Acute Coronary Syndrome (ACS): Evidence Based Trends and Treatment

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Opening Questions

- Patient arrives in ED with chest pain. ECG with no ST changes. Chest pain continues. How often is it beneficial to repeat ECG?
- How do you definitively differentiate unstable angina from NSTEMI (Non ST Elevation MI)?
- Patient non NSTEMI admitted to CCU. When do you proceed to catheterization?
- Name 3 “high risk” clinical features that place the ACS patient with no ST elevation at increase risk of death.
- Which of the following medications have mortality benefit in the treatment of STEMI (ST Elevation MI)?
  - Nitroglycerin
  - Morphine Sulfate
  - Metoprolol
  - Diltiazem
Hot Off the Press

**ARMYDA-6**
- 1sr prospective, randomized trial comparing 600 mg loading of clopidogrel with a standard 300-mg dose in STEMI patients undergoing PCI.
- Results confirm that the higher dose is preferable.

**Bivalrudin**
- Randomized PROBI-VIRI trial (Prolonged Bivalirudin Infusion Versus Intraprocedural only) compared to heparin plus abciximab.
- Continuing bivalirudin for four hours after a percutaneous intervention for ST-segment elevation myocardial infarction (STEMI) improves microvascular reperfusion without extra bleeding complications.
- Follow Up Study: Multicenter randomized clinical trial called the Effect in Reducing Infarct Area of a prolonged infusion of Bivalirudin in Primary PCI study.

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Hot Off the Press

- Focus on DIDO time in addition to Door to Device Time

- New sensitive troponin assays

- GI Bleeding common after PCI in STEMI

- Gaps persist in under treatment of women with ACS
  - ACE-I
  - Lipid lowering agents
Acute Coronary Syndrome refers to any rupture of plaque or thrombotic event that leads to symptomatic ischemia or infarction.

Acute Coronary Syndrome (ACS)

- No ST Elevation
  - NonSTEMI
  - Unstable Angina
- ST Elevation
  - STEMI
Hospitalizations in the U.S. Due to ACS

Acute Coronary Syndromes*

1.57 Million Hospital Admissions - ACS

UA/NSTEMI†  STEMI

1.24 million Admissions per year  0.33 million Admissions per year

*Primary and secondary diagnoses. †About 0.57 million NSTEMI and 0.67 million UA.

Pathophysiology of ACS

- Deposit of lipids, calcium, fibrin, and other cellular substances within the lining of the arteries.
- Initiates a progressive inflammatory response in an effort to heal the endothelium.
- End result of inflammatory process: the production of a fibrous atherosclerotic plaque.
- Plaque can progress to cause coronary stenosis.
- Plaque can also rupture prior to causing significant stenosis.
Acute Myocardial Infarction

- Development of myocardial necrosis caused by a critical imbalance between the oxygen supply and demand of the myocardium
- 10 seconds of oxygen deprivation: Ischemia
- 1 minute of Ischemia: Myocardial function affected
- 20 minutes of oxygen deprivation: Irreversible cell damage

STEMI

NSTEMI

Plaque

- Stable plaque of stable angina
  - Thick fibrous caps separate the lipid core from the endothelium
  - Less complicated than vulnerable plaques
  - Tend to have smooth outlines

- Vulnerable plaque of ACS
  - Thin caps
  - Edge of the fibrous cap is a particularly vulnerable area and is commonly the location of ruptured plaque

- Limitations of stress testing and cardiac catheterization
- Intravascular ultrasound
Differential Diagnosis of Chest Pain

Assessment of Pain
Linking Patient History and Risk factors
Cardiac Biomarkers
ECG Findings

ACS Symptoms

<table>
<thead>
<tr>
<th>Classic Symptoms</th>
<th>Symptom Variations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stable angina</td>
<td>Women</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>Elderly</td>
</tr>
<tr>
<td>MI</td>
<td>Diabetics</td>
</tr>
</tbody>
</table>
Angina

Stable
- Occurs with physical exertion or emotional stress
- Relieved by rest or sublingual nitroglycerin
- Predictable pattern
- Predictable = triggered by the same amount of physical or emotional stress and should be easily relieved by rest or sublingual nitroglycerin.

