Matters of the Heart: Angina, Acute Coronary Syndrome, Congestive Heart Failure, and Valvular Heart Disease

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Faculty Disclosure

- No associations with any pharmaceutical companies or other medically-related groups (though some of my 401k might invest in pharm co’s – I have no idea, as I don’t keep up with them).

Review/Overview of Major Cardiology Topics

- Review important info you’ve learned
- Provide you with relevant diagnostic & therapeutic info
- Drop a few pearls

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Practice Board Question

1. What is the most common cause of death among women in the US?
   A. Breast cancer
   B. Lung cancer
   C. Cardiovascular disease
   D. Gynecological cancers
   E. Influenza + pneumonia

Practice Board Question

1. What is the most common cause of death among men in the US?
   A. Breast cancer
   B. Lung cancer
   C. Cardiovascular disease
   D. Gynecological cancers
   E. Influenza + pneumonia

Practice Board Question

1. What is the most common cause of death among adults in the US?
   A. Breast cancer
   B. Lung cancer
   C. Cardiovascular disease
   D. Gynecological cancers
   E. Influenza + pneumonia
Case 1

- 58 y.o. Caucasian M presents with 6 mo H/O intermittent LUE pain associated with any activity, not just LUE movements. Further questioning reveals H/O non-painful tightening in chest during the same period – also seems associated w/activity.

- 67 y.o. M, Chest/Arm Pain:  PQRST
  - Provocative: rapid walking, playing w/grandkids, sex
  - Palliative: rest
  - Quality: aching, +/- squeezing
  - Radiation: now that he thinks about it, maybe the LUE pain comes after the vague chest tightness
  - Severity: mild
  - Temporal: 6 mo, intermittent, fairly quick onset w/activity, ? slowly progressive.

Case 1

- PMH: negative
- FH: negative
- SH: former smoker, 1 ppd X 20 years, quit 15 years ago

Practice Board Question

- Which of the following statements is true regarding this patient?
  A. A normal ECG effectively rules out cardiovascular disease
  B. He has likely had an acute myocardial infarction
  C. Costochondral tenderness dramatically reduces the likelihood of significant cardiovascular disease
  D. Response to sublingual nitroglycerin, in office or at home, is a specific test to differentiate cardiovascular from non-CV cause
  E. Treatment with a beta-blocker is appropriate
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Angina: A Manifestation of CAD

- Myocardial ischemia
- Supply ≠ demand (i.e., insufficient supply or excessive demand)
  - ↑ demand: ↑ HR, ↑ wall tension (preload, afterload)
  - ↓ supply: CAD, ↓ O₂ capacity (anemia—Hgb <8?)
- The most common clinical manifestation of cardiac ischemia
  - 6.3 million Americans
  - 350,000 new cases/yr

Angina

- Retrosternal chest discomfort:
  - Squeezing, pressure-like.
  - Not necessarily pain.
  - Also: heaviness, burning, choking.
- Brought on by exertion, emotional stress, cold temperature, eating.
- Relieved by rest, NTG (NOT a specific therapeutic challenge—esophageal spasm responds).
- Duration 1 – 5 minutes. If only lasts few seconds ⇒ unlikely to be angina (pearl).

Angina: Where’s the Pain?

- Pain in the:
  - Epigastrium
  - Back
  - Neck
  - Jaw
  - Shoulder(s)
- Radiates to:
  - Arms
  - Shoulders
  - Neck
**Angina Pearl**

Pain above the mandible OR below the epigastrium is unlikely to be angina.

**Angina in Women**

- Women are more likely than men to have atypical sx:
  - Pain: more intense, sharp, burning
  - Location: more often in neck, throat than men
  - Provocative factors: more likely associated with sleep, rest, mental stress
- Women underestimate their own CAD risk.
  - Physician assessment is affected by pt affect.
  - Women are managed less aggressively.

**Women & Angina**

- However, more women experience angina than men!
- Women w/CAD have a slightly higher mortality rate than men (studies equivocal).
  - More atypical sx.
  - They get CAD ~ 10 years later than men.
- Pearl: High index of suspicion for CAD in women with risk factors or sx.

**Grading Angina**

- **Canadian CV Society**
  1. Sx only w/strenuous activity
  2. Sl limitation—sx w/vigorous activity
  3. Mod limitation—sx w/everyday living activities
  4. Severe limitation—sx w/any activity or @ rest

- **NYHA**
  1. No limitation of activity—no sx w/ ordinary activity
  2. Sl limitation—sx w/ordinary activity
  3. Mod limitation—sx w/less-than-ordinary activity
  4. Severe limitation—sx w/any activity or @ rest
**Physical Exam in Angina**

- Exam generally WNL.
- Levine sign (clenched fist over chest) not sensitive or specific.
- Palm sign (open palm) more common, no more accurate.
- Signs of CV risk:
  - HTN
  - Xanthelasma
  - Reduced pulses

**ECG in Angina**

- Stable angina typically reveals transient ST depression during episode (compare to Prinzmetal’s).

**CAD Risk Factors**

- Per JNC-7:
  - Age—65 F (or premature menopause), 55 M
  - BP (HTN)
  - Cigarettes (smoking)
  - DM
  - Exercise lack (physical inactivity)
  - FH—65 F, 55 M
  - Good cholesterol too low (HDL-C <40)
  - High LDL-C
  - Obesity (BMI > 30)

**CAD Risk Factors—2**

- Per NCEP ATP-III:
  - Age: male 45, female 55 or premature menopause w/o E replacement therapy
  - BP—HTN (>140/90 mmHg, or on med
  - Current cigarette smoking
  - Family Hx premature coronary heart disease: definite MI or sudden death before 55 in male 1° relative, before 65 in female 1° relative
  - Good cholesterol low—HDL <40 mg/dL
  - LDL is implicit—algorithm tells you when to treat

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**HDL-C: The Good Cholesterol**

- In both JNC-7 & NCEP ATP III, **HDL-C >60** is a negative risk factor – removes 1 point.

