Chest Pain in the Emergency Department
“The Big Five”
Five life-threatening causes of Chest Pain

- Acute coronary syndrome
- Aortic dissection
- Pulmonary Embolism
- Tension Pneumothorax
- Esophageal Rupture
The Immediate Goals

1. ABC’s/Stabilization/Resuscitation
   • IV, O2, monitor, pulse oximeter
2. ECG
3. R/O the “The Big Five”
Acute coronary syndromes

**Etiology**
- *Ischemia*: imbalance between oxygen demand and oxygen supply
- *Fixed atherosclerotic lesion vs. plaque disruption with platelet/thrombi aggregation vs. spasm*
Chest Pain

- How do we decide whose chest pain could be an ACS?
  - History
  - Physical
  - ECG
  - Cardiac Enzymes
Clinical History of ACS

- **Classic**
  - Substernal chest pressure/tightness with radiation and associated symptoms

- **Questions:**
  - What % of acute MI’s are silent?
  - Are cardiac risk factors useful in the E.D.?
Clinical History of ACS

**Answers:**

- 30% of acute MI’s are clinically “silent”
- Cardiac risk factors are very poor predictors of risk for ACS in the E.D.
  - Predictive of the presence of CAD in a population of asymptomatic patients
  - The presence of chest pain outweighs the predictive value of any of these
Physical Exam in ACS

- Not helpful in distinguishing patients with ACS from those with noncardiac chest pain unless an alternative diagnosis is clear
  - e.g. pneumothorax
- Normal cardiopulmonary exam is most common
- $S_3$ in 15-20% with AMI
- Chest wall tenderness to palpation in ~15% with ACS
The ECG
The Diagnostic Key?

• The standard ECG is the single best test to identify patients with an AMI upon E.D. presentation
• But sensitivity is still far from ideal
  • ST elevation in 50% of AMI’s
  • 1-5% of AMI’s have a normal initial ECG
  • 4 - 23% of pts. with unstable angina have a normal ECG

Question:
• Within how many minutes of arrival in the E.D. with chest pain should ECG be done?
The ECG

**Answer:**

- The national guidelines require that the ECG be obtained within **10 minutes** of arrival in the E.D. for patients with a possible ACS.
Cocaine-related chest pain

Epidemiology:
- 6% of pts. with cocaine-associated chest pain have an AMI
- 20-60% have transient myocardial ischemia
- Often atypical chest pain
- Can be delayed for hours to days after the most recent use
Cocaine-related chest pain

**Question:**
What are the effects of cocaine on the coronary vasculature?

- A. SPASM
- B. INCREASED MYOCARDIAL OXYGEN DEMAND
- C. CLOT FORMATION
Cocaine-related chest pain

**Answer:**

- **Acute:**
  - **SPASM:** coronary artery vasoconstriction
  - **INCREASED MYOCARDIAL OXYGEN DEMAND:** Hypertension/tachycardia
  - **CLOT FORMATION:** Platelet aggregation and *in situ* thrombus formation

- **Chronic:**
  - Accelerated atherosclerosis and LVH
Cocaine-related chest pain

**Diagnosis:**
- ECG less sensitive and specific for MI
- CK-MB less sensitive
- Troponin I may be more useful

**Prognosis:**
- Favorable short-term prognosis
- 1 year mortality primarily due to comorbidities and/or continued cocaine use
Acute Coronary Syndromes: Treatment

Question:

- Which Meds decrease mortality from AMI?
Acute Coronary Syndromes: Treatment

- **Answer:**
  - Aspirin reduces mortality to the same degree as streptokinase: 23%
Acute Coronary Syndromes: Treatment

- **Chest pain, R/O MI:**
  - Aspirin (160-325 mg p.o.)
    - Inhibits thromboxane A2...decreased platelet aggregation
  - Nitrates *prn*
    - Decreased preload; decreased afterload; increased coronary perfusion in obstructed vessels
    - Especially in AMI with Hypertension or CHF
    - Not in Right Ventricular Infarction (cautious in IWMIs)
  - Clopidogrel
Acute Coronary Syndromes: Treatment

