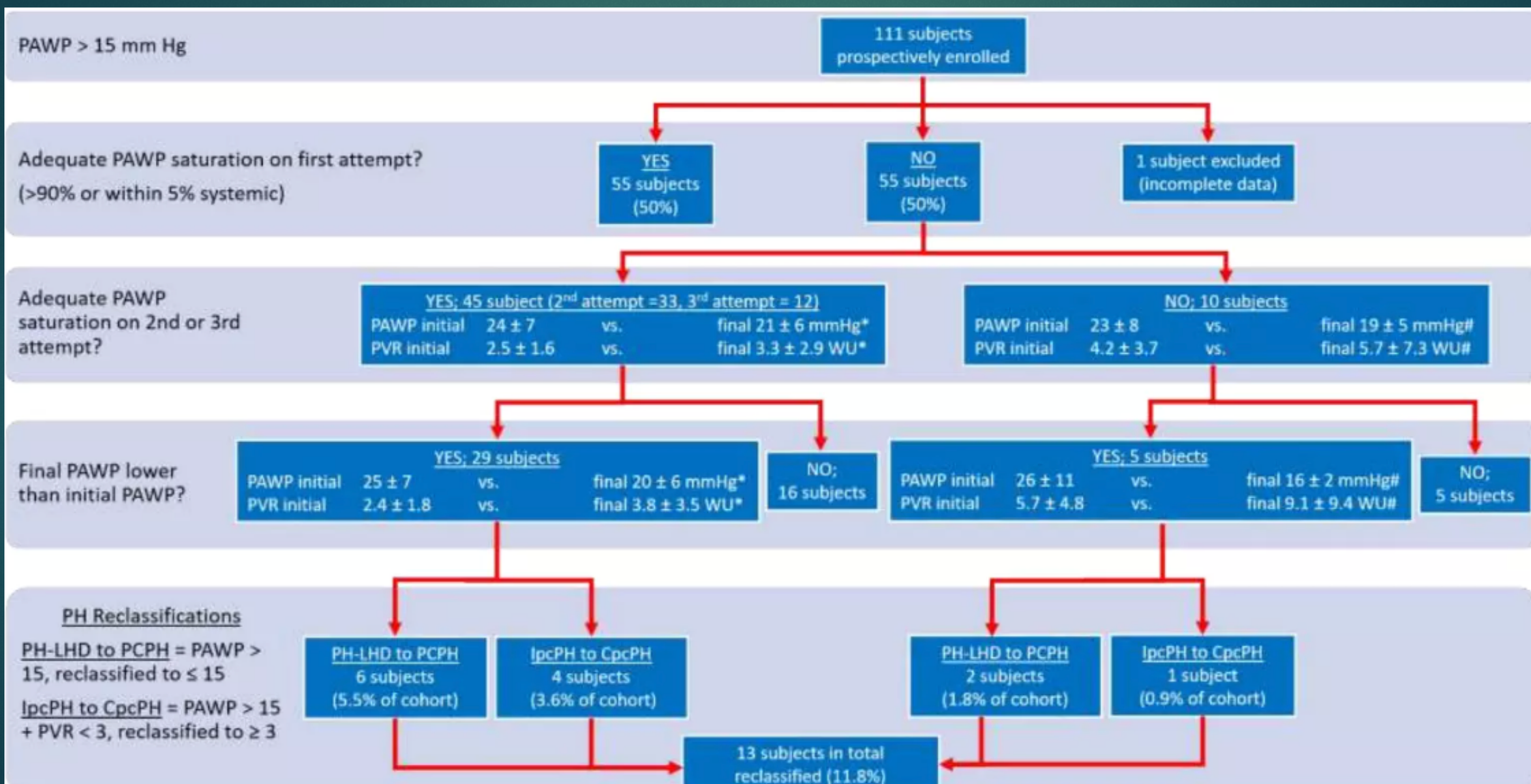
Pulmonary Capillary Wedge Tracing

- Approximation of LAP and – in absence of MS – LVEDP
- Measure at end-expiration and end-diastole → MV is open and LAP (and PCWP) should be equal to LVEDP
  - In sinus rhythm, average the A wave
  - In afib, measure the pressure 130-160 msec after QRS and before the V wave
- Normal PCWP is 12 mmHg or less (up to 15-18 mmHg may not lead to congestion in those with increased pulmonary capillary lymphatic drainage)
- Check a PCWP saturation whenever >15 mmHg to confirm occlusion
  - Truly wedged catheter → oxygen saturation >90% or within 5% systemic oxygen saturation