**CAD Equivalents (NCEP ATP III)**

- DM
- Symptomatic carotid artery dz
- AAA
- Peripheral arterial dz
- 10-yr risk of 1st event >20%
- (probably CKD—not in ATP III yet)

**Gender**

- Note that gender is an implicit risk factor in both NCEP & JNC-7
  - Risk in men increases ~ 45/55
  - Risk in women increases ~55/65

**Newer CAD Risk Factors**

- **hsCRP > 3 – 4**
  - Higher CRP → higher risk at any LDL level.
  - May be most useful in those w/intermediate CAD risk to direct further eval & therapy for 1st prevention (per CDC & AHA).
  - My caveat FYI: most CRP studies done by Ridker, who holds patent on hsCRP assay.
- Homocysteine (though Rx does not ↓ risk).
- Coronary artery calcification (CAC) on electron beam CT

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Likely Risk Factors We Know About But Don’t Include

- CKD
- Probably microalbuminuria
- LVH
- H/O pre-eclampsia
- (BNP, nT-pro-BNP)

Primary Prevention: 5CDE

- Cessation of smoking
- Control BP
- Control cholesterol
- Control DM
- Control weight (obesity)
- Diet: ↑ fiber, vegetables, fruits, whole grains, Ω-3
- Exercise

ASA in Primary Prevention

- Much smaller benefit than in 2° prevention.
- Same risk of GI bleeding.
- Women: >65 → lower risk of ischemic stroke, probably lower risk of MI. No benefit to mortality, other endpoints, <65.
- USPSTF: all healthy M/W w/5-yr risk ≥3%.
- AHA: all healthy M/W w/10-yr risk ≥ 10%.
- You will not have Framingham calculators, so guesstimate.

Framingham Risk Guesstimation

- Men:
  - >70 yo → 10% risk.
  - The younger you are, the more a high cholesterol smoking adversely impacts you.
- Women:
  - >75, you have to also have Total Chol > 280 + HDL <40 to have 10% risk.

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Noninvasive Dx of CAD

• ACC/AHA:
  – Exercise is the preferred form of stress for pts who can exercise to 85% of MHR (0.85 X [220-age]).
  – Intermediate probability: exercise ECG (treadmill). Includes RBBB, <1mm ST ↓.
  – Use stress test w/imaging for:
    • ECG interpretive interference: LBBB, >1mm ST ↓ (incl LVH, dig), ventricular pacer, pre-excitation (WPW).
    • Inadequate exercise (<85% MHR).
    • Need localization or functional significance of lesion or myocardial viability—prior revascularization.

http://www.uptodate.com/online/content/topic.do?topicKey=chd/59850&selectedTitle=2~150&source=search_result

Noninvasive Dx of Angina: Clinical Dx

• Pearl: A clinical dx of angina has up to a 90% predictive accuracy for the presence of CAD.
  – Cheaper than other noninvasive tests.
  – Make your diagnosis carefully.

Noninvasive Dx of CAD—Tidbits

• Anti-ischemic drugs, including β-blockers, should be withheld for 4 – 5 half-lives before testing.
  – ~48 hrs.
  – Withdraw gradually.
• Lower predictive accuracy in women.
• Perfusion imaging modalities are similar:
  – SPECT MPI—more sensitive
  – Stress echo—more specific
  – Thallium MPI
• ETT specificity ~ thallium, SPECT

Treatment of Stable Angina/CAD

• Pharmacotherapy:
  – Aspirin
  – β-blockers
  – Ca++ channel blockers
  – Nitrates
• Non-pharmacologic therapy
  – Risk factor reduction
ASA

- ACCP: ASA indefinitely for all pts w/chronic stable angina, or clinical or lab evidence of CAD
- In pts w/occlusive CV event (nonfatal MI or non-hemorrhagic stroke), ASA reduces risk of subsequent MI, stroke, vascular death

ASA Dosing

- Largest trial used 75 – 325 mg
- Other trials have shown that low-dose (75 – 150 mg) is equivalent to medium-dose (160 – 325 mg)
- FDA: 75 – 325 mg
- ACC/AHA: 75 – 162 mg
- ACCP: 75 – 100 mg
- Acute events: wait a few minutes...

ASA in Diabetes

- Recommended for all diabetics w/evidence of cardiovascular dz (2° prevention)
- Recommended for 1° prevention in all diabetics w/at least 1 risk factor:
  - Age >40 y.o.
  - BP (HTN)
  - Cigarette smoker
  - FH CAD
  - "ABC ABC Famil-ee"

Type of ASA Formulation

- It doesn't matter.
  - Enteric coating may reduce gastric erosions, but has no effect on GI bleeding.
- Acute events are a different issue....
ASA Intolerance

• ~5% cannot tolerate ASA
• Clopidogrel (Plavix) is an acceptable alternative
  – May have lower GI bleeding incidence
  – Higher incidence of other side effects (rash, diarrhea)
  – High cost, high NNT (i.e., low absolute benefit)

Non-ASA NSAID’s

• Do not have beneficial effect on cardiovascular dz.
• May reduce efficacy of ASA in CV prevention.
  – FYI: if real, this effect may be negated by giving ASA ≥ 2 hrs before NSAID
• May, in and of themselves, have negative effects on CAD risk (COX-2, naproxen, ibuprofen, others)

Beta-Blockers in Angina/CAD

• Reduce HR
  – Reduces myocardial oxygen demand
  – Reduces ischemic threshold
• Reduce myocardial contractility
  – Reduce oxygen consumption
• In HTN β₁-blockade of juxtaglomerular apparatus in kidneys is important (decreases renin secretion)

Effects of β-Blockers in Stable Angina

• Improve exercise capacity
• Reduce exercise-induced ST-depression
• Reduce frequency of angina episodes
• Reduce sublingual NTG use
• Pearl/Board answer: in chronic stable angina β-blockers do not reduce MI or mortality – only in pts w/hx of MI or CHF
Choice of β-Blockers

- All β-blockers are equally effective
- Cardioselective, longer-acting agents generally preferred
  - Atenolol 25 mg daily (max 200 daily)
  - Metoprolol 25 mg twice daily (max 200 bid) – may switch to metoprolol succinate (Toprol)
- Resting bradycardia: use agent with intrinsic sympathomimetic activity (ISA)
  - Pindolol
  - Acebutolol

Adequate β-Blockade

- Resting HR 50 – 60.
- Lower HR may be acceptable for pts w/persistent sx, if bradycardia <50 is asymptomatic and there is no heart block.
- Therapeutic efficacy—fewer angina episodes, less use of NTG.
- If exercise testing is done, peak HR & BP during exercise should be blunted.

