

Evaluation of Patients with Chest Pain

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Acknowledgement

- **Mark L. DeBard MD, FACEP**
 - ✓ **Professor of Emergency Medicine**
 - ✓ **Attending Emergency Physician, OSU University Hospital East**
 - ✓ **Co-lecturer with Dr. Moseley for many years on the topic of chest pain evaluation in Emergency Medicine for medical students, residents, and faculty physicians.**

Overview

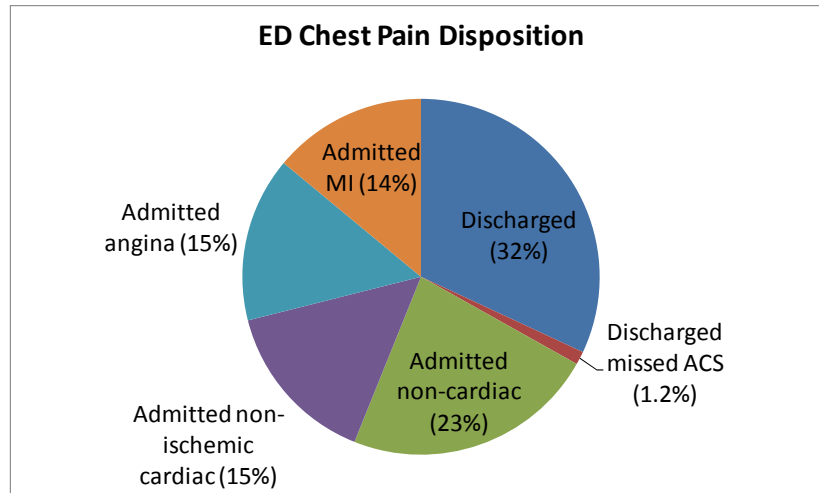
- **Chest pain risk stratification using the H and P, ECG, labs, and imaging**
- **Disease Specific Evaluations:**
 - ✓ **Acute Coronary Syndrome**
 - ✓ **Pulmonary Embolism**
 - ✓ **Aortic Dissection**
 - ✓ **Pneumothorax**
 - ✓ **Esophageal Rupture**
 - ✓ **Valvular Heart Disease**
 - ✓ **Pericardial/Myocardial Disease**

The Challenge of Chest Pain

- **Whether in the office, ED, or inpatient setting, the patient with chest pain presents a diagnostic dilemma**
- **You can not treat all patients with chest pain the same due to the broad differential diagnosis that ranges from benign causes to immediately life threatening events**
- **Must have a strategy that will help identify higher risk patients and differentiate them from lower risk patients**

ED Chest Pain Disposition (%)

- 6 million ED Chest Pain Patients (6% of all ED patients)



Differential Diagnosis

- Life Threatening
 - ✓ ACS
 - ✓ Pulmonary embolism
 - ✓ Aortic dissection
 - ✓ Tension pneumothorax
 - ✓ Esophageal rupture
 - ✓ Pericardial tamponade
- Non-Life Threatening
 - ✓ Muscular
 - ✓ Skeletal
 - ✓ Pneumonia
 - ✓ Simple pneumothorax
 - ✓ Pericarditis
 - ✓ GERD
 - ✓ Valvular heart disease
 - ✓ Zoster
 - ✓ Radiculopathy

Chest Pain Evaluation

- Things in common that can be useful for the evaluation of any patient with chest pain regardless of the setting:
 - ✓ History
 - ✓ Physical Exam
 - ✓ ECG
 - ✓ Lab Testing
 - ✓ Imaging

History

- Remains the cornerstone of diagnosis
- The importance of obtaining a thorough chest pain history cannot be over-emphasized

History

- **Remains the cornerstone of diagnosis**
- **Specific characteristics of pain can help with better defining the differential diagnosis:**
 - ✓ **Chronicity**
 - ✓ **Onset**
 - ✓ **Duration**
 - ✓ **Intensity**
 - ✓ **Exacerbating factors**
 - ✓ **Remission/relieving factors**
 - ✓ **Associated symptoms**

History

- **It is helpful to key in on the patient's own description of the pain:**
 - ✓ **Pain vs. discomfort**
 - ✓ **Pressure vs. sharp**
 - ✓ **Intensity at onset**
 - ✓ **Where is it located**
 - ✓ **Is the pain positional**
 - ✓ **What was the patient doing at the time**
 - ✓ **Worse with exertion**
 - ✓ **Worse with deep breaths**

History

- **Important to ask about associated symptoms that accompany the pain:**
 - ✓ **Shortness of breath**
 - ✓ **Nausea and/or vomiting**
 - ✓ **Diaphoresis**
 - ✓ **Syncope**
 - ✓ **Dizziness or weakness**
 - ✓ **Pain in other locations that might be related**