PCWP Sat


Distal PA
To main PA
To pulmonary capillaries and pulmonary veins
Distal PA
To main PA
To pulmonary capillaries and pulmonary veins

PCWP Sat

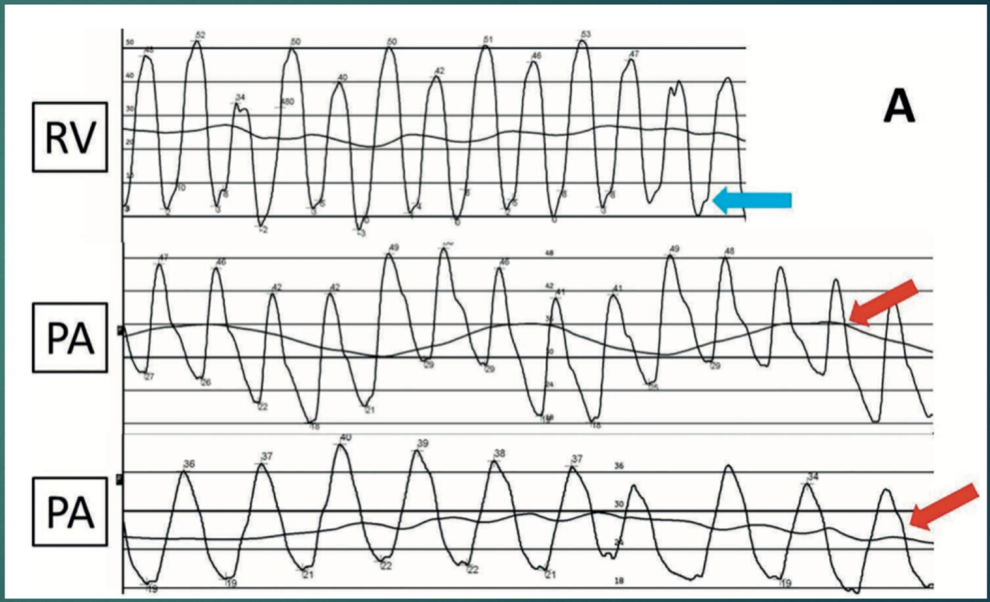

PAWP > 15 mm Hg
111 subjects prospectively enrolled
Adequate PAWP saturation on first attempt? (>90% or within 5% systemic)
YES 55 subjects (50%)
NO 55 subjects (50%)
1 subject excluded (incomplete data)
Adequate PAWP saturation on 2nd or 3rd attempt?
YES; 45 subject (2nd attempt =33, 3rd attempt = 12)
PAWP initial 24 ± 7 vs. final 21 ± 6 mmHg*
PVR initial 2.5 ± 1.6 vs. final 3.3 ± 2.9 WU*
NO; 10 subjects
PAWP initial 23 ± 8 vs. final 19 ± 5 mmHg#
PVR initial 4.2 ± 3.7 vs. final 5.7 ± 7.3 WU#
Final PAWP lower than initial PAWP?
YES; 29 subjects
PAWP initial 25 ± 7 vs. final 20 ± 6 mmHg*
PVR initial 2.4 ± 1.8 vs. final 3.8 ± 3.5 WU*
NO; 16 subjects
YES; 5 subjects
PAWP initial 26 ± 11 vs. final 16 ± 2 mmHg#
PVR initial 5.7 ± 4.8 vs. final 9.1 ± 9.4 WU#
NO; 5 subjects
PH Reclassifications
PH-LHD to PCPH = PAWP > 15, reclassified to ≤ 15
IpcPH to CpcPH = PAWP > 15 + PVR < 3, reclassified to ≥ 3
PH-LHD to PCPH 6 subjects (5.5% of cohort)
IpcPH to CpcPH 4 subjects (3.6% of cohort)
PH-LHD to PCPH 2 subjects (1.8% of cohort)
IpcPH to CpcPH 1 subject (0.9% of cohort)
13 subjects in total reclassified (11.8%)