β-Blocker Side Effects

- Bradycardia
- Heart block
- Bronchoconstriction
- Worsening of PAD
- Can precipitate or exacerbate CHF – esp decompensated HF (6% w/carvedilol)

Weird β-Blocker Side Effects

- Fatigue – 1.8% (NNH = 57)
- Sexual dysfunction – 0.5% (NNH =200)
- Weight gain (1.2 kg – study in HTN, not CAD)
- + ANA ~5 –10%. However, clinical sx are unusual.
  - Acebutolol may be biggest offender.
  - Reversible upon discontinuation of drug.
Side Effects Probably NOT Attributable to β-Blockers

- Recent studies have not shown more hypoglycemia in diabetics on cardioselective or ISA β-blockers
- Only minor ↑ in K have been shown, & hyperkalemia is rare without other stimuli
- No increased risk of depression

Common/Significant Drug Interactions With β-Blockers

<table>
<thead>
<tr>
<th>Drug</th>
<th>Effects</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>Cardiac arrest</td>
<td>Extreme caution</td>
</tr>
<tr>
<td>Antidiabetic agents</td>
<td>HTN, poss ↓ glc</td>
<td>Monitor</td>
</tr>
<tr>
<td>Rate-sparing CCB</td>
<td>Brady, CHF, hypotension</td>
<td>Avoid (few clinical issues, however)</td>
</tr>
<tr>
<td>Antagonists (diltiazem, verapamil)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>Worsening bradycardia</td>
<td>Monitor. OK in angina + low EF (? benefit)</td>
</tr>
<tr>
<td>Epinephrine, sympathomimetics</td>
<td>HTN crisis</td>
<td>Avoid if possible</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>↑ lido level</td>
<td>↓ lido dose (NOT listed in ACLS protocol)</td>
</tr>
</tbody>
</table>

Calcium Channel Blockers

- Coronary & peripheral vasodilation → increased coronary artery flow
- Reduced BP & negative inotropy → reduced myocardial oxygen demand
- Reduce angina sx & increase exercise tolerance (no survival benefit)

When to Use CCB’s in Angina

- β-blockers contraindicated
- Side effects from β-blockers
- Add to β-blocker if β-blocker monotherapy ineffective (caution—drug interactions)
- Agent of choice for Prinzmetal’s angina
Variant (Prinzmetal’s) Angina

- Spontaneous coronary artery spasm.
- Angina occurring at rest.
- Usually normal exercise tolerance.
- May be provoked by hyperventilation.
- Tends to occur in early morning.
- Pain may be severe.
- Pts tend to have fewer risk factors, usually younger.

Variant (Prinzmetal’s) Angina – ECG

- Transient ST elevation during episode.
  - Probably represents transmural ischemia.
  - T wave inversions may follow prolonged or severe ischemia in variant angina.
- Arrhythmias may be a life-threatening complication.
  - RCA → heart block.
  - LCA → VT

Dx of Variant Angina

- Dobutamine echo—sensitive, specific.
- Cardiac cath.
  - Ergonivine provokes vasospasm.
- Standard exercise treadmill testing is useless.
- Note: may coexist with obstructive CAD.

Treatment of Variant Angina

- CCB’s: nifedipine, verapamil, diltiazem.
- Nitrates: may be used in monotherapy, or added to CCB.
- Avoid:
  - β-blockers—esp nonselective.
  - ASA—caution (inhibits prostacyclin).
  - Triptans (provoke vasospasm).
Efficacy of Antianginal Drugs

- β-blockers = CCB’s.
- Felodipine (Plendil™) + metoprolol > either alone.
- Insufficient data on nitrates vs others.

Non-Pharmacologic Therapy: Risk Factor Reduction

- The usual:
  - Treat HTN
  - Treat hyperlipidemia (statins)
  - Weight loss if indicated
  - Glycemic control in diabetes
  - Stop smoking
  - Aerobic exercise – start low, go slow. Better than stenting!

Brief Review/Preview of LDL Goals

- 0 – 1 risk factors: goal LDL = 160
- 2 risk factors: goal 130, OPTION of 100
- CAD or CAD equivalent: 100, OPTION of 70, esp for “very high risk pts”:
  - Multiple risk factors (esp DM) OR
  - Severe/poorly controlled risk factors (esp smoking) OR
  - Metabolic syndrome (esp TG’s > 200 + HDL < 40 + non-HDL-C ≥130) OR
  - Acute coronary syndrome

PCI vs Optimal Medical Therapy of Stable Angina

- NO difference in outcomes (all-cause mortality, nonfatal MI, composite CV dz endpoint) in pts receiving optimal medical therapy vs those who received PCI (bare metal stent):
  - Antianginal drug
  - Ant platelet drug
  - Stating
Pts in Whom Revascularization MAY Be Useful

- Medical therapy does not provide sufficient relief.
- Intolerant of medical therapy.
- "High risk":
  - L main dz
  - 3 vessel dz, esp w/low LVEF

Stress Testing in Stable Angina

- 2005 ACC/AHA guidelines recommend stress testing for most pts w/stable angina in order to:
  - Evaluate efficacy of therapy
  - Obtain prognostic info
  - Identify "high risk" pts who might need PCI
- Editorial comment: let's see what they say now post-COURAGE trial.