History

- **Does the patient have a previous medical history that is relevant?**
 - ✓ **Known coronary artery disease**
 - **Previous re-vascularization?**
 - **Was there chest pain associated with that prior CAD event?**
 - **Is the current chest pain similar or different than previous chest pain?**
 - ✓ **Hypertension**
 - ✓ **Diabetes**
 - ✓ **CVA/TIA**
 - ✓ **Peripheral vascular disease**

History

- **Risk Factors:**
 - ✓ Framingham risk factors (population based)
 - ✓ Connective tissue diseases
 - ✓ High potency stimulant use
- **Realize that risk factors only convey a lifetime statistical risk for disease processes:**
 - ✓ Presentation with chest pain has already selected for a higher risk group
 - ✓ Don't ignore them totally

History

- **Avoiding Pitfalls:**
 - ✓ Don't discount risk solely based on age, gender, or lack of traditional risk factors
 - ✓ Remember that some groups of patients tend to have atypical presentations:
 - Women
 - Elderly
 - Diabetics
 - ✓ Focus the history on life threats first and then broaden to include less concerning causes

History

- **Pearls:**
 - ✓ Abrupt onset think TAD, PE, PTX
 - ✓ Pleuritic pain think pericarditis, PE, pneumonia
 - ✓ Don't rely on response to therapy like NTG or GI cocktails
 - ✓ Radiation to jaw, arms, or neck increases likelihood of ACS
 - ✓ Consider sudden onset of symptoms, associated syncope or near syncope, or CHF like symptoms associated with the chest pain to be high risk

Physical Exam

- Realize that most patients with chest pain will have a normal physical examination
- The physical exam should be used to make diagnosis more likely, not totally exclude a diagnosis:
 - The absence of a single physical exam sign or combination of signs lacks the sensitivity and specificity to exclude disease
- Don't fixate on reproducible chest wall pain!
 - 15% of patients having an active AMI report a tender chest wall

Physical Exam

- **Focus on vital signs, general appearance, and positive findings:**
 - Clinical signs of CHF (S3/S4, JVD, fluid overload)
 - Friction rub (pericarditis)
 - New onset murmur (aortic dissection, AMI)
 - Hamman's crunch (esophageal rupture)
 - Asymmetric pulses (aortic dissection)
 - Swollen extremities (PE, CHF)
 - Diaphoresis (non-specific, but concerning)

High Risk Features to the H and P

- Sudden onset of pain
- High risk associated symptoms:
 - ✓ SOB, diaphoresis, vomiting, syncope, etc.
- Exertional pain or persistent rest pain
- Abnormal vital signs
- Elderly patients and those with multiple co-morbid conditions

ECG

- You can classify the ECG based on ACC/AHA guidelines:
 - ✓ STEMI
 - ✓ NSTEMI/high risk unstable angina
 - ✓ Non-diagnostic
- The ECG is a slice in time only:
 - ✓ <50% of initial ECGs are diagnostic
 - ✓ Serial ECG's recommended, but timing is unclear
 - ✓ Progression with AMI
 - Hyper-acute T-waves
 - STE
 - T-wave inversion
 - Q waves

ECG

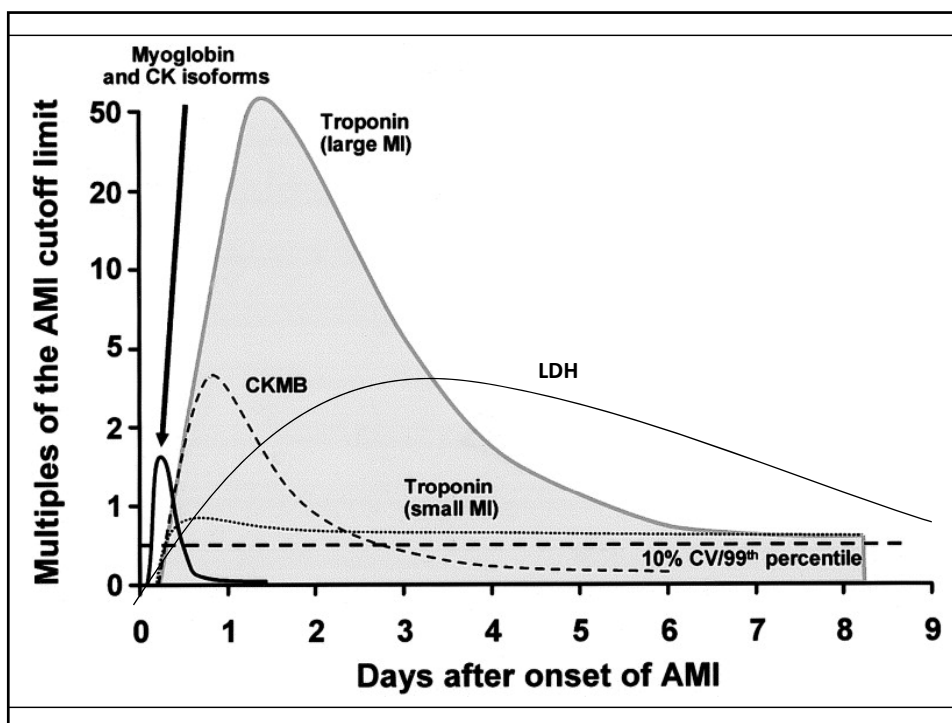
- If the ECG is abnormal, its important to compare it to an older ECG if available;
 - ✓ Can be critical for conditions like bundle branch blocks (old vs. new) in the setting of new onset chest pain as it helps determine intervention
- In addition to ACS:
 - ✓ ECG changes in PE
 - ✓ ECG in TAD
 - ✓ ECG in pericarditis
 - ✓ Arrhythmias