Abnormal PCWP Tracing

- Large V wave (>10 mmHg larger than mean PCWP or 2X mean PCWP)
  - Severe MR → LA fills from venous return and regurgitant mitral flow
  - Decompensated LV systolic of diastolic failure → impaired LA compliance and increased LA volume beyond LA compliance
  - MS, VSD, other causes of impaired atrial compliance (atrial fibrosis)
- Large A wave → impaired LV compliance, AV dysynchrony, MS
- Absent A wave → A fib

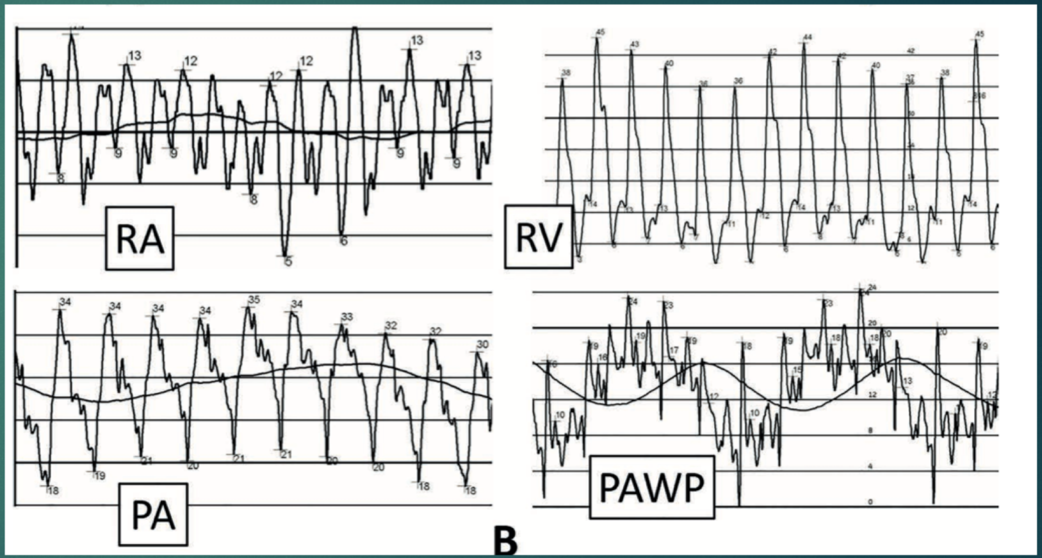
Overdampening

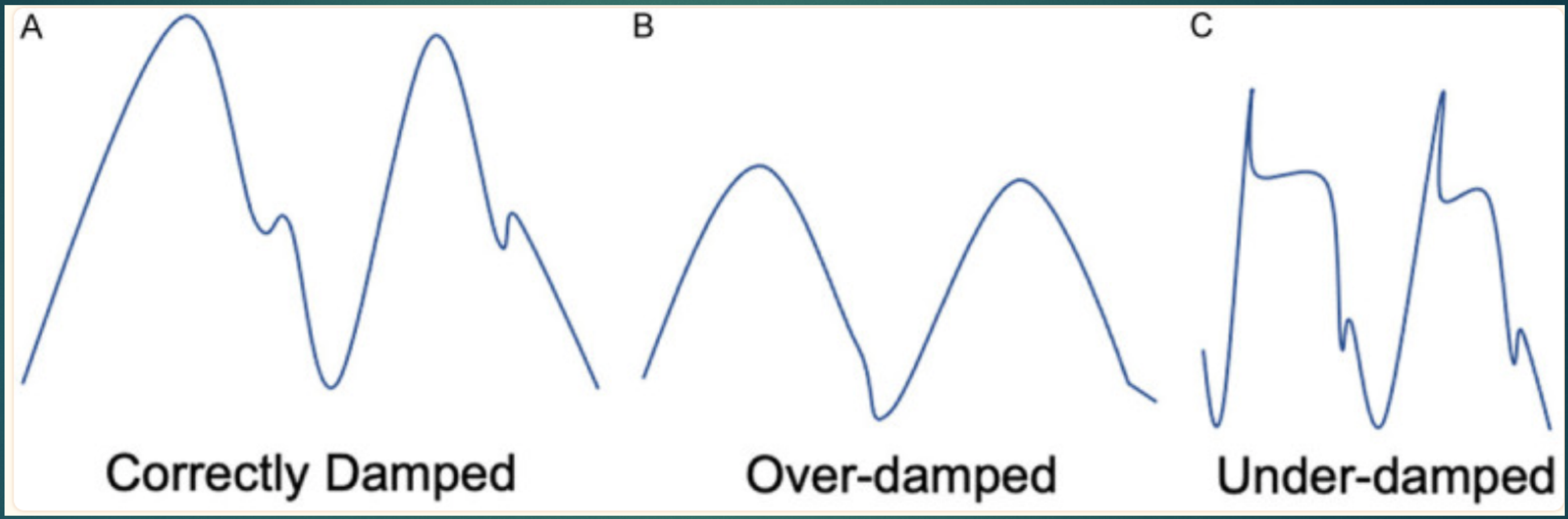
- Occurs when air is introduced into catheter or tubing
  - Loss of dichrotic notch in PA tracing
  - Blunted RVED inflection point
  - Reduction in amplitude of tracing
- Address by flushing



Catheter Ringing (Underdampening)

- Occurs when frequency of transmitted waveform (HR) approximates natural resonance frequency of transducer system