Unstable
- Occurs with minimal exertion
- OR increased dose of nitroglycerin is required to achieve relief.
- Prolonged rest angina is also considered unstable angina.
- Angina that increases in severity or is very severe on first presentation
- Caused by unstable or ruptured plaque that causes abrupt closure of a coronary artery which may spontaneously reperfuse.

Assessment of Angina

- N = Normal
- O = Onset
- P = Precipitation / provoking / palliative factors
- Q = Quality or quantity
- R = Radiation and region
- S = Severity
- T = Time
Characteristics of Angina

Sensation of pressure, tightness, heaviness, burning, or squeezing.

- Rarely described as a sharp or stabbing pain.
- Should not worsen with changes in position or respiration.

Location behind the sternum and in the upper back, shoulder, arm, jaw, or epigastric area.

- Not usually located in the middle to lower abdomen and does usually not radiate to the lower extremities.

Associated symptoms (or stand alone symptoms) of dyspnea, nausea, palpitations, or diaphoresis.

Duration typically defined in minutes.

- Not typically defined in seconds or hours.

CAUTION WHEN ASKING THE PATIENT ABOUT “PAIN”!

Angina in Women

- Delay presenting with symptoms
- Attribute symptoms to other non-cardiac causes
- Presentation
  - epigastric discomfort
  - less specific complaints: dyspnea or fatigue
  - symptoms of discomfort from nose to navel should be evaluated for presence of CAD
- Less documented stenotic disease of major epicardial coronary arteries
- More likely to have unstable angina than MI
- Older women have higher incidence of complications
Angina in the Elderly

- Generalized symptoms
  - Dyspnea, diaphoresis, N&V, and syncope
  - Confusion
- Symptoms often attributed to the aging process
  - Importance of assessment with activity tolerance
- Cardiac and non cardiac co-morbidities complicate the diagnosis of ACS and increase the risk

- Don’t complain about chest pain
  - 37% of patients > 65
  - 42% of patients > 75 years
  - 75% of those > 85 years

Unique Presentation Features in Elderly

- Silent MIs account for 60% of MIs in those > 85 years of age
  - STEMI
    - < 65 years = 90% pain
    - > 85 years = 57% pain
  - NSTEMI
    - 44% not diagnosed on admission but diagnosis present on discharge
- Often co-existing heart failure masks the ACS diagnosis
  - HF with STEMI
    - < 65 years = 12%
    - > 85 years = 45%
- Age related risk versus disease specific risk
  - More suspicious with non diagnostic ECG and absence of chest pain
  - More suspicious in presence of another acute illness
- Diagnosis of “Other”
  - < 65 years = 5%
  - > 85 years = 24%
- Eighty-five percent of people who die from CHD are older than age 65.
Angina in Diabetics

- Autonomic dysfunction can affect symptoms experienced with angina
- Less likely to experience pain.
- 25% of all patients presenting with ACS are diabetic
- Have severe multi-vessel disease
- Have higher rates of complications
- Have a greater proportion of ulcerated plaques resulting in intracoronary thrombi

Acute MI Symptoms

- Symptoms occur spontaneously and are not relieved by rest or nitroglycerin
- Chest pressure or discomfort may be accompanied by nausea, vomiting, or diaphoresis
- Patient may have hemodynamic instability or cardiac arrest from ventricular fibrillation
- Acute MI patients have positive biomarkers and are classified as STEMI or NSTEMI based on ECG presentation
STEMI

< 25% of ACS patients
Complete occlusion of a vessel by a thrombus
Fibrin stable clot (red clot)
 Classified more specifically by the portion of the left ventricle suffering injury.
Mortality is greatest within the first 24 to 48 hours of symptom onset
TREATMENT FOCUS = REPERFUSION

