PULMONARY HYPETENSION

Normal mean PAP ranges from 8-20 mmHg at rest. Pulmonary hypertension is defined as mean PAP at rest of > 25 mmHg.

Causes of pulmonary hypertension

Pulmonary hypertension results from an increased resistance to blood flow through the pulmonary vasculature. The World Health Organization categorizes the causes of pulmonary hypertension into a number of groups that share similar pathophysiology, clinical features and therapeutic approaches:

- **Group 1: Pulmonary arterial hypertension**: e.g. idiopathic, heritable, drug or toxin induced, connective tissue disease (SLE and systemic sclerosis), persistent pulmonary hypertension of the new-born, pulmonary veno-occlusive disease and/or pulmonary capillary haemangiomatosis
- **Group 2:** Pulmonary hypertension **due to left heart disease**: e.g. valvular disease (MR and MS), LV systolic or diastolic dysfunction (ischaemic CM), left to right shunts.
- **Group 3: Lung diseases and/or hypoxaemia**: e.g. COPD, ILD, sleep-disordered breathing
- **Group 4: Chronic thromboembolic disease**: e.g. pulmonary embolism
- **Group 5:** Pulmonary hypertension with **unclear multifactorial mechanisms**

In addition, PH can be categorized into either post-capillary when the PWP is > 15 mmHg (group 2) or pre-capillary when the PWP is <15mmg (groups 1, 3 & 4, in addition to PH with unclear multifactorial mechanisms).

Echo assessment of pulmonary hypertension

1. Calculation of PASP: using CW Doppler, measure the peak velocity of regurgitant flow through the tricuspid valve (TR Vmax) in m/s. This is best measured in A4C, PSAX (AV level) or parasternal RV inflow. The TR is driven by, and therefore reflects, the pressure gradient between RVSP and RAP, and this pressure gradient can be calculated using the simplified Bernoulli equation: RVSP − RAP = 4 × (TR V_{max})^2. Assuming there is no pulmonary stenosis, the RVSP is approximately equal to the PASP and, accordingly, PASP = (TR V_{max})2 + RAP. Recent guidelines have placed more of an emphasis on using the value of TR V_{max} itself to identify individuals with pulmonary hypertension, rather than trying to calculate the PASP. The BSE defines the normal upper limit of TR Vmax as 2.6 m/s (or 2.8 m/s in obese patients, or 2.9 m/s in individuals aged >60 years). A peak TR velocity > 2.5 m/s is associated with increased mortality, increased hospitalisation and higher incidence of heart failure.

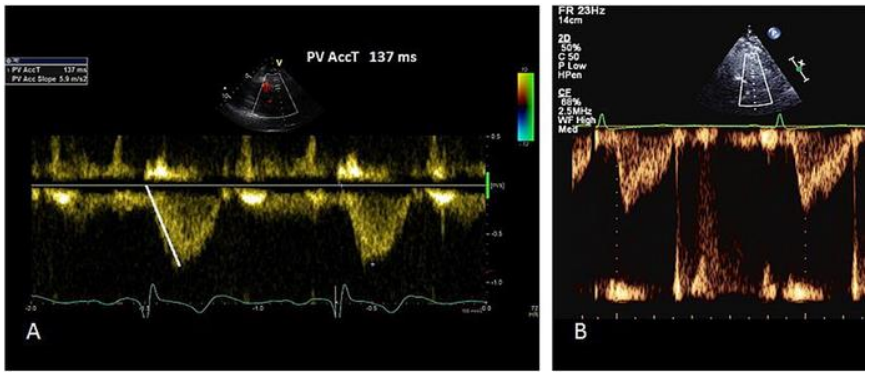
Similarly, The VSD gradient is driven by, and therefore reflects, the pressure gradient between LVSP and RVSP, and this pressure gradient can be calculated using simplified Bernoulli equation: RVSP = LVSP – (VSD V_{max})^2 and assuming there is no pulmonary stenosis, the RVSP is approximately equal to the PASP and, accordingly PASP = SBP – (VSD V_{max})^2.

Remember: The TR jet calculation overestimates the PA pressure in the presence of PS, and PA pressure in this instance should be calculated by PAP = (TR gradient + RAP) –PS gradient.