  - Falsely increases amplitude of waveform
  - May be exacerbated by microbubbles in system
- Address by flushing or introducing a denser fluid (blood) into catheter to alter resonant frequency




A
B
C
Correctly Damped
Over-damped
Under-damped

High-Yield Hemodynamics

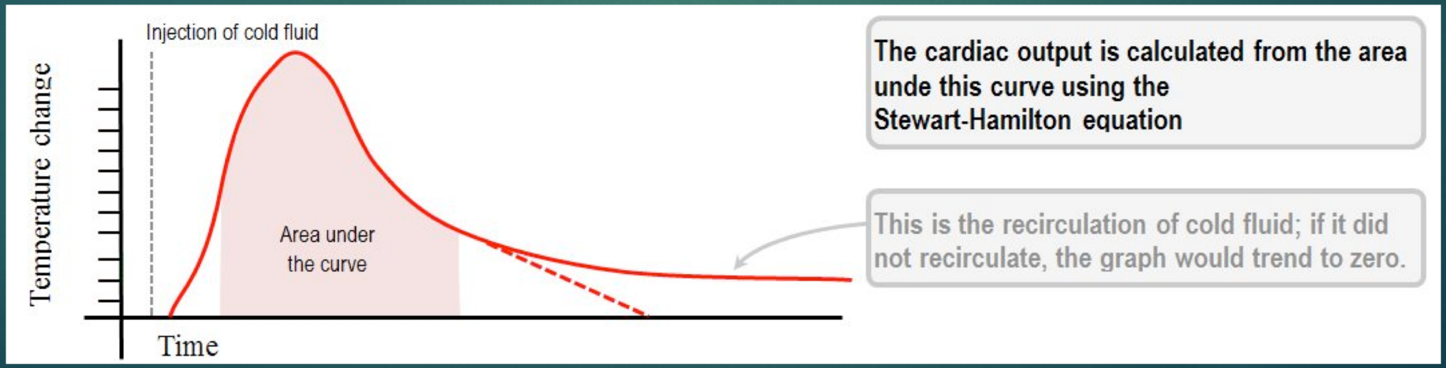
Cardiac Output

- Gold standard is direct Fick…but requires VO2 measurement
- Therefore, usually use indirect Fick or thermodilution
  - TD → thermal registering device measures changes in temp distal to proximal injection of saline of known temp and volume
    - Preferred method, even if TR or low CO
  - Indirect Fick → uses estimated values for O2 uptake from patient populations that were lean and homogenous (regarding age and race) → not the most applicable to HF, PH, obese population

