Cardiac Tamponade

Schiller and Foster. “Echocardiographic evaluation of the pericardium.” *UptoDate* 2004, v 11.3

### Key Points:
- **Think about tamponade in a patient who presents with hypotension, tachycardia, and elevated neck veins**
- **Know how to measure a pulsus paradoxus and understand the pathophysiology**
- **Do not delay when tamponade is suspected!**

### Definition:
Hypotension that immediately reverses with pericardial drainage

### Physiology:
- Pericardial effusion of significant volume OR rapidly accumulated leads to increased pressure in pericardial space throughout the cardiac cycle
- During inspiration, as RV volume increases, the RV is unable to expand into the maximally stretched pericardium. Therefore, the interventricular septum bulges to the left, decreasing LVEDV and thereby decreasing cardiac output, causing a decrease in SBP during inspiration
- Diastolic equalization of pressures
- Acute vs. subacute tamponade: small volumes at fast rates vs. ability for pericardial stretch if fluid gradually accumulates
- *In both acute and subacute, there is a point at which intrapericardial pressure reaches an almost vertical ascent with a small amount of fluid -> acute decompensation*

### Etiologies
- Acute tamponade: Usually due to traumatic rupture of ventricle as a result of a procedure or blunt trauma; also in aortic dissection or myocardial infarction with ventricular rupture
- Subacute tamponade:
  - Infection: More commonly purulent than viral
  - Malignancy: Particularly lung, breast, Hodgkin’s, mesothelioma
  - Post-MI, post-CT surgery, post-procedure
  - Uremia
  - Post-XRT
  - Drugs
  - Collagen-vascular disease: in particular SLE
  - Idiopathic
  - HIV
- External to pericardial sac (pleural effusions have caused tamponade physiology)

### Clinical Presentation:
- Symptoms: chest pain, dyspnea, near-syncope
o Physical exam: will frequently demonstrate tachycardia, hypotension with narrow pulse pressure (but be aware that BP may remain normal/elevated until cardiovascular collapse is imminent)
  ▪ Elevated neck veins – absent y descent (absent or late diastolic filling of ventricle)
  ▪ Decreased heart sounds
  ▪ Pulsus paradoxus (decrease in SBP of >10 mm with inspiration; mechanism above)
    • Get a manual BP cuff and inflate until above SBP; then very slowly release until you start to hear Korotkoff sounds. At first, you should just hear them during expiration; slowly release until you hear a sound with every beat in the cardiac cycle. The difference between when you first hear the sounds and when you hear them with every cycle is the pulsus paradoxus.
  • May also be seen on A-line tracing
  • DDx: Any cause of severe respiratory distress, hypovolemic shock
  • Absent in tamponade if: rapid HR, irregular HR, profound hypotension, severe AI, or pre-existing cardiac disease like LV diastolic dysfunction
    ▪ Pericardial rub often absent
  o EKG: May show low volts and/or electrical alternans (specific)

• Diagnosis:
  o Tamponade is a clinical diagnosis
  o Echocardiogram
    ▪ Effusion: size doesn’t always matter (correlates with risk of tamponade, but not always and size is not precisely quantifiable)
    ▪ IVC plethoric and fails to collapse with inspiration – high negative predictive value (will r/o tamponade)
    ▪ RV expiratory collapse in early diastole – less sensitive, more specific
    ▪ RA expiratory collapse in late diastole – specific if for >30% of cycle
    ▪ LA collapse – 25% sensitive, but highly specific
    ▪ Small chamber sizes
    ▪ Reciprocal size changes with respiration between right and left ventricles and their valves
    ▪ Findings often absent in patients with pulmonary hypertension or RVH
  o PA catheter: equalization of diastolic pressures

• Treatment:
  o IV fluids to temporize (sometimes brings out tamponade physiology and physical signs)
  o Pericardiocentesis: paraxiphoid (left), needle at 15 degree angle to skin, toward left shoulder, with patient sitting forward
  o Pericardial drain or window