Pulmonary Hypertension

Clinical Features

1. Dyspnea initially on exertion & later at rest.
2. Dull, retrosternal chest pain (due to coronary blood flow to a marked hypertrophied RV)
3. Syncope or near syncope due to fixed cardiac output.

Signs

- Inspection
  - Prominent a wave in JVP
  - Cyanosis in late stage due to systemic V.C resulting from markedly reduced COP

- Palpation
  - Left parasternal heave (due to RV pressure overload → RVE)
  - Palpable PS in pulmonary area (due to dilated pulmonary artery)

- Auscultation
  - Accented S2
  - S1
  - S4
  - Murmurs (PS, PR, TR)
  - Pansystolic murmur (due to pulmonary artery pressure exceeds aortic artery pressure → reversal shunt)

Investigations

- ECG
  - RV hypertrophy
  - RA hypertrophy

- X-ray
  - RV enlargement
  - RA dilatation
  - Enlargement of Pulmonary artery & its main branches

- Echo
  - RV & RA enlargement
  - Detection of heart defects
  - Thickened interventricular septum
  - Abnormal septal motion due to RV pressure overload

- Cardiac catheterization
  - Confirm the diagnosis e.g. detection the defect
  - Measure pulmonary artery pressure to assess severity
  - Measure the O2 level in chambers and arteries

- VSD, ASD, PDA causes pulmonary hypertension (Left to Right shunt → increasing pulmonary blood flow)
  - 1. Signs of pulmonary hypertension

- VSD, ASD → causes LOCP (signs & symptoms of LOCP)
- PDA → hyperdynamic circulation (similar to peripheral signs of AR)

- VSD, ASD, PDA: accentuated P2 component
  - Splitting of S2:
    - VSD → Wide splitting
    - ASD → Wide fixed splitting
    - PDA → Paradoxical splitting

- S3:
  - VSD → due to LV volume overload
  - ASD → due to RV volume overload

- S4:
  - VSD, ASD, PDA → if caused pulmonary hypertension

VSD

<table>
<thead>
<tr>
<th>Inspection</th>
<th>Palpation</th>
<th>Auscultation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gian (a) wave</td>
<td>Palpable P2</td>
<td>Palpable P2</td>
</tr>
<tr>
<td>(a) wave equal to (v) wave</td>
<td>Right ventricular heave</td>
<td>Right ventricular heave</td>
</tr>
<tr>
<td>If developed pulmonary hypertension</td>
<td>Relative stenosis causes no thrill</td>
<td>RV pressure overload</td>
</tr>
<tr>
<td></td>
<td>Apex displaced outward</td>
<td>Apex displaced outward</td>
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<tr>
<td></td>
<td>(with localized apex)</td>
<td>only (with diffuse apex)</td>
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</tbody>
</table>

ASD

- S2 accentuated with wide splitting
- S3
- S4

- Pansystolic murmur with thrill
- Murmurs of relative MS (due to increase blood flow through mitral valve)
- Murmurs of AR in high defects
- Murmurs of relative PS (due to RV volume overload)
- in advanced: TR (RVE → dilated ventricle → affect valve) Systolic Ejection click in pulmonary area

PDA

- S2 accentuated with wide fixed splitting
- S3
- S4
- ASD itself doesn't cause murmur:
  - Murmurs of relative PS (due to RV volume overload)
  - Murmurs of relative TS (if the shunt is large)
  - in advanced: TR (RVE → dilated ventricle → affect valve) Systolic Ejection click in pulmonary area

- S2 accentuated with paradoxical splitting
- S4
- Continuous machinery murmur
  - N.B with moderate degree of pulmonary hypertension, the diastolic component of murmur disappears leaving a systolic murmur only.
  - Mid-diastolic murmur: may be audible at the apex as a result of the increased volume of blood flow across the mitral valve.

Systolic Ejection click in pulmonary area

Systolic Ejection click in pulmonary area

Systolic Ejection click in pulmonary area