Understand Hemodynamics: CO/CI by Thermodilution

Principle: Time to normalize RV temp after injection of cold saline will vary according to CO

- ↑ time = ↓ output
- ↓ time = ↑ output



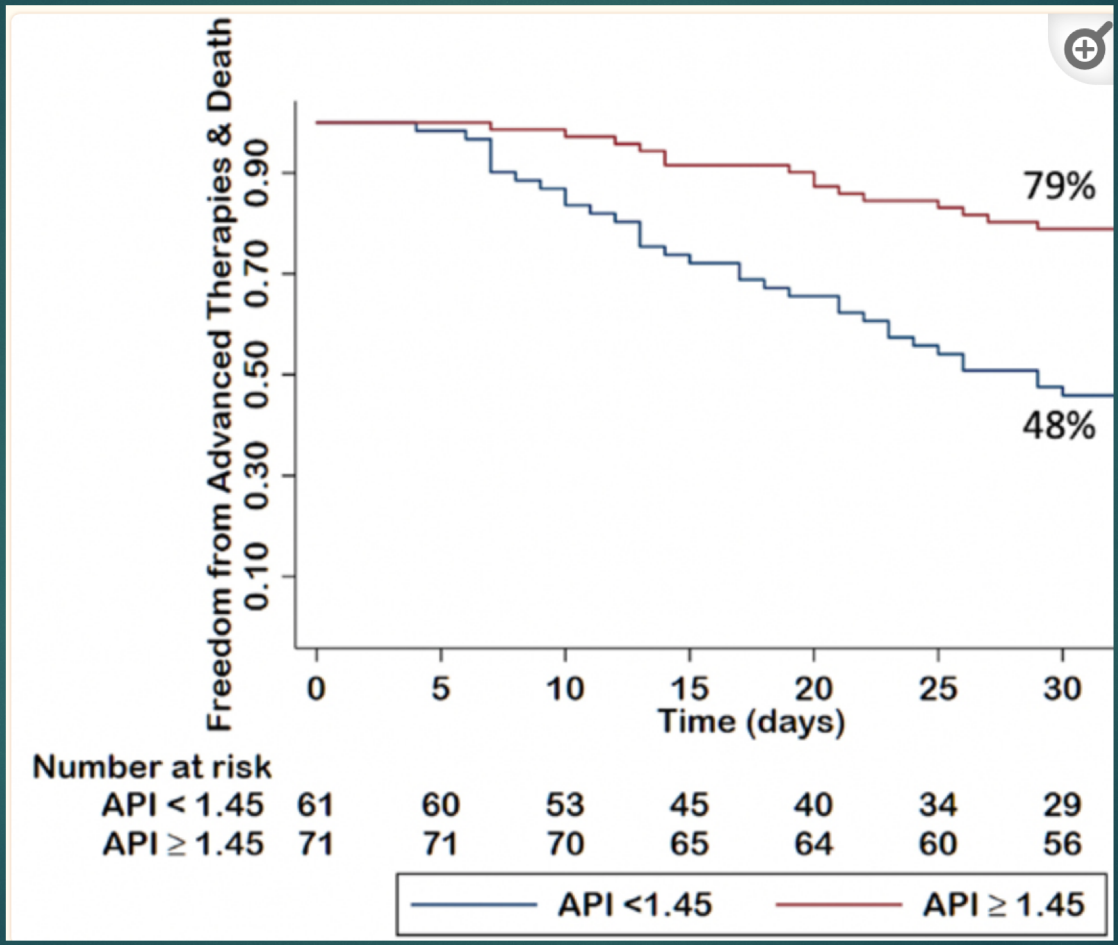
Calculations Focused on LV Performance

- CO → normal 4.0-8.0 L/min
- CI = CO/BSA → normal 2.5-4.0 L/min/m2
- SV index = CI/HR X 1000 → normal is above 35 mL
- CPO = [(MAP-RAP) X CO)]/451 → Abnormal if under 0.6
- Aortic Pulsatility Index (API) = (SBP – DBP)/PCWP → under 1.45 is abnormal

Should We be Using API Over CPO in HF-CS?