Lab Testing

- In patients sick enough to come to the ED or be admitted for chest pain, patients usually require:
 - ✓ Basic chemistries
 - ✓ Complete blood count
- Based on the history and physical examination, utilize a targeted approach to laboratory testing:
 - ✓ Coagulation profile (if on coumadin), cardiac markers, d-dimer, BNP, LFT's, Lipase
 - ✓ Tox screen if concern for high potency stimulant use

Lab Testing

- Cardiac Markers
 - ✓ Myoglobin
 - First to peak, non-specific
 - Good NPV
 - ✓ CK-MB
 - Former gold standard; supplanted by troponin
 - Elevation in muscle damage, renal failure, sepsis, CVA
 - ✓ Troponin
 - Prolonged elevation in serum (7-14 days)
 - More sensitive and specific for ACS than CKMB; detects micro infarcts
 - Elevated troponin in UA = 9x increase in death due to MACE



Lab Testing

- **BNP:**
 - ✓ Helpful in the assessment of acute shortness of breath of unclear etiology or for assessment of CHF
 - ✓ Know your lab assay
 - ✓ Level <100 = unlikely CHF
 - ✓ Level >500 = likely CHF
 - ✓ Level between 100 and 500 = uncertain
 - Chronic elevation
 - Chronic renal insufficiency
 - Severe COPD with right sided failure
 - PE
 - Elderly women

Lab Testing

- **D-dimer:**
 - ✓ Helpful for the assessment of PE
 - ✓ ELISA test preferred; know your lab assay
 - ✓ Excellent sensitivity, but poor specificity
 - ✓ Need to combine d-dimer testing with some assessment of pre-test probability
 - ✓ Low or moderate pre-test probability and negative ELISA d-dimer, means you have excluded to the limits of testing
 - ✓ If positive d-dimer, need to follow up with further definitive test like VQ scan or CTPA to exclude disease

Imaging

- Let the history and physical examination guide the decision about imaging
- Increasingly many options and difficult to apply the available literature to the patient sitting in front of you:
 - ✓ CT coronary angiogram with calcium scoring
 - ✓ Cardiac MRI with vasodilator stress
 - ✓ 80 lead ECG vest with regional pain mapping

Imaging

- For most patients that are being assessed for chest pain, the PA and Lateral Chest X-Ray remains quite useful:
 - ✓ Good screening test for many conditions
 - ✓ Assessment of vasculature, lungs, bones, etc.
 - ✓ Can provide alternative diagnoses in some cases
- Be cautious about sensitivity and specificity and using the CXR to “rule out” diagnoses
- Better to utilize to “rule in” based on findings of the examination