EACVI defines pulmonary hypertension as PASP>50 mmHg at rest and > 60 mmHg at exercise

2. Calculation of PADP: estimation of PADP relies on the presence of PR. You can assess PR using CW Doppler in PSAX (AV level) or parasternal RV outflow view. Using CW Doppler, measure the PR V_{max} in m/s. This is best measured in PSAX (AV level) or parasternal RV outflow. The PR is driven by, and therefore reflects, the diastolic pressure gradient between the PADP and the RVDP, and this pressure gradient can be calculated using simplified Bernoulli equation: PADP − RVDP = 4 × (PR V_{max})^2. The actual RVDP is taken to be the same as RAP and, accordingly, **PADP** = (PR V_{max})^2 + RAP

3. PAT (Pulmonary Acceleration Time), also called RVOT acceleration time or Time to peak velocity– normal pulmonary flow has a symmetric contour with a peak velocity occurring in mid-systole. In PH, peak velocity occurs earlier in systole and the Doppler profile begins to resemble aortic flow with a mid/late-systolic notch similar to the dicrotic notch seen in normal aortic flow. The acceleration time (time from onset to peak flow velocity) of the RVOT flow can be measured on PW tracing in PSAX with breathing held at end expiration. Reduced acceleration time <105 ms is supportive of PH. However, the short time to peak velocity in the PA due to PHT is offset in RV dysfunction by the inability of the RV to contract sufficiently vigorously, causing the time to peak velocity to appear normal.



RVOT acceleration time method for assessing pulmonary pressure. A—Pulmonary acceleration time measurement. B—Rapid rise and mid-systolic notching suggesting elevated pulmonary pressure.

4. RV Isovolumetric relaxation time ($IVRT_{RV}$)- is a measure of RV diastolic dysfunction. It can be measured by placing a PW Doppler midway between the MV and AV in the A5C, so that trans-mitral and trans-aortic flows are picked up; the time duration between them is the IVRT. It can also be measured using PW TDI in the A4C, placing the sample volume at the basal lateral RV myocardium. The measurement is taken at end-expiration from the offset of S' to onset of E'. The $IVRT_{RV}$ is prolonged in PH because the high PA pressures cause the PV to close earlier than normal (mid-systolic closure of PV). A shortened $IVRT_{RV}$ < 40 msec. has a high negative predictive value for PHT.

Additional features suggestive of pulmonary hypertension:

- RA dilatation
- RV dilatation, hypertrophy and/or impairment.
- 'RV: LV' diastolic ratio- this is used to assess RV enlargement. It is best viewed in the PLAX. RV: LV diastolic ratio > 0.5 suggests pulmonary hypertension
- D-shaped LV is a pathological finding, suggesting the presence of pulmonary hypertension
- Paradoxical septal motion
- Dilatation of the pulmonary artery. Main PA diameter > 2.1 cm in the PSAX is considered abnormal. A PA diameter greater than the aorta suggests pulmonary hypertension, but a main PA diameter of > 3 cm is also suggestive.
- Pulmonary valve closes increasingly earlier as PA diastolic pressure rises in pulmonary hypertension
- Hepatic vein diastolic flow reversal is seen in PH and constrictive pericarditis. Respiratory variation helps differentiate between them. It is augmented with expiration in constriction, whilst in PH it remains constant.
- Pulmonary and/or tricuspid regurgitation
- Abnormalities of pulmonary valve by M-mode:
  - **Absent "A" wave of the pulmonary valve**, in spite of normal sinus rhythm due to ↑ the PA diastolic pressure → ↑ the RV diastolic pressure → the RA contraction will have no effect on the pulmonic valve end-diastolic position
  - **Mid-systolic notching of the pulmonary valve (flying "W" sign)** due to early closure of pulmonary valve because of high PVR
  - **Flat pulmonic EF slope** of the pulmonary valve

Estimation of RAP (mmHg)

Normal sized and totally collapsing IVC → 0-5 mmHg
Normal sized and > 50% collapsing IVC → 5-10 mmHg
Normal sized and > 50% collapsing IVC → 10-15 mmHg
Dilated (> 2.1) and < 50 % collapsing IVC → 15-20 mmHg
Dilated and not collapsing IVC → >20 mmHg

RAP (mmHg)	**0–5**	**5–10**	**10–15**	**15–20**	**> 20**
IVC size (cm)	< 1.5	1.5–2.5	1.5–2.5	> 2.5	> 2.5
IVC Respiratory/sniff variation	collapse	↓> 50%	↓< 50%	↓< 50%	No change
RA size	normal	normal	↑	↑↑	↑↑
Hepatic vein size				↑	↑↑

Hints:

- Dilated hepatic veins usually occur when RA pressure has reached at least 15 mmHg
- Positive pressure ventilation changes the normally negative intra-thoracic pressures required to cause inspiration, therefore evaluation of the IVC collapse due to respiration is not helpful.
- According to the AHA scientific statement on management of PHT, fibrinolysis may be considered for patients with submassive acute PE judged to have clinical evidence of adverse prognosis (new hemodynamic instability, worsening respiratory insufficiency, severe RV dysfunction, or major myocardial necrosis) and low risk of bleeding complications (Class IIb; Level of Evidence C).
- **McConnell's sign** (akinesis of the basal segment with normal apical and mid-wall contraction of the RV) is seen sometimes in large PEs, albeit an unreliable sign. Unhelpfully, the reverse McConnell sign is also recognised in acute PEs.