- HF-CS is a distinct pathophysiologic entity from AMI-CS
  - Area within PV loop represents stroke work, which correlates with CPO
  - In AMI-CS, acute drop in contractility → reduction in SV → CPO decreases (CPO highly prognostic)
  - Remodeling over time (RAAS) → volume retention, ventricular dilation, increased LVEDV → restores near-normal SW (CPO), but PV loop shifts right
  - AS PV loop shifts right, efficiency of heart (SW/ (potential energy + SW)) decreases
  - API accounts for loading conditions and cardiac efficiency, a feature absent in CPO

API


Freedom from Advanced Therapies & Death
0.10
0.30
0.50
0.70
0.90
79%
48%
0
5
10
15
20
25
30
Time (days)
Number at risk
API < 1.45 61 60 53 45 40 34 29
API ≥ 1.45 71 71 70 65 64 60 56
API <1.45
API ≥ 1.45

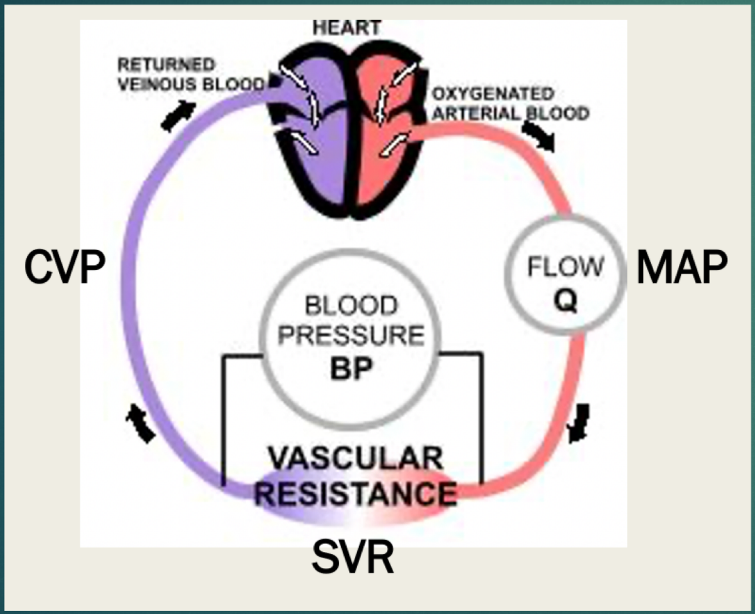
The RV!!!!!!

- PAPI (PA Pulsatility Index)
  - (PASP – PADP)/RAP
  - Abnormal cutoff depends on population
    - >2.0 is normal
    - <0.9 predicts RV failure and in-hospital mortality in inferior MI
    - <1.5-1.8 predicts RV failure in LVAD
- RA/PCWP
  - >0.6 associated with RV failure

HD Profiles of Shock

Hemodynamic Variables	Preshock Normotensive Hypoperfusion[25,26]	Preshock Hypotensive Normoperfusion[26]	LV Dominant Shock[1]	RV Dominant Shock[23,24]	BiV Shock[24]
Systolic arterial pressure, mm Hg	>90	<90	<90	<90	<90
CVP, mm Hg	Variable	Variable	<14	>14	>14
PCWP, mm Hg	Variable	Variable	>18	<18	Variable
CVP/PCWP	Depends on degree of LV and RV involvement	Depends on degree of LV and RV involvement	<0.86	>0.86	>0.86
PAPi (PAS – PAD)/RA[24,28–30]	Depends on degree of RV involvement	Depends on degree of RV involvement	>1.5	<1.5*	<1.5
Cardiac index, L/min/m^2	<2.2	≥2.2	<2.2	<2.2	<2.2
SVR, dynes-s/cm^{-5}	>1600	800–1600	800–1600	800–1600	800–1600
CPO, W[27]	Variable	Variable	<0.6	<0.6	<0.6