NSTEMI

Higher mortality and morbidity than STEMI
Nationally under treated according to evidence based practice guidelines (Crusade Registry)
Pathophysiology often involves a platelet plug or white clot
Less stable clot
Opportunity for spontaneous reperfusion
Differentiated from unstable angina by troponin levels
TREATMENT FOCUS = ANTIPLATELET THERAPY
Causes of UA/NSTEMI*

- Thrombus or thromboembolism, usually arising on disrupted or eroded plaque
  - Occlusive thrombus, usually with collateral vessels
  - Subtotally occlusive thrombus on pre-existing plaque
  - Distal microvascular thromboembolism from plaque-associated thrombus
  - Thromboembolism from plaque erosion
- Non-plaque-associated coronary thromboembolism
- Dynamic obstruction (coronary spasm or vasoconstriction) of epicardial and/or microvascular vessels
- Progressive mechanical obstruction to coronary flow
- Coronary arterial inflammation
- Secondary UA
- Coronary artery dissection


Physiological Changes in the Elderly

- Decreased arterial compliance
- Increased cardiac afterload
- Diastolic dysfunction
- Co-morbid diseases
  - Aortic stenosis
  - Renal dysfunction
- Frailty
  - 25% of those > 85 years of age
- Inflammatory dysregulation
- Alterations in drug metabolism
Evaluation of Oxygen Supply and Demand

- Increase myocardial oxygen demand:
  - Hyperthermia
  - Hypertension
  - Tachycardia
  - Conditions producing over stimulation of the sympathetic nervous system (cocaine use, hyperthyroidism)
- Decrease myocardial oxygen delivery:
  - Anemia
  - Pulmonary disease.
- Increase myocardial oxygen demand and decrease myocardial oxygen supply:
  - Aortic stenosis
  - Hypertrophic cardiomyopathy

Elderly are at risk for secondary coronary events related to supply and demand imbalance.

Cardiac Risk Factors

- Non-Modifiable Risk Factors
  - Previous history
  - Family history
    - 1st degree relative (parents, siblings)
    - Men < 55; Women < 65
  - Age
  - Gender
  - Socioeconomic Factors and Ethnicity

- Modifiable Risk Factors
  - Smoking
  - Hypertension
  - Dyslipidemia
  - Diabetes
  - Obesity
  - Metabolic Syndrome
  - Inactivity
  - Alcohol

9 easily measured and potentially modifiable risk factors account for over 90% of the risk of an initial acute MI.
Other Pertinent History

- CAD
- Cerebral Vascular Disease
- Peripheral Vascular Disease

Cardiac Biomarkers

- Released into the blood when necrosis occurs as a result of membrane rupture of the myocytes
- Used in the evaluation of ACS
- Myoglobin
  - Rises the earliest
  - Within 2 hours after damage
  - Very sensitive, not specific
- CK (creatine kinase)
  - Enzyme present in the heart, brain, and skeletal muscle
  - Elevations are not specific to myocardial damage.
- CK-MB
  - More specific to the heart
  - Helpful in identifying more than minor amounts of myocardial damage
  - Rapidly rises in the presence of myocardial damage.
Cardiac Biomarkers

- Troponin I and T
  - Found only in cardiac muscle
  - Most sensitive indicator of myocardial damage
    - Capable of diagnosing small amounts of myocardial necrosis not measured by rises in CK-MB levels
  - Approximately 30% of patients with NSTEMI and normal CK-MB levels will test positive
  - Of equal sensitivity and specificity
  - Troponin remains elevated for a long period (late presentation)
  - Positive troponin + ECG changes of injury / ischemia = infarct
  - Non CAD causes of troponin elevation (sepsis, pulmonary emboli and chronic kidney disease)
  - Troponin I more specific in renal dysfunction