Acute Coronary Syndrome

- Definition of ACS: MI or unstable angina.
- Compromised blood flow to viable heart muscle.

Unstable Angina

- Unstable angina:
  - Angina at rest (usu > 20 min)
  - New onset angina limiting physical activity
  - Increasing angina
    - More frequent
    - Longer duration
    - Occurs with lower exertion
UA vs MI

- Unstable angina:
  - NO elevation in cardiac enzymes
  - +/- ischemic ECG changes—transient
- MI:
  - Elevated cardiac enzymes
  - Evolving ECG changes
- Cardiac enzymes may not rise for several hours, so UA may be indistinguishable from non-ST elevation MI at presentation.

Other Life-Threatening Causes of Chest Pain

- Aortic dissection
- Pulmonary embolism
- Tension pneumothorax
- Esophageal rupture
- Perforated peptic ulcer

Step 1

- ABC’s
- ECG
- Monitor
- Oxygen (Keep S\textsubscript{p}O\textsubscript{2} ≥90%)
- IV, labs

Step 1a: Initial Meds

- Immediate ASA 162 – 325 mg chewed.
  - Need rapid absorption—do NOT use EC.
  - Do not use if anaphylactic reaction.
- Sublingual NTG 0.4 mg q 5 min X3
- Morphine 2 – 4 mg IV. Repeat prn.
  - Relieves pain, anxiety.
  - Reduces sympathetic stimulation caused by pain, anxiety
Nitrate Precautions in ACS

- Contraindicated if PDE-5 inhibitors within 24 hrs (hypotension):
  - Sildenafil (Viagra)
  - Vardenafil (Levitra)
  - Tadalafil (Cialis) – may need to wait 36 hrs.
- Extreme caution if inferior MI & possible right ventricular involvement
  - Dependent on preload to maintain cardiac output

Step 2: Look at the ECG

- ST elevation
  - ST-elevation MI
  - Prinzmetal’s angina
- No ST elevation
  - ST depression – angina or NSTEMI
  - T wave inversions – NSTEMI or increased risk for acute MI

Initial ECG in ACS

- Initial ECG may be non-diagnostic in 45%, normal in 20%
- Early abnormalities include hyperacute T waves
- If initial ECG is non-diagnostic in a pt in whom there is high suspicion of MI (including continued sx), repeat ECG every 5 – 10 minutes (ACC/AHA).

http://www.uptodate.com/online/content/topic.do?topicKey=ad_emer/2821
Diagnosis of Acute MI

- Rise & fall of cardiac biomarkers AND at least one of:
  - Ischemic sx
  - ECG changes
  - Imaging evidence of new myocardial loss or wall motion abnormality

Cardiac Enzymes

- Troponins most sensitive & specific
- CK-MB & myoglobin rise first
- Low sensitivity until ≥4 – 6 hrs after sx onset
- Enzymes may not rise for 12 hrs

Timing and Cardiac Enzymes

- 1st 6 hrs:
  - CK-MB most sensitive
  - CK-MB = myoglobin for specificity

<table>
<thead>
<tr>
<th>Biomarker</th>
<th>Onset</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoglobin</td>
<td>1 – 4 hr</td>
<td>6 – 7 hr</td>
<td>24 hr</td>
</tr>
<tr>
<td>CK-MB</td>
<td>3 – 12 hr</td>
<td>18 – 24 hr</td>
<td>36 – 48 hr</td>
</tr>
<tr>
<td>Troponins</td>
<td>3 – 12 hr</td>
<td>18 – 24 hr</td>
<td>7 – 10 days</td>
</tr>
</tbody>
</table>
Nonischemic Causes of Elevated Cardiac Enzymes

- CHF
- Myocarditis
- Cardiac ischemia/injury without infarction
- PE
- Rapid atrial fib
- Chronic (or acute) renal insufficiency

ECG Localization of MI: ST Elevations

- V1 – V2: septal, anteroseptal
- V3 – V4: anterior, anteroseptal
- V4 – V6: lateral
- I, aVL: lateral
- II, III, aVF: inferior
  - Check V4R – V6R (ACC/AHA)

- ST depressions in V1-V2: consider posterior MI (check V7-V9)

STEMI

- ST elevation ≥1 mm in 2 contiguous leads
  - Not due to pericarditis or LV aneurysm
  - If ST elevation resolves after NTG, repeat ECG while pain-free to R/O variant angina.
  - In setting of high suspicion of ACS, new LBBB should be considered STEMI.

Management of STEMI

- Job #1: Reperfusion
  - If <12 hrs since onset of sx, PCI or thrombolysis is indicated.
  - PCI is preferred if it can be accomplished within 90 minutes.
  - If PCI not available or not within 90 min, thrombolysis is an acceptable alternative.
- Do not wait for cardiac biomarkers. Just do it!

http://www.uptodate.com/online/content/topic.do?topicKey=ad_emer/2821#7
Meds in STEMI

- Already received ASA.
- Clopidogrel has benefits (↓ death, MI) in pts undergoing thrombolysis, and maybe in those undergoing PCI (cardiologists use it in both). Continue 1 – 12 months.
- GP IIb/IIIa inhibitors (abciximab = ReoPro) prior to PCI.
- Heparin (per ACC/AHA, even though evidence poor).

“PACH” The Ailing Heart

- Platelet inhibitors (GPIIb/IIIa)
- ASA
- Clopidogrel
- Heparin

More Meds in STEMI

- β-blockers if not contraindicated.
- IV NTG if persistent pain, CHF, HTN
  - D/C if BP too low—more important to give β-blockers
- Replete K if below 4 (2X ↑ in VF if < 3.6).
- Prior to D/C:
  - ACEI, esp if abnormal LVEF or uncontrolled risk factors.
  - Statin—high dose (atorvastatin 80 mg).