Vascular Resistance



- Resistance = ΔP / Q
- ΔP is the difference in pressure across the circulatory bed
- Systemic vascular resistance (SVR) = (MAP – RAP)/ CO
- Pulmonary vascular resistance (PVR) = (mPAP – PCWP)/ CO
  - mPAP – PCWP = transpulmonary gradient (normal < 12 mmHg)
- These equations give results in Wood units
  - Multiply by 80 to get metric units in dyne*sec*cm^-5

Vascular Resistance

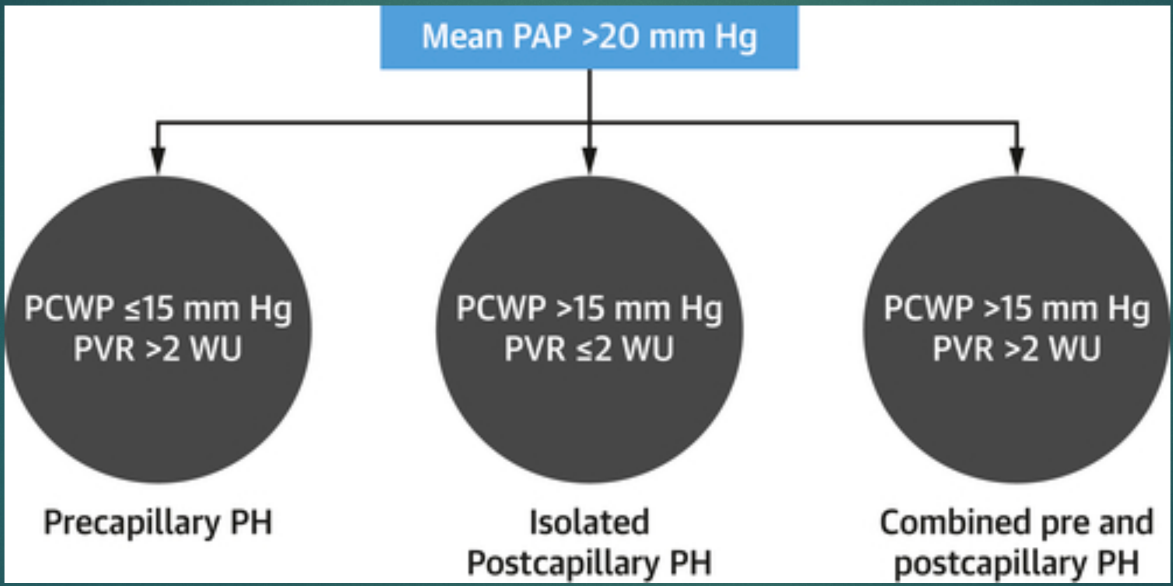
SVR

- Normal is 800-1200 dyne*sec*cm^-5
- Increased
  - Cardiogenic shock
  - HTN
- Decreased
  - AVF
  - Vasodilation (sepsis, thyrotoxicosis)
  - Inappropriately high CO (cirrhosis)

PVR

- Normal <2 WU (<240 dyne*sec*cm^-5)
- Increased in PHTN

Pulmonary Hypertension



Vasodilator Testing Algorithm

Pulmonary Hypertension
(mPAP > 20 and/or PVR > 3 WU)

Pre-Capillary
(PCWP ≤ 15 mmHg)

Pulmonary vasodilator testing
- Inhaled Nitric Oxide
- 20 ppm for 4 min followed by 40 ppm for 4 min

Post-Capillary
(PCWP > 15 mmHg)