Cardiac Biomarker Summary

<table>
<thead>
<tr>
<th>Cardiac Biomarker</th>
<th>Specificity / Sensitivity</th>
<th>Rise</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoglobin</td>
<td>Sensitive but not specific</td>
<td>Within 2 hours</td>
<td>4 to 10 hours</td>
<td>&lt; 24 hours</td>
</tr>
<tr>
<td>CK-MB</td>
<td>Highly specific</td>
<td>4 to 6 hours</td>
<td>18 to 24 hours</td>
<td>2 to 3 days</td>
</tr>
<tr>
<td>Troponin I or T</td>
<td>Highly specific and sensitive</td>
<td>4 to 6 hours</td>
<td>18 to 24 hours</td>
<td>10 or more days</td>
</tr>
</tbody>
</table>
Timing of Release of Various Biomarkers After Acute Myocardial Infarction

Emerging Biomarkers for ACS and Risk Stratification

- BNPt
- C Reactive Protein
- Myeloperoxidase
- Ischemia modified albumin
STAT ECG Indications

- Chest pain or severe epigastric pain, non traumatic in origin, with components typical of myocardial ischemia or MI:
  - Central/subternal compression or crushing chest pain
  - Pressure, tightness, heaviness, cramping, burning, aching sensation
  - Unexplained indigestion, belching, epigastric pain
  - Radiating pain in neck, jaw, shoulders, back, or 1 or both arms
- Associated dyspnea
- Associated nausea/vomiting
- Associated diaphoresis

If non diagnostic:
- Repeat q 15 to 30 minutes
- Use ST segment monitoring
- Perform V7-V9

Source: ACC / AHA NSTEMI Guidelines 2007

Diagnostic Testing

- Non Invasive
  - ECG Evaluation
    - > age 85 delay of 7 minutes for initial ECG
    - 43% non diagnostic for age > 85
    - 23% for those < 65 years
  - Stress Testing
    - Chemical Stress Testing
  - CT / CTA
- Invasive: Cardiac Catheterization
  - IVUS
Stress Testing in Patients Presenting with Chest Pain

- Indicated when ECG and biomarkers are not diagnostic
- Should be done before discharge or within 72 hours as outpatient
- Precautionary pharmacotherapy for low risk patients being done on outpatient basis
  - ASA
  - SL NTG
  - Beta blockers

Stress Testing

- Exercise Stress Test with or without myocardial imaging
  - Nuclear Scanning
  - Echocardiogram
  - Future

- Patient conditions requiring myocardial imaging with stress testing due to lack of reliable ECG interpretation include:
  - Left bundle branch block
  - > 1 mm ST-segment depression at rest
  - Paced ventricular rhythm
  - Wolf-Parkinson-White syndrome
Exercise Stress Testing

- Treadmills or bicycles
- Able to exercise on a treadmill for 6 to 12 minutes
- While exercising
  - Myocardial oxygen demand increases
  - Coronary arteries dilate in response to increased demand
- If CAD
  - Coronary arteries not able to adequately dilate to meet the needs of the increased myocardial oxygen demand
  - Abnormalities occur on 12-lead ECG or imaging studies
- Consideration with beta-blockers
  - Hold beta-blockers approximately 48 hours prior to testing
  - May not hold if determining effectiveness
- Exercise stress testing is less sensitive in women than in men

Chemical Stress Testing

- Dipyridamole, adenosine, regadenoson
  - Done in conjunction with myocardial imaging
- Dipyridamole and adenosine
  - Non specific adenosine receptor blockers
  - Stimulation of these other receptors is what causes the unwanted side effect of atrioventricular (AV) block (A1 receptor) and bronchospasm (A2b and A3 receptors).
  - Contraindications:
    - Severe lung disease or if wheezing
- All 3 agents: Stimulation of Adenosine A2a receptor causes coronary vasodilation
  - Causes coronary microvascular dilatation similar to the coronary artery vasodilation that occurs with exercise
- Regadenoson is A2a selective
  - Another advantage over other two agents:
    - Rapid dosing
    - Non weight based
  - Antidote: Aminophylline for all 3 agents
  - Patients should be off aminophylline or related products prior to testing with these chemical agents
Chemical Stress Testing

