EKG of pericardial tamponade
Hemodynamic Consequences of Pericardial Effusion

- Depends on:
  - Volume of effusion
  - Acuity of accumulation
  - Pericardial compliance
  - Myocardial performance and compliance
Rise in Intrapericardial Pressure As a Result Of Pericardial Effusion

- Rapidly Developing Pericardial Effusion
- Slowly Developing Pericardial Effusion

Increasing Intrapericardial Pressure (mm Hg)

Increasing Pericardial Fluid Volume (ml)
Hemodynamic Consequences of Cardiac Tamponade

- Pandiastolic resistance to RV filling
- Elevated RAP with prominent “X” descent and blunted “y” descent
  - Continuous compression, so flow ONLY in systole => no y descent (unlike constriction)
- Equalization of diastolic pressures
Hemodynamic Consequences of Cardiac Tamponade

- Intracardiac volumes decreased
- IVC, RA, RV compressed on ECHO
- SV and CO are low, SVR and HR are elevated
- Inspiratory increase in venous return leads to pulsus paradoxus
Cardiac Tamponade

- Pulsus paradoxus absent if ASD, localized compression, severe pulmonary hypertension, atrial fibrillation, severe systemic hypotension

- Ventricular interdependence => pulsus paradoxus and LVSP vs. RVSP discordance
Cardiac Tamponade

- Inspiration => fall in transmitral flows (Doppler), but fall in RA pressure (so no Kussmaul’s sign)

- Effusive-constrictive physiology: elevated RA pressure (with prominent y descent and dip & plateau) after intrapericardial pressure returns to zero
Paradoxus in Tamponade

Necessary Conditions

- Pericardial resistance affects both ventricles
- Intact atrial and ventricular septae
Paradoxus in Tamponade

**Mechanisms**
- Interventricular competition
- Augments right heart filling, lowers LV filling
- Inspiratory effects on pulmonary vascular pooling
Other Causes of Paradoxis

- Obstructive airway disease
  - Acute and chronic
- Constriction
- Restriction
- Pulmonary embolism
- RV infarction
- Circulatory failure
Echo Findings in Tamponade

- RA Diastolic Collapse
- RV Early Diastolic Collapse
- LA Collapse
- Abnormal inspiratory TV flow increase and MV flow decrease
- Abnormal inspiratory increase in RV size and decrease in LV size
- Swinging heart
- IVC plethora
<table>
<thead>
<tr>
<th>Hemodynamic Structure</th>
<th>Lower Limit</th>
<th>Upper Limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrium (a/v/m)</td>
<td>2-10</td>
<td>2-10/0-8</td>
</tr>
<tr>
<td>Right ventricle (s/ed)</td>
<td>15-30</td>
<td>0-8</td>
</tr>
<tr>
<td>Pulmonary artery (s/d/m)</td>
<td>15-30</td>
<td>4-12/9-16</td>
</tr>
<tr>
<td>PCW (a/v/m)</td>
<td>3-15</td>
<td>3-15/1-10</td>
</tr>
<tr>
<td>Left ventricle (s/ed)</td>
<td>100-140</td>
<td>3-12</td>
</tr>
<tr>
<td>Aortic (s/d/m)</td>
<td>110-140</td>
<td>60-90/70-105</td>
</tr>
</tbody>
</table>
Assessment of the Patient

- Insignificant effusion
  - Flat neck veins
  - Normal BP, HR, RR, good perfusion

- Hemodynamically significant-Compensated
  - Elevated JVP
  - Mild paradox, No hypotension or tachycardia
  - Good perfusion
  - Mild RV collapse
Assessment of the Patient

- Hemodynamically Severe-Max Compensation
  - Elevated JVP
  - Prominent paradox, Tachycardia
  - No hypotension-adequate perfusion
  - Chamber collapse on ECHO

- Hemodynamically Severe-Decompensated
  - Elevated JVP
  - Tachycardia, tachypnea
  - Hypotension with paradox
  - Chamber collapse, swinging heart
Constriction

- Heart encased in thick, inelastic shell
- When not fully filled (systole and early diastole) no resistance
- Resistance to filling throughout greater part of diastole
- Heart incapable of further distension with inspiration