Systemic vasodilator testing
- IV nitroprusside
- Increase by 0.25 mcg/kg/min every 2 minutes
- Until hypotension or max dose of 5 mcg/kg/min

Pulmonary Vasodilator Responsiveness

- Decrease in the mean PAP of at least 10 mmHg to a value of less than 40 mmHg during inhaled nitric oxide
- Indicates potential responsiveness to calcium channel blocker therapy in primary pulmonary hypertension

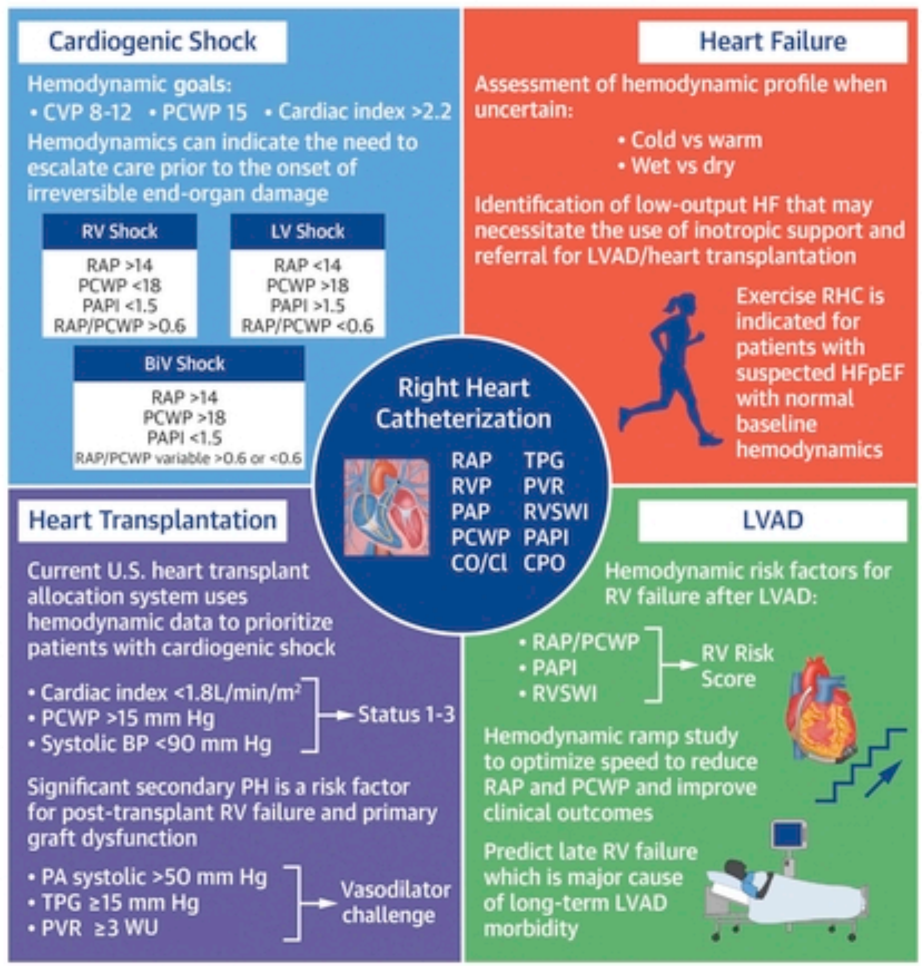
Systemic Vasodilator Testing

- Evaluate response to vasodilator therapy in patient with left sided heart failure
- Ensure that PVR is reversible in patients being evaluated for heart transplant
- Irreversible PVR > 4-5 mmHg is a contraindication to transplant
  - Indicates intrinsic pulmonary vascular remodeling and is a risk factor for graft failure after transplant

Equations to Know for RHC

- Transpulmonary gradient
- PVR (in WU)
- SVR (in dyne * s/cm^2)
- PAPi
- CPO
- API

CENTRAL ILLUSTRATION: Use of Right Heart Catheterization in Heart Failure



Rajagopalan N, et al. J Am Coll Cardiol HF. 2024;12(7):1141-1156.