Acute Coronary Syndrome

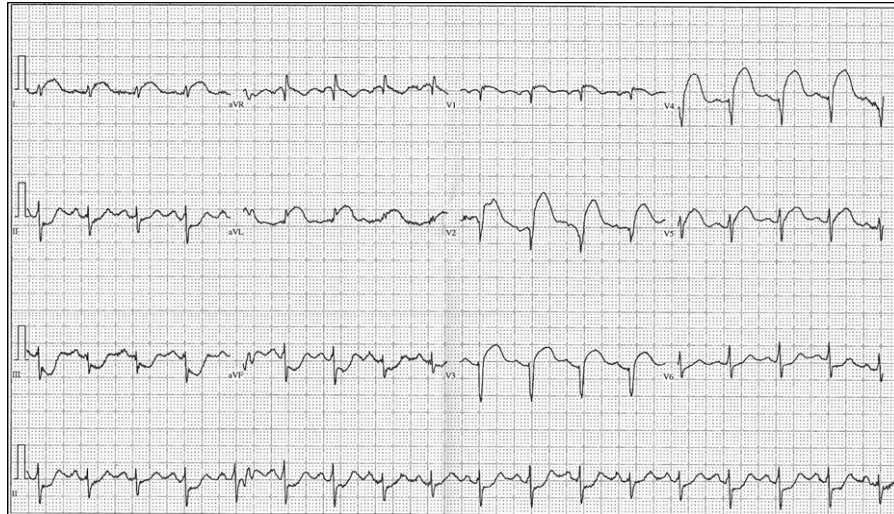
Acute Coronary Syndrome

- **ECG:**
 - ✓ You can classify the ECG based on ACC/AHA guidelines:
 - STEMI
 - NSTEMI/high risk unstable angina
 - Non-diagnostic
 - ✓ The ECG is a slice in time only:
 - Serial ECG's recommended, especially if the patient's symptoms change

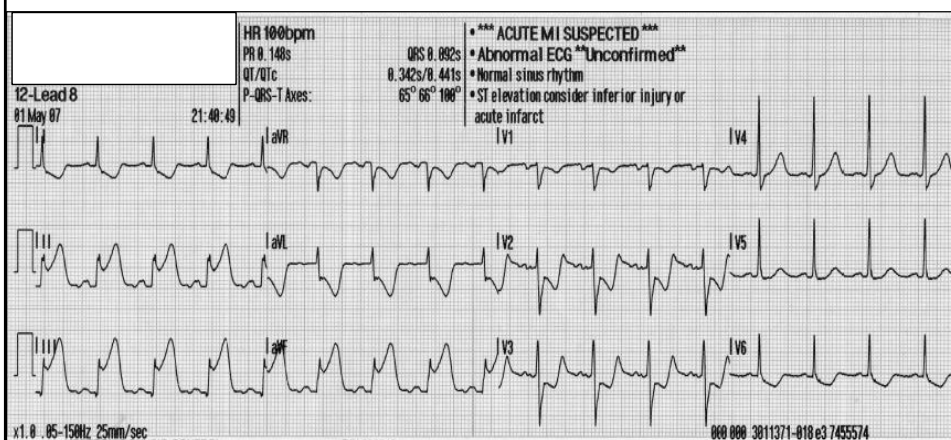
Acute Coronary Syndrome

- **ECG**
 - ✓ Normal ECG does not rule out ACS
 - A circumflex MI may be ECG-"silent"
 - ✓ Review AMI ECGs and anatomic lesions:
 - Anterior = LAD
 - Lateral = Circumflex, diagonal
 - Anterolateral = LAD or Left Main
 - Inferior = RCA (90%), Circumflex (10%)
 - Posterior = Usually RCA; associated with inferior, lateral AMI

LAD STEMI



Inferior STEMI



Acute Coronary Syndrome

- **LEVEL 1: ST ELEVATION MI (STEMI)**
 - ✓ ST-segment elevation or presumed new LBBB is characterized by ST-segment elevation 1 mm in 2 or more contiguous precordial leads or 2 or more adjacent limb leads.
- **LEVEL 2: HIGH RISK UA/NON-ST ELEVATION MI (NSTEMI)**
 - ✓ Ischemic ST-segment depression 0.5 mm or dynamic T-wave inversion with pain or discomfort. Transient ST-segment elevation 0.5 mm for 20 minutes is also included in this category.
- **LEVEL 3: INTERMEDIATE OR LOW RISK UA**
 - ✓ Normal or non-diagnostic changes in ST segment or T waves are inconclusive and require further risk stratification. This classification includes patients with normal ECGs and those with ST-segment deviation of 0.5 mm or T-wave inversion. Serial cardiac studies (and functional testing) are appropriate.

Clinician's Guide to ACS

- Imperative to follow some sort of evidence based strategy for acute coronary syndrome.
- Lots of literature to guide practice, but hard to keep up with.
- Algorithmic approach that standardizes care and minimized practice deviation.

The Ohio State University Medical Center Evidence-Based Practice Clinical Resources

Clinician's Guide to Acute Coronary Syndrome (ACS)

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Approved April 30, 2008. Reapproved April 28, 2010

Contents

- Initial evaluation and management of ACS
- Secondary prevention and long-term management of ACS
- Acute myocardial infarction (AMI) quality measures and related contraindications/documentation requirements

Quality Measures

- 2008 ACC/AHA STEMI/NSTEMI Measures:
- Aspirin at arrival*
 - Aspirin at discharge*
 - Beta-blocker at discharge*
 - Statin at discharge
 - ACEi or ARB for LVF*
 - Time to fibrinolytic therapy*
 - Fibrinolytic therapy received within 30 minutes of hospital arrival*
 - Time to PPCI*
 - Smoking cessation advice/counseling*
 - Evaluation of LVF*
 - Reperfusion therapy
 - Cardiac rehabilitation patient referral