ABC’s of STEMI

- ASA
- B-blockers
- Clopidogrel if reperfusion
Testing After STEMI

- **LVEF**: ↓ EF → ↑ mortality.
  - Echo—wait for recovery after reperfusion (stunned myocardium) – > 14 days.
- **Stress test** (guide CV rehab, eval for residual ischemia):
  - If revascularization: few wks after D/C.
  - No revascularization: pre-D/C, if no recurrent angina or CHF.

Activity Level After STEMI

- Encourage daily activity.
- Activity guided by cardiac rehab.
- Most return to work within 2 weeks.

Non-ST Elevation ACS

- ACS without ST elevation – unstable angina or NSTEMI
  - UA: little/no rise in cardiac enzymes
  - NSTEMI: enzymes rise—wait 4 – 12 hrs
- (Risk stratification: TIMI risk score
  - High risk: may benefit from early PCI)

NSTEMI Management

- Similar to STEMI except no reperfusion
- ASA
- β-blocker
- Oxygen
- Morphine
- NTG
NSTEMI Management: Differences vs STEMI

- Enoxaparin instead of heparin (if no renal failure, and no CABG within 24 hr)
- No reperfusion \(\Rightarrow\) pre-D/C stress test (as in STEMI)
- Measure LVEF – echo (same)
- Statin (same)
- ACEI if EF <40%, DM, HTN (same)

ACS Without Chest Pain

- Up to 1/3 w/ACS do not have chest pain.
  - SOB
  - N +/-V
  - Palpitations
  - Syncope
  - Cardiac arrest
- More likely to be older, diabetic, women.
- Higher mortality

Diagnosis of AMI w/LBBB

- 1 – 7% of AMI pts have LBBB
- 50% of LBBB pts with MI have no chest pain
- Fewer than 1/3 of LBBB pts presenting w/chest pain had MI

Look at ST Segments

- Serial ECG changes most sensitive (67%).
- ST elevation: \(\geq\) 1mm in same direction as QRS—highly specific

http://www.uptodate.com/online/content/topic.do?topicKey=ecgs/6119&selectedTitle=3~150&source=search_result
More Signs of MI In LBBB—Advanced Course

- Initial + deflection in V1 AND Q in V6—only 20% sensitive, but highly specific (~100%) for anteroseptal MI.
- Cabrera’s sign: prominent notch in ascending part of S wave in V3 & V4—specific, not sensitive for MI
- Chapman’s sign: prominent notch in ascending part of R in V5 or V6—specific, not sensitive

Congestive Heart Failure

- Heart is unable to deliver blood at a rate sufficient to meet the body’s metabolic needs.
- >5 million Americans w/CHF.
- Nearly 1 million hospitalizations per year in US.
- Readmission rate within 6 months is up to 50%.

Mortality in CHF

- Depends on:
  - Age
  - NYHA class
  - Gender (women have better prognosis)
- Diastolic HF has better prognosis
  - Systolic HF: 15 – 19%/yr mortality
  - Diastolic HF: 8 – 9%/yr
  - Matched controls: 1 – 4%/yr

Causes of Death in CHF

- Progressive pump failure
- Arrhythmias/SCD

http://uptodateonline.com/online/content/topic.do?topicKey=hrt_fai/18548#7
Risk Factors for CHF

- Myocardial ischemia – #1 cause of systolic HF
  - Severe CAD
  - h/o MI
  - Evidence of hibernating myocardium
  - NB: single vessel dz with no MI ⇒ prognosis = nonischemic cardiomyopathy
- HTN (#1 cause of diastolic HF)
- LVH
- African-Americans, Latinos, Native Americans (probably related to HTN)

Types of CHF

- Systolic: ventricles don’t pump well
  - Poor contractility
  - Low LVEF
- Diastolic: ventricles don’t fill well
  - Noncompliant (stiff) heart can’t relax
  - Hypercontractility
  - NI – high LVEF
- ~ 50/50

Causes of Systolic CHF

- Ischemic myocardial disease, CAD
- Alcoholic cardiomyopathy
- Diabetic cardiomyopathy
- Cocaine cardiomyopathy
- Drug-induced cardiomyopathy (eg, doxorubicin)
- Idiopathic cardiomyopathy
- Peripartum cardiomyopathy
- Myocarditis
- Preterminal valvular heart disease
- Congenital heart disease with severe pulmonary hypertension

Causes of Diastolic CHF

- Hypertension
- Severe aortic stenosis
- Hypertrophic cardiomyopathy
- Restrictive cardiomyopathy
- Ischemic myocardial disease, coronary artery disease
Clinical Clues to CHF Causes

- H/O angina: ischemic heart dz
- Sx after flu-like illness: myocarditis
- HTN
- ETOH
- Murmur: valve dysfunction
- Med list: verapamil, β-blockers, NSAID’s

CHF Symptoms

- History alone is sufficient to make dx.
- Fluid accumulation
  - SOB
  - Edema
  - RUQ discomfort (liver), ascites (esp w/R HF)
- Reduced cardiac output
  - Fatigue
  - Generalized weakness/malaise
  - Anorexia

Dyspnea in CHF

- Usual order of sx progression:
  - Exertional dyspnea is earliest
  - Orthopnea is next (chemistry: adjacent)
  - Paroxysmal nocturnal dyspnea
  - Dyspnea at rest
  - Acute pulmonary edema

Clinical Clues to Diastolic HF

- Asymptomatic diastolic HF is more common than symptomatic.
- Exercise intolerance – may occur in pts w/diastolic dysfunction w/o overt CHF.
- Tolerate tachycardia poorly (incomplete filling).
- Tolerate AFib poorly (need atrial kick).
- Acute ischemia raises LA & pulmonary venous pressures ⇒ respiratory sx (SOB, wheeze, pulmonary edema) – may occur w/o typical angina ⇒ angina equivalents.
Acuity of Presentation

- Acute/subacute CHF (days – weeks) tends to present w/respiratory sx (DOE, orthopnea).
  - Acute RHF may present w/RUQ discomfort
- Chronic CHF (months) tends to present with systemic sx (fatigue, anorexia) and edema.