- **Unstable Angina:**
  - Above plus Heparin
    - Binds AT III, inactivates thrombin
    - Reduces mortality in unstable angina (with ECG changes)
  - or LMWH (enoxaparin 1 mg/kg SQ)
  - GP. IIb - IIIa

- **AMI:**
  - Above plus:
    - Beta blocker
      - Antiarrhythmic, anti-ischemic, anti-hypertensive
      - Decreased infarct size, cardiovascular complications, mortality
  - Consider PTCA, Fibrinolytics, GIIb/IIIa inhibitors
Treatment of Cocaine-associated chest pain

• Aspirin, nitrates, + heparin as for non-cocaine users
• BENZODIAZEPINES
  • Treats hypertension, tachycardia, anxiety)
• Avoid Beta blockers
• +/- Calcium channel blockers
Aortic Dissection

- Definition:
  - Intimal tear with entry of blood into the media
  - “dissects” between the intima and adventitia
- #1 site: ascending aorta at the ligamentum arteriosum
- Stanford Classification:
  - A: involves Ascending aorta (w/ or w/o descending)
    - 80% of dissections
  - B: descending aorta only
- DeBakey Classification:
  - Type I: Ascending and descending
  - Type II: Ascending
  - Type III:
    - a. ↓ Thoracic aorta
    - b. ↓ Thoracic and Abd. Aorta
Aortic Dissection

Figure 51.1. Classification of aortic dissections. (A) Dissection of ascending aorta. (B) Dissection of descending aorta.
Aortic Dissection

- Increased risk:
  - Group A: >50 yoa with hypertension
  - Group B: younger pts. with Marfan’s, Ehler-Danlos, pregnancy

- Mortality
  - Type A:
    - Untreated: 75%
    - Surgically treated: 15-20%
  - Type B:
    - 32-36% with or without surgery
Aortic Dissection:
Clinical Presentation

• History:
  • >90% with **abrupt and severe pain** in the chest or between the scapulae
    • “tearing” or “ripping”
    • Can be dull or pressure-like
    • Anterior chest ~ ascending aorta; Back ~ descending
  • Nausea, vomiting, diaphoresis common
Aortic Dissection: Clinical Presentation

Question: Which of the following presentations can be seen with aortic dissection?

A. Stroke
B. Paraplegia
C. Abdominal pain
D. Aortic insufficiency
E. Pericardial tamponade
F. Hoarseness
G. Wheezing
H. Dysphagia
Aortic Dissection: Clinical Presentation

- **Associated symptoms based on progression of dissection:**
  - *Carotid arteries*: stroke
  - *Spinal arteries*: paraplegia
  - *Abdominal aorta/renal arteries/iliacs*: Abdominal/flank pain
  - *Coronary arteries*: aortic insufficiency; pericardial effusion/tamponade
  - *Laryngeal nerve compression*: hoarseness
  - *Tracheal compression*: dyspnea/stridor/wheezing
  - *Esophageal compression*: dysphagia
Aortic Dissection: Clinical Presentation

- **Physical Exam:**
  - Symptoms/signs as above
  - Most commonly: normal heart and lungs
    - Aortic insufficiency murmur in 16-20%
  - Unequal, decreased, or absent peripheral pulses only found in 50%
Aortic Dissection:
Diagnosis

- **CXR**
  - 85% with some abnormality
    - widened mediastinum most common
    - left pleural effusion; indistinct aortic knob; displaced, calcified intima > 6mm from outer aortic wall