*National Inpatient Hospital Quality Measures (publicly reported)

- Test Measures:
- LDL cholesterol assessment
 - Lipid-lowering therapy at discharge (CMS test measure)
 - Excessive initial heparin dose
 - Excessive initial enoxaparin dose
 - Excessive initial abiximab dose
 - Excessive initial tirofiban dose
 - Anticoagulant dosing protocol
 - Anticoagulant error tracking system
 - Clopidogrel prescribed at discharge for medically treated AMI patients

Additional CMS Measures:

- AMI 30-day mortality rate*
- AMI 30-day readmission rate*

Disclaimer: Clinical practice guidelines and algorithms of The Ohio State University Medical Center (OSUMC) are standards that are intended to provide general guidance to clinicians. Patient choice and clinician judgment remain central to the selection of diagnostic tests and therapy. OSUMC's guidelines and algorithms are reviewed periodically for consistency with new evidence; however, new developments may not be represented.

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Acute Coronary Syndrome

- **Cardiac Marker Summary:**
 - ✓ No marker is 100% sensitive all the time
 - ✓ Troponin is the gold standard for AMI diagnosis
 - ✓ Patients with positive markers have distinctly higher all cause morbidity and mortality in the literature
 - ✓ Cardiac marker analysis should not delay coronary intervention in high risk patients (STEMI)

Acute Coronary Syndrome

- **Non-Invasive Testing:**
 - ✓ Numerous studies show the prognostic value of stress testing to help risk stratify chest pain patients
 - Negative stress
 - 2% with MACE at 6 month
 - Positive or inconclusive stress
 - 17% with MACE
 - Combining treadmill or pharmacologic stress with nuclear imaging or echocardiography greatly increases sensitivity.
 - Modality less important than timing of testing
 - Testing is more sensitive if patient actually having symptoms at the time of testing

Acute Coronary Syndrome

- **CT Coronary Angiogram:**
 - ✓ In setting of acute chest pain, pt should be low risk
 - ✓ Non-inferior to stress nuclear imaging in low risk ED chest pain patients:
 - Stress nuclear imaging:
 - Sensitivity=71%, Specificity=90%, NPV=97%
 - Multidetector CT:
 - Sensitivity=86%, Specificity=92%, NPV=99%
 - ✓ What's the physiological significance of lesions it finds?
 - ✓ Radiation exposure and cost for low risk patients?

Acute Coronary Syndrome

- **Cardiac Catheterization:**
 - ✓ Considered the gold standard for ACS diagnosis
 - Anatomical and functional assessment
 - Can fix what you find (PCI)
 - Variability in lesion designation (visual assessment)
 - Some centers now using IV ultrasound to look at plaque stability
 - ✓ **Complications**
 - ATN from IV dye load
 - Bleeding (groin hematoma, RP bleed)
 - Pseudoaneurysm

Acute Coronary Syndrome

- **Treatment:**
 - ✓ **Protocol based diagnostic and treatment algorithm:**
 - IV, Oxygen, Cardiac Monitor (or call 911)
 - CXR, cardiac markers, other labs
 - ASA (if they have a chest, given them ASA)
 - Chewed, 162-325 mg unless taken
 - Clopidogrel if true ASA allergy

Acute Coronary Syndrome

- **Treatment**
 - ✓ **Nitroglycerin**
 - 0.4 mg SL q 5 min X 3
 - IV drip if needed for CP, HTN, CHF
 - Not if BP <90, HR <50, RV MI
 - ✓ **Beta Blocker**
 - Should be used, but timing?
 - ✓ **Morphine**
 - Should it be used?
 - ✓ **Others**
 - ACE, statin important, but not acutely

Acute Coronary Syndrome

- **Treatment**

- ✓ **Anticoagulation**

- Anticoagulate high risk ACS patients aggressively
 - Don't give high risk therapies to low risk groups
 - Know local practices, but remember that guidelines are increasingly national and expected
 - Positive troponin and ECG changes should get
 - ASA
 - Heparin/LMWH
 - Clopidogrel
 - Glycoprotein IIb/IIIa Inhibitor

Acute Coronary Syndrome

- **ACS Treatment Summary**

- ✓ **STEMI**

- ASA, NTG, B-blocker, UFH/LMWH, Clopidogrel, IIb/IIIa Inhibitor
 - Thrombolytics or PCI
 - Disposition = ICU

- ✓ **NSTEMI/High Risk UA**

- ASA, NTG, B-blocker, UFH/LMWH, Clopidogrel, IIb/IIIa Inhibitor
 - Disposition = ICU

- ✓ **Some Risk for UA**

- ASA, NTG, +/- on B-blocker
 - Disposition: Floor telemetry admission vs. short stay unit (depends on local resources)