Physical Exam in CHF

- $S_3$ gallop:
  - Early finding
  - Specific, but not sensitive
  - High interobserver variability
- Neurohumoral (sympathetic) activation:
  - Sinus tachycardia
  - Diaphoresis (sympathetic cholinergic)
  - Cool, pale extremities (vasoconstriction)

Pulsus Alternans in CHF

- Alternating strong & weak pulses
- Upon slow release of BP cuff, phase I Korotkoff sound (1st BP sound) heard only with strong pulses
- Indicates severe LV failure

Volume Overload Signs in CHF

- Pulmonary congestion
  - Crackles
  - More prominent in acute/subacute HF
- Peripheral edema
  - Ascites
  - Hepatosplenomegaly
  - Hepatojugular reflux
- Elevated jugular venous pressure
Subtler Signs

- Accentuated P2 – pulmonary HTN
- Systolic murmur
  - Mitral regurg
  - Tricuspid regurg
  - Due to ventricular dilation
  - Resolve or improve with better compensation

Severity of CHF

- NYHA
  - Class I - symptoms of HF only at activity levels that would limit normal individuals
  - Class II - symptoms of HF with ordinary exertion
  - Class III - symptoms of HF with less than ordinary exertion
  - Class IV - symptoms of HF at rest
- Therapeutic decisions still based on this system

New ACC/AHA Staging System

- Stage A — High risk for HF, without structural heart disease or symptoms
- Stage B — Heart disease with asymptomatic left ventricular dysfunction
- Stage C — Prior or current symptoms of HF
- Stage D — Refractory end stage HF

Diagnostic Modalities in CHF

- CXR: cephalization of flow, cardiomegaly, Kerley (not curly) B lines, pulmonary edema, pleural effusion
- ECG: arrhythmia, AV block, evidence of ischemic heart dz, LVH
Echocardiography in CHF

- Recommended for all new CHF pts (ACC/AHA)
  - LVEF < 55% ⇒ systolic failure
  - Diastolic dysfunction, abnormal filling, LVEF >75% suggest diastolic failure.
    - Reduced E:A ratio
    - Early flow across mitral valve / atrial contraction
    - Impaired LV relaxation (diastolic dysfx) ⇒ ↓ early diastolic mitral flow + increased late diastolic filling due to atrial kick

Laboratory W/U of CHF

- CBC (anemia exacerbates CHF)
- Lytes, Cr (follow diuretics, ACEI, ARB)
- LFT’s (hepatic congestion)
- Fasting glc (DM)
- If dilated cardiomyopathy of ? cause:
  - TSH
  - Fe studies (%sat) – hemochromatosis

B-Natriuretic Peptide in CHF

- BNP & nT-proBNP rise with worsening CHF
- BNP or nT-proBNP added to clinical judgment is better than either alone
- Normal levels are sensitive for ruling out CHF in pts with dyspnea
  - nT-proBNP < 300 ⇒ 98% negative predictive value
  - BNP < 100 ⇒ 90% NPV; <50 ⇒ 97%
- ↑ level ⇒ ↑ mortality in hospitalized pts & post-hospitalization (nT-proBNP better).
Limitations of BNP Measurement

- Atrial fib raises BNP $\Rightarrow$ reduced specificity in AF (must use higher cutoff).
- Both are higher CKD, PE, lung disease (RHF), acute noncardiac illness (sepsis).
- Both are lower in obese pts.

Treatment of CHF

- Chronic systolic HF:
  - Correct underlying causes (HTN, CAD, valvular dz)
  - Treat contributing/complicating conditions (hypothyroid, DM)
  - Reduce sodium intake
  - D/C ETOH
  - Healthy weight
  - Exercise training reduces sx & hospitalization, increases survival & QOL!

Pharmacotherapy of Systolic HF

- Loop diuretics—sx relief only, no survival benefit.
- ACEI in all pts (ARB if ACEI-intolerant).
  - Improved survival (asymptomatic – severe).
  - Check K & Cr 1-2 wk after dosage changes.
- β-blockers once stable on ACEI.
- Digoxin if continued sx, or for rate control in A fib.

β-Blockers in Systolic HF

- Use in all systolic HF pts unless contraindicated.
- Carvedilol, metoprolol (esp XL), bisoprolol.
- Reduce all-cause mortality, hospitalization.
- Best evidence in NYHA class II & III HF, probably class IV.
- Avoid agents with ISA.
- Sx may worsen for 4 – 10 weeks prior to improving.

http://uptodateonline.com/online/content/topic.do?topicKey=hrt_fail/6883&selectedTitle=2~150&source=search_result#49
Role of Digoxin in Systolic HF in 21st Century

- LVEF <40%
  - Especially NYHA II – IV with persistent sx despite optimal therapy.
  - Maintain serum concentration 0.5 – 0.8.
  - Serum concentration ≥ 1.2 in women is associated with greater mortality.
- May also be used in AFib pts for rate control.
- No survival benefit

Aldosterone Antagonists

- Spironolactone (cheap, old standby), eplerenone (Inspra™, fewer side effects but much more costly).
- Survival benefit in moderate-severe systolic HF.
- Closely monitor Cr, calculated GFR, K.
- Beware hyperkalemia

African-Americans & Systolic HF

- ACEI’s may be less effective in prolonging survival.
- β-blockers may have reduced survival benefit.
- May derive greater benefit than Caucasians from hydralazine + nitrates, esp NYHA III - IV.
  - May be reasonable in any pt who does not respond to regimen—can be added.