- **CT vs. TEE vs. aortogram**
Aortic Dissection: CXR
Aortic Dissection: CT Scan

Ascending Aorta

Descending Aorta
Aortic Dissection: Arteriogram
Aortic Dissection: Treatment

- Considering it?
  - 2 large bore IV’s, monitor, T&C, ECG

- Blood pressure:
  - Decrease the shear force on the intima to minimize progression
    - Lower arterial blood pressure
    - Decrease LV contractility

- Question:
  - Why not nipride alone?
Aortic Dissection: Treatment

- **Answer:**
  - Vasodilation causes reflex tachycardia, increasing the shear force on the dissection

- Goal: SBP 100-110 mm Hg; HR 60-80

- Options:
  - A. Nipride + esmolol
  - B. Labetalol

- Early CT surgery involvement
Pulmonary Embolus

- 650,000 cases/year in the U.S.
- Mortality
  - 2-10% if diagnosed and treated
  - 30% if undiagnosed
  - #3 cause of death overall
  - #1 cause of nonsurgical maternal death in the peripartum period
- The source is lower extremity DVT in 80-90% of cases
  - Upper extremity DVT in 10-15%
  - Others: pelvic vein thrombosis; fat emboli; septic emboli; right heart thrombosis
Question:
A. What is Virchow’s triad?
B. Does it have any relevance?
Pulmonary Embolus: Risk factors

- *Virchow’s triad:*
  1. **Venous stasis**
     - Prolonged travel; bed rest; etc.
  2. **Hypercoagulability**
     - Pregnancy; malignancy; estrogen therapy; deficiencies of protein C, protein S, AT III
  3. **Endothelial damage**
     - Recent surgery, trauma

- #1 risk factor = prior DVT/PE
- 10-15% of patients will have no identifiable risk factor at the time of presentation
Pulmonary Embolus: Clinical Presentation

- “Classic Triad”:
  - Dyspnea, hemoptysis, pleuritic CP in only 20% of pts.

- Three notable findings:
  - Pleuritic chest pain in 74%
  - Dyspnea in 84%
  - Respiratory rate > 16 in 92%

- The presence of any one of these should make you consider PE
  - The absence of all three argues strongly against PE
    - But then why would it be in your differential diagnosis?

- Heart rate > 100 in only 44%
Clinical Prediction

Well’s score

Clinical criteria for predicting likelihood of VTE

Add points \(\rightarrow\) score \(\rightarrow\) probability of PE or DVT

Positive Predictive Value:

<table>
<thead>
<tr>
<th>Score Level</th>
<th>PE Probability</th>
<th>DVT Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low (&lt; 2)</td>
<td>2.5%</td>
<td>3%</td>
</tr>
<tr>
<td>Moderate 2-6</td>
<td>28%</td>
<td>16%</td>
</tr>
<tr>
<td>High more than 6</td>
<td>55%</td>
<td>80%</td>
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</table>
## Clinical Prediction

### Well’s score

<table>
<thead>
<tr>
<th>Clinical History &amp; Since</th>
<th>Criteria</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sign’s of DVT</td>
<td>Leg swelling, objectively measured and pain with palpation deep vain region</td>
<td>3</td>
</tr>
<tr>
<td>Pulse more than 100</td>
<td></td>
<td>1.5</td>
</tr>
<tr>
<td>Immobilization</td>
<td>Bed rest, more than 3 days or surgery in previous 4 weeks</td>
<td>1.5</td>
</tr>
<tr>
<td>Previous DVT or PE</td>
<td>Must have been objectively diagnosed</td>
<td>1.5</td>
</tr>
<tr>
<td>haemoptysis</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Malignancy</td>
<td>Receiving active treatment for Ca or have received treatment for Ca in previous 6 months or receiving palliative treatment for Ca</td>
<td>1</td>
</tr>
<tr>
<td>PE as likely or more likely than alternative diagnosis</td>
<td>No specific criteria</td>
<td>3</td>
</tr>
</tbody>
</table>
Pulmonary Embolus: Diagnosis