Cocaine Associated Chest Pain

- Very common in the ED patient population
- Acute intoxication causes vasospasm, inflammatory mediator release, platelet aggregation
- ACS can result
- Treatment = routine ACS + lorazepam
 - ✓ Exception = concern over use of beta blockers – this may worsen vasoconstriction and hypertension
- Conservative management in most cases

Pulmonary Embolism

Pulmonary Embolism

- **Diagnostic:**
 - ✓ **Physical exam is unreliable**
 - Hohman's sign
 - ✓ **Symptoms from HPI helpful like dyspnea, rapid heart rate, palpitations, pleurisy, hemoptysis, calf pain, etc.**
 - ✓ **Literature strongly supports some type of structured pre-test probability assessment**
 - Well's Criteria
 - Charlotte Rule

Pulmonary Embolism

- **D-dimer:**
 - ✓ **Helpful for the assessment of PE**
 - ✓ **ELISA test preferred; know your lab assay**
 - ✓ **Excellent sensitivity, but poor specificity**
 - ✓ **Need to combine d-dimer testing with some assessment of pre-test probability**
 - ✓ **Low or moderate pre-test probability and negative ELISA d-dimer, means you have excluded to the limits of testing**
 - ✓ **If positive d-dimer, need to follow up with further definitive test like VQ scan or CTPA to exclude disease**

Pulmonary Embolism

- **Diagnostic:**
 - ✓ **DVT**
 - **Duplex Ultrasonography**
 - Non-invasive and first line
 - More sensitive proximally
 - Serial exams necessary?
 - Availability
 - **Venography**
 - Gold standard (does anyone do this?)
 - CTV often combined with CTA

Pulmonary Embolism

- **Diagnostic:**
 - ✓ **PE**
 - **VQ Scan** – have to do structured PTP assessment
 - PLOPED I
 - Low PTP and normal or very low prob = essentially rules out PE
 - High PTP and high prob = rule in PE
 - Anything else needs further testing
 - **CT scan** – high quality scanner and radiologist
 - PLOPED II
 - Study of choice; often gives alternative diagnosis
 - Literature unclear what to do with negative CTA when you have a high PTP

Pulmonary Embolism

- **Diagnostic:**
 - ✓ **PE**
 - **CT Scan**
 - Can increase sensitivity if combining CTA with CT venography
 - Helps define pelvic vein VTE, and can catch proximal femoral DVT
 - **Pulmonary Angiography**
 - Gold standard
 - Not available
 - Not trivial morbidity

Pulmonary Embolism

- **Treatment:**
 - ✓ **Anticoagulate with UFH or LMWH**
 - LMWH superior for treatment of DVT
 - Unclear about PE; safe to use, but many still start with UFH
 - ✓ **IVC filter to prevent further clot in those with contra-indications to anticoagulation**
 - ✓ **Thrombolytics or surgical embolectomy for massive PE and hemodynamic instability**
 - Immediate consultation with ICU and cardiothoracic surgery if available

Aortic Dissection

Aortic Dissection

- **Path:**
 - ✓ **Sheer stress leads to intimal tear**
 - ✓ **Risk factors:**
 - Hypertension (most common)
 - Trauma
 - Pregnancy
 - Coarctation
 - Bicuspid AV
 - High potency stimulants
 - Syphilis

Aortic Dissection

Stanford

- Type A
 - ✓ Any involvement of ascending aorta
- Type B
 - ✓ Limited to descending aorta

DeBakey

- Type I
 - ✓ Ascending and descending aorta
- Type II
 - ✓ Ascending only
- Type III
 - ✓ Descending only

Aortic Dissection

- Clinical:
 - ✓ Sudden onset of intense, ripping, tearing pain that often is in the chest radiating to the back or abdomen (suspect when pain above and below diaphragm)
 - ✓ Absent pulses, discrepancy in UE/LE BP, ischemic limb
 - ✓ New murmur of AR
 - ✓ Cardiac tamponade
 - ✓ Neuro symptoms like CVA, syncope, cauda equina syndrome
 - ✓ Mesenteric ischemia

Aortic Dissection

- **Diagnostic:**
 - ✓ **ECG**
 - Beware AMI mimic; beware anticoagulation
 - ✓ **CXR**
 - More helpful in trauma; normal CXR not sensitive enough to rule out the diagnosis
 - Review classic findings in trauma
 - ✓ **CT scan with IV contrast**
 - Study of choice in stable patient

Aortic Dissection

- **Diagnostic:**
 - ✓ **Conventional angiography**
 - Gold standard, but not readily available
 - Being supplanted by CTA
 - ✓ **Echocardiography**
 - TTE helpful to rule out complications but not great sensitivity
 - TEE is the test of choice for unstable patient; often done perioperatively
 - Dubious availability even with cardiology back up

Aortic Dissection

- **Treatment:**
 - ✓ Close HD monitoring; arterial line
 - ✓ Control BP (<120 SBP) and HR (<60)
 - Beta blockers are mainstay
 - Prevent sheer stress and further tear
 - Classic combination is esmolol and nitroprusside
 - ✓ Classic dictum of Stanford A managed surgically and Stanford B managed medically
 - Blurred with new stent grafts and PCI?