Ω-3 Fatty Acids

- Ω-3 polyunsaturated fatty acids (PUFA’s) reduce all-cause mortality and combined endpoint of mortality + CV-related hospitalization.
Drugs to Avoid or Use With Caution in CHF

- NSAID’s
- Thiazolidinediones ("glitazones")
- Metformin
- Cilostazal (Pletal™) – for claudication: ↑ mortality
- PDE-5 inhibitors (sildenafil, vardenafil, tadalafil).

Treatment of Diastolic HF

- Less evidence.
- ACC/AHA recommends only 4 modalities:
  - Control of systolic and diastolic HTN
  - Control of ventricular rate in pts w/atrial fib
  - Control of pulmonary congestion and peripheral edema with diuretics
  - Coronary revascularization in patients with CAD in whom ischemia is judged to have an adverse effect on diastolic function

Preload & Diastolic HF

- Poorly compliant, hypercontractile LV is very sensitive to preload reduction.
  - Worsening cardiac output.
  - Hypotension.
- Caution with:
  - Diuretics
  - ACEI (no survival benefit in these pts; may reduce myocardial fibrosis, improving diastolic function)
  - Nitrates (β-blocker or CCB preferred in CAD)
- Sx of ventricular underfilling: weakness, dizziness, near syncope, syncope

Digoxin in Diastolic HF

- There is no role for digoxin in diastolic HF.
  - Contractility is already fine.
  - No benefit.
Treatment of Acute Decompensated CHF

- Consider hospitalization—esp if sick.
- 2 g Na restriction.
- Fluid restriction.
  - 2L/day if Na < 130.
  - Stricter if Na < 125 or worsening.
- Oxygen

Pharmacotherapy of Acute Decompensated HF

- Basically, systolic = diastolic
- IV loop diuretics (expect & tolerate mild ↑Cr)
- Consider IV morphine, esp w/acute MI
- Consider vasodilators:
  - For rapid sx improvement in admitted pts on diuretics—if no hypotension.
  - Rapid sx relief in pulm edema or severe HTN.
  - Persistent severe HF despite aggressive tx.

Vasodilators in CHF

- IV NTG
  - Hypotension
  - HA
- IV nitroprusside
  - Cyanide accumulation
  - Limit to 48 hrs
- IV nesiritide
  - BNP analogue
  - Increased mortality

Continuation of Chronic HF Meds

- ACEI/ARB: continue med during decompensation.
  - Reduce in hypotension, hyperkalemia, ARF.
- β-blockers:
  - Mild decomp: continue med (↑ mortality if not).
  - Moderate-severe decomp: reduce or hold med initially.
  - If not on, start prior to D/C, but not in earliest phase.
Summary of Differences in Management of Decompensated HF

- Systolic:
  - Reduce or hold ACEI, β-blocker (usual chronic systolic meds!!) if mod-severe decomp.
- Diastolic:
  - Control BP & tachycardia, so ACEI & β-blocker may be useful acutely.
  - Do not use inotropes.

So Who Gets an ICD in Chronic CHF?

- Survivors of SCD
- Symptomatic VTach
- Ischemic CM + LVEF ≤30%
- Nonischemic CM:
  - NYHA II – III + LVEF ≤35%
  - NYHA III – IV + LVEF ≤35% + wide QRS (>120) – use combo ICD + biventricular pacer

Asymptomatic LV Dysfunction

- Treatment with ACEI (enalapril) in pts w/LVEF ≤40% delays onset of overt CHF and prolongs life.
  - Low EF defines LV dysfunction.
  - However, CHF requires signs/symptoms.
  - Therefore, this is not treatment of CHF.

Heart Murmurs

- Timing
  - Systolic – SEM, holosystolic, late systolic
  - Diastolic – early, mid-late
  - Continuous – systolic + diastolic
- Intensity
- Location (where is it loudest?)
- Radiation
Systolic Murmurs
- Ejection type (crescendo-decrescendo) → outflow tract stenosis or flow murmur.
  - Aortic outflow tract
    - Aortic stenosis (AS)
    - Hypertrophic obstructive cardiomyopathy (HOCM)
    - Aortic flow murmur
  - Pulmonary outflow tract
    - Pulmonic valve stenosis
    - Pulmonic flow murmur

More Systolic Murmurs
- Holosystolic (= pansystolic) → AV valve regurgitation (mostly)
  - Mitral regurgitation (MR)
  - Tricuspid regurgitation (TR)
  - VSD
- Late systolic → AV prolapse (+/- regurg)
  - Mitral valve prolapse (MVP)
  - Tricuspid valve prolapse

Diastolic Murmurs
- Early diastolic → analogous to early systolic
  (i.e., SEM): outflow tract – but regurgitation
  - Aortic regurgitation (AR)
  - Pulmonic regurgitation
- Mid-late diastolic → analogous to holosystolic: AV valve, but stenosis (mostly)
  - Mitral stenosis (MS)
  - Tricuspid stenosis
  - Mitral or tricuspid flow murmur

Aortic Stenosis
- Congenital (bicuspid, hypoplastic) tends to present earlier.
- Degenerative (calcific) tends to be more prevalent with age.
- Rheumatic
  - #1 in world, less common in US.
  - Usually occurs w/mitral valve dz.
Aortic Stenosis – 2

• May be asymptomatic even with severe dz.
• Once sx appear, course tends to progress fairly rapidly (2-3 yr avg survival).

Aortic Stenosis:  Sx

• Classic triad: angina, syncope, HF (once sx appear, it turns you to ashes). Esp w/exertion.
• Exertional dyspnea is most common presenting sx (↓ exercise tolerance).
• Atrial fibrillation, pulmonary hypertension are preterminal findings.
• Rare complication of calcific AS = Ca++ emboli

Aortic Stenosis:  Exam

• Pulsus parvus et tardus
• Prominent S4 (LVH).
• Crescendo-decrescendo murmur loudest @ base (R 2nd ICS).
  – Radiates to B carotids.
  – May be prominent @ apex in elderly
  – More severe stenosis ⇒ longer & later murmur (lousier). Intensity not related.
  – ↓ w/valsalva

Aortic Stenosis:  Other

• ECG: LVH common.
• CXR: rounded LV border.
AS Management

• Risk factor reduction.
  – High prevalence of CAD.
  – Cautious treatment of HTN is appropriate.
• Exercise:
  – Mild AS: competitive sports OK w/F/U.
  – Asymptomatic mod AS: exercise testing to level of exertion.
  – Asymptomatic mod AS w/SVT or other complex tachyarrhythmias: low-intensity.
  – Severe AS: no sports.