- Clinical suspicion
- ECG
  - often normal
  - >40% with nonspecific ST and Tw abn.
  - Sinus tachycardia is the most common rhythm disturbance
- Question:
  - What is the “classic” ECG finding of PE?
  - What % of pts. with PE have this finding?
Pulmonary Embolus: Diagnosis

**Answer:**
- S1Q3T3 is seen in only 6% of pts. with PE
Pulmonary Embolus: The CXR

- Normal in ~30%
  - and a concerning finding in the setting of dyspnea and hypoxemia w/o RAD
- Atelectasis in ~50%
- Elevated hemidiaphragm in 40%
- Greatest roles of the CXR in PE are:
  - to r/o other causes of patients symptoms (pneumothorax/pneumonia)
  - For the subsequent interpretation of the V/Q scan
Pulmonary Embolus: The CXR

**Question:**
- What are:
  - A) Hampton’s hump
  - B) the Westermark sign?
- Are they clinically relevant?
Pulmonary Embolus: The CXR

- **Answer:**
  - Hampton’s Hump:
    - Pleural based wedge shaped infiltrate
  - Westermark sign:
    - Proximally dilated pulmonary artery with abrupt cut-off
- These are rare findings
  - Minimal clinical application
Pulmonary Embolus:
The V/Q Scan

- Overall:
  - 98% sensitive; 10% specific in diagnosing PE
  - Results must be combined with pretest probability
    - D,Dimer negative test r/o P.E

- Positive test does not diagnose it
Pulmonary Embolus:
Further tests/options

- LE Doppler US
- Pulmonary angiography
- Spiral CT the most sensitive test
- MRI
Pulmonary Embolus: Treatment

- Considering it:
  - IV, O2 prn, monitor, pulse ox.

- High pretest probability:
  - Anticoagulate 1st, then order your study
  - Heparin 80 U/kg i.v. bolus; 18 U/kg/hr i.v. drip

- Low (+/- intermediate) pretest probability:
  - Study 1st, then anticoagulate if appropriate
Spontaneous pneumothorax

- Especially tall, thin male smokers
- Only 10 – 20% occur with exertion
- Most thought to result from rupture of a subpleural bleb
- Symptoms vary with size and rate of progression of pneumothorax
Spontaneous pneumothorax: Clinical presentation

- Acute pleuritic CP in 95%
- Dyspnea in 80%
- Decreased breath sounds over the affected lung in 85%
- Tachypnea > 24 in only 5%
- Hyperresonance in <1/3
Question:

What should you see on the CXR of a patient with signs and symptoms of a tension pneumothorax?
Spontaneous pneumothorax: Diagnosis and Treatment

- **Answer:**
  - A reinflated lung (S/P needle decompression)

- **Tension pneumothorax:**
  - Clinical presentation of pneumothorax with hemodynamic compromise
  - Treatment is immediate needle decompression

- “Non-tension” spontaneous pneumothorax
  - Upright PA CXR is 83% sensitive
Pneumothorax: CXR
Spontaneous pneumothorax: Treatment options

- Tube thoracostomy
  - Minicatheter or standard chest tube
- Catheter aspiration
  - Single or sequential
- Observation x 6 hrs. with repeat CXR:
  - Stable; minimal/no symptoms; <25% ptx
  - No significant comorbidities
Chest Pain: Summary

- Remember the “Big Five” life threatening causes of chest pain
- ABCs; IV, O$_2$, monitor, pOx
- The diagnosis of ACS/PE/dissection may be subtle
- ACS:
  - ECG early; aspirin is a life saver
  - Cocaine is bad; avoid beta blockers
- Aortic dissection:
  - Lower the BP with labetalol or Nitroprosside+esmolol
Chest Pain: Summary

- **PE:**
  - V/Q scan is far from perfect; if high risk – heparinize before the test

- **Pneumothorax:**
  - Don’t diagnose a tension pneumothorax by CXR

- In all cases, do what’s safest for the patient