Pneumothorax

Pneumothorax

- Sudden onset of sharp unilateral chest pain
- Traumatic usually obvious by history and part of standard trauma evaluation
- Spontaneous 1:13,000; 6x more common in young males
- Treatment depends on size:
 - ✓ Observation
 - ✓ Small lumen catheters
 - ✓ Heimlich valves
 - ✓ Traditional tube thoracostomy for all larger PTX

Esophageal Rupture

Esophageal Rupture (Boerhaave's Syndrome)

- History is usually protracted vomiting, followed by severe chest pain and dysphagia:
 - ✓ Constant pain; usually much worse with swallowing
 - ✓ Dyspnea also common
 - ✓ If late presentation, patients are critically ill
 - ✓ Commonly described in alcoholics
 - ✓ 92% iatrogenic or trauma, 8% spontaneous
- Hammond's Crunch:
 - ✓ cellophane sound of mediastinum due to SQ emphysema
- Pneumomediastinum, Left pleural effusion
- Gastrografin swallow, CT, antibiotics, OR, ICU

Valvular Heart Disease

Mitral Valve Prolapse

- **Path**
 - ✓ Familial incidence
 - ✓ Increased risk of dysrhythmia, endocarditis, sudden death
- **Clinical**
 - ✓ Atypical chest pain and palpitations; advanced disease leads to MVR
- **Treatment**
 - ✓ Beta blockers help for atypical chest pain and palpitations

Aortic Stenosis

- **Path**
 - ✓ <65 = rheumatic HD and congenital bicuspid
 - ✓ >65 = calcifications
 - ✓ Obstruction of LV outflow track lead to CHF and sudden death
- **Clinical**
 - ✓ Classic triad of angina, dyspnea, and syncope
 - ✓ Harsh murmur with radiation to carotids
- **Treatment**
 - ✓ Extreme caution with preload/afterload reduction (get help with acutely decompensated patients)
 - ✓ Valvuloplasty stabilizing, replacement definitive

Pericarditis

Pericarditis

- **Path**
 - ✓ **Idiopathic (most common)**
 - ✓ **Infectious (viral, TB was classic cause)**
 - ✓ **Malignancy (lung and breast)**
 - ✓ **Drug induced (procainamide, hydralazine)**
 - ✓ **Post radiation**
 - ✓ **Post MI (Dressler's syndrome)**
 - ✓ **Uremia**
 - ✓ **Connective tissues (RA, lupus)**

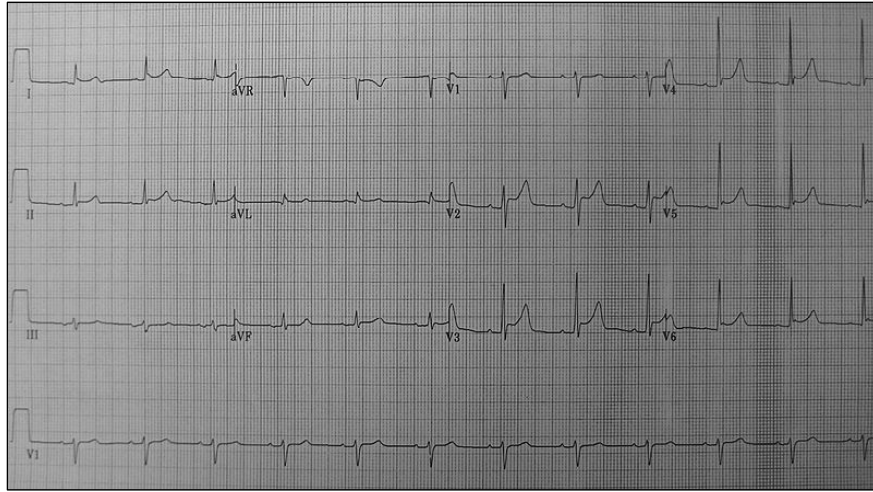
Pericarditis

- **Clinical**
 - ✓ Sharp, stabbing, pleuritic CP
 - ✓ Worse with supine, better when sitting up
 - ✓ Friction rub (classic) but usual transient and difficult to auscultate
 - ✓ Viral symptoms (low grade fever, malaise, URI symptoms)
 - ✓ Usually tachycardic