AS Surgical Treatment

• Valve replacement
  – Symptoms
  – Rarely for asymptomatic pts
• Valve replacement prolongs life and improves sx
• Complications of surgery:
  – Stroke
  – Prolonged ventilation
  – Perioperative mortality
• Valvuloplasty may help congenital AS

Aortic Regurgitation (Insufficiency)

• Etiology:
  – Valve leaflet damage
    • Endocarditis
    • Congenital bicuspid aortic valve (#1 in US)
    • Rheumatic (#1 in world)
  – Aortic root dilation
    • Marfan’s
    • Syphilis
    • Dissection
• More common with advancing age

Aortic Insufficiency Sx

• Chronic:
  – Long asymptomatic period
  – Progressive exertional dyspnea, CHF
  – Pounding heart, palpitations (worse when supine or on L side—heart vs chest wall)
• Acute: cardiogenic shock
Physical Exam in AI

- Wide pulse pressure
- Water-hammer pulse (↑ stroke vol ➔ rapid pressure fall d/t regurg)
- Blowing, decrescendo, early diastolic murmur
  - LSB/base: valvular
  - RSB/apex: root dilation
- More severe AI ➔ longer duration of murmur

Prognosis in AI

- More severe heart failure ➔ worse prognosis
- More dilated LV ➔ worse prognosis
- Activity limitations based on severity of AI + LV diameter
- NS VT ➔ restrict activity

Management of AI

- Vasodilators (nifedipine, ACEI) slow progression of AI and reduce risk of CHF
  - Use in symptomatic pts.
  - May help asymptomatic.
- Avoid β-blockers – slow rate prolongs diastole, may worsen regurg.
  - Exception: mild AI w/bicuspid valve
- Consider surgery (AVR) if symptomatic, increasing LV dilation, worsening LV function.

Mitral Stenosis

- Usually rheumatic
- Sx begin in 30’s
  - SOB, DOE
  - Pulmonary edema assoc w/exertion, fever, anemia.
  - Occasional hemoptysis.
  - PE may occur.
- Endocarditis rare.
MS Exam

- Loud S1.
- Opening snap.
- Diastolic rumbling murmur @ apex.
- R sided CHF signs occur later.

MS Management

- Treat CHF
  - Percutaneous balloon valvuloplasty.
    - Uncomplicated MS, limited valve Ca++. Contraindicated in severe MS, LA thrombus.
  - Surgical valvotomy—good results.
  - Valve replacement.
    - Moderate – severe disease + CHF
    - Not candidate for valvotomy
  - Complication: A fib.

Mitral Regurgitation

- Trivial (“physiologic”) MR occurs in up to 70% of adults.
- Etiology of pathologic MR:
  - Mitral valve prolapse (#1 in developed areas)
  - Endocarditis
  - Ischemic heart dz – MI can cause acutely
  - Trauma
  - Rheumatic (undeveloped nations)
  - Congenital
  - Drugs: cabergoline, diet pills, ergotamine

Symptoms of MR

- Chronic:
  - Asymptomatic until severe w/LV failure or AF
  - DOE, ↓ exercise tolerance, fatigue (↓ CO)
  - A fib
  - Higher risk of endocarditis w/severe dz
- Acute:
  - Pulmonary edema
  - Hypotension, cardiogenic shock
Physical Exam in MR

- Enlarged LV ➔ leftward PMI, usu hyperdynamic
- Murmur:
  - Usually holosystolic
  - Loudest @ apex, radiates to L axilla, may radiate to back
  - Intensity of murmur does not correlate with severity of MR
  - Murmur may vary depending on leaflet involved
- Prolapse has midsystolic click, sl later murmur

Management of MR

- Vasodilators (ACEI) – controversial in asymptomatic pts (often recommended
- Treat ischemia if present
- Treat signs of CHF
- Manage A fib
- Consider surgery if sx.
  - Repair preferred for most pts (better results)
  - Ideal to operate before EF deteriorates
  - MVR if extensive Ca++ of leaflet or annulus.

Who Gets SBE Prophylaxis?

- Prosthetic heart valves
- Prosthetic material used in valve repair
- Prior h/o endocarditis
- Transplant valvulopathy
- Congenital heart dz:
  - Unrepaired cyanotic dz
  - Repaired w/prosthetic material—for 6 mo p-procedure
  - Repaired w/residual defects at or near prosthetic device

Circumstances for Prophylaxis

- Dental procedures involving manipulation of gums or pariapical tissue, or perforation of mucosa.
- Incision or bx of respiratory tract mucosa (T & A, bronch w/bx)
- Infected skin or musculoskeletal tissue undergoing surgery

http://www.uptodate.com/online/content/topic.do?topicKey=endocard/7638&selectedTitle=2-6#8
NO Prophylaxis

- Other valve lesions
- No GI or GU procedures
  - Exception: established UTI w/hi risk CV condition
- Vaginal or Cesarean delivery
  - Exception: hi risk lesion w/chorioamnionitis or pyelonephritis

Meds for Endocarditis Prophylaxis

- Single dose 30 – 60 min before procedure — no more 2\textsuperscript{nd} dose!
- Amoxicillin 2 g
  - NPO: IV/IM amp (2g), cefazolin, ceftriaxone (1g)
  - PCN allergic:
    - Po cephalexin 2g, clinda 600 mg, or azithro or clarithro 500 mg
    - IM/IV cefazolin, ceftriaxone, or clinda