Pericarditis

- **ECG**
 - ✓ Electrical alternans with big effusions
 - ✓ First stage = ST elevation, PR depression
 - ✓ Second stage = ST isoelectric
 - ✓ Third stage = T wave inversion
 - ✓ Fourth stage = resolution
 - ✓ BEWARE OF MIMIC TO AMI!
- **CXR**
 - ✓ Limited value (alternative diagnosis)
 - ✓ With acute effusion can have normal silhouette

Pericarditis



<http://en.wikipedia.org/wiki/File:PericarditisECG.JPG>

Pericarditis

- **Diagnostic**
 - ✓ **Echocardiogram**
 - Everyone with pericarditis eventually needs this to rule out significant effusion
 - Tamponade is associated with right heart diastolic collapse
 - Remember that tamponade is a clinical diagnosis (muffled heart sounds, hypotension, JVD)
 - ✓ **Lab tests**
 - Somewhat dependent on suspicion of etiology
 - Cardiac markers? Can have small troponin leak
 - ESR and CRP are non-specific but usually elevated

Pericarditis

- **Treatment**
 - ✓ NSAIDs unless contra-indicated
 - ✓ Not steroids in most cases
 - ✓ Recent studies show colchicine helpful
 - ✓ Pericardiocentesis or pericardial window for tamponade
- **Disposition**
 - ✓ Depends
 - ✓ Classically outpatient management

Myocarditis

- **Path**
 - ✓ Inflammation of heart muscle
 - ✓ Broad range of etiologies
 - ✓ Most common etiology in U.S. is viral (enteroviruses and echoviruses)
 - ✓ Most common worldwide is parasitic
 - ✓ Often mixed pericarditis and myocarditis picture

Myocarditis

- **Clinical**
 - ✓ **Cardiac symptoms 7-14 days following viral illness (URI or GE)**
 - ✓ **CP reflects pericardial irritation**
 - ✓ **Fatigue, DOE**
 - ✓ **Fever, tachycardia out of proportion to fever**
 - ✓ **CHF and pulmonary edema in severe cases**

Myocarditis

- **Diagnostic**
 - ✓ **ECG**
 - **Sinus tach, dysrhythmias, acute LVH**
 - ✓ **CXR**
 - **cardiomegaly**
 - ✓ **Echo**
 - **Best single test; also evaluates for complications like pericardial effusion**
 - **Many centers now using cardiac MRI**
 - ✓ **Lab tests**
 - **Often elevated cardiac markers**
 - **ESR and CRP usually elevated, but non-specific**

Myocarditis

- **Treatment**

- ✓ **Primarily supportive and monitoring for complications**
- ✓ **Antimicrobials if appropriate for infectious etiology**
- ✓ **Steroids, IVIG, and antiviral agents are all controversial**
- ✓ **Treatment of heart failure if present**
- ✓ **LVAD and cardiac transplantation in severe cases**

Other Etiologies of Chest Pain

Hypertension

- Significant hypertension in conjunction with hypertensive heart disease can manifest as chest pain
- This is alleviated with reduction of blood pressure
- It is important to exclude co-existing coronary artery disease in these patients as presentation may be very similar
- Patients with HTN and diastolic dysfunction are prone to chest pain when blood pressure is uncontrolled

GERD / Esophagitis

- A not entirely uncommon manifestation of GERD is atypical chest pain
- Depending on the person, rather than classic "heartburn," "chest pain" may be the presenting symptom
- Something to consider once more worrisome chest pain causes are excluded and to clinically correlate with overall presentation
- Consideration for empiric therapy and EGD/GI evaluation

GERD / Esophagitis

- Things to remember:
 - ✓ Do NOT assume chest pain is from GERD in a patient with a history of GI complaints
 - ✓ Urgent / life-threatening causes MUST be excluded first
 - ✓ Do NOT assume pain relief from Maalox, GI cocktail, etc, correlates to a GI cause of the pain
 - This is similar to reproducible chest pain – it may still be from coronary ischemia / angina

Esophageal Spasm

- This is a somewhat controversial entity in regards to correlation with chest pain
- Occasional correlation between evaluation with manometry and chest pain
- Occasional relief with calcium channel blockers
- This is a difficult diagnosis to evaluate and should be done in conjunction with gastroenterology in the appropriately selected patient population

Costochondritis

- Inflammation of the cartilage at the rib/sternum junction
- More common in women than men
- “Tietze’s syndrome” if significant inflammation at the rib/sternum cartilaginous region with possible overlying erythema
- Diagnosis is clinical with tenderness to palpation seen
- It is important to know that angina can have reproducible chest wall pain and distinguishing these two conditions is imperative
- Treatment is supportive and with NSAIDs