- Dobutamine
  - High-dose dobutamine increases contractility and heart rate
  - Increasing myocardial oxygen demand
  - More closely mimics exercise stress testing
  - May be done with Echo instead of nuclear scan
  - Side effect: Tachyarrhythmias
  - Antidote: Beta blocker

Contraindications to Stress Testing

- Acute MI < 2 days old
- Acute myocarditis or pericarditis
- Acute pulmonary embolism
- Acute aortic dissection
- Symptomatic heart failure
- Severe aortic stenosis
- Symptomatic arrhythmias
- High-risk unstable angina
CT Angiography “FAST CT”

- 64 slice and beyond
- Detailed 3D Image
- Fast
- Coronary artery calcium scoring
  - Shows calcified plaque
  - Predictor of non-calcified plaque
- Coronary artery anatomy
- Myocardial function
- Need to lower heart rate
- Radiation exposure
- Good negative predictor

Cardiac Catheterization

- Indications
  - Patients with disabling angina despite medical treatment
  - Patients with high-risk criteria for coronary heart disease (CHD) on noninvasive testing
  - Patients who have survived sudden cardiac death
  - Patients with angina and clinical signs of CHD
  - Patients with low ejection fraction and ischemia on noninvasive testing
  - Patients with inadequate information obtained from noninvasive testing
STEMI Management

- **Reperfusion is number one treatment strategy**
- Primary Coronary Intervention (PCI) preferred treatment strategy if within 90 minutes
  - Goal: 90 minutes from 1st medical contact
- Fibrinolytics within 30 minutes of hospital presentation (or 30 minutes from EMS to fibrinolytics)

  Facilitated PCI with full dose fibrinolytics is not recommended.

  Rescue PCI may be done after failed fibrinolytics

Reperfusion Therapy

- Fibrinolytic Therapy
- Primary PCI

*Primary PCI has clear outcome advantage in those > 65 years:*
  - ✓ Mortality
  - ✓ Stroke
  - ✓ Intracranial Hemorrhage

Reperfusion has proven benefit up to age 85.
Special Considerations for Fibrinolytics

It is reasonable for high-risk* patients who receive fibrinolytic therapy as primary reperfusion therapy to be transferred as soon as possible where PCI can be performed either when needed or as a pharmacoinvasive strategy.

Consideration should be given to initiating an antithrombotic (anticoagulant plus antiplatelet) regimen before and during patient transfer to the catheterization laboratory.

Class II b (2009)

Fibrinolytics

Contraindications to the administration of fibrinolytics:
- Prior intracranial hemorrhage
- Known structural cerebral vascular lesion
- Malignant intracranial neoplasm
- Significant closed head injury within last 3 months
- Ischemic stroke within last 3 months (unless within last 3 hours)
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Symptoms greater than 24 hours old
- ST-segment depression (unless indicative of a true posterior wall MI)

Successful reperfusion with Fibrinolytics:
- Relief of presenting symptoms
- Reduction of at least 50% of initial ST-segment segment elevation on repeat ECG
- Hemodynamic and electrical stability
- Reperfusion arrhythmias such as accelerated idioventricular rhythm
- Early peaking of the CKMB
Fibrinolytics

**Issues specific to elderly:**
- Poorly controlled HTN
- Prior CVA
- Dementia
- Chronic Anticoagulation
- Patient Preference

**Reasons for Delayed or Missed Reperfusion Therapy**

- Missed performance of ECG due to atypical symptoms
- Unrecognized unequivocal ECG
- Delay in diagnosis of subtle ECG
- Failure to perform serial ECGs
- Age > 75
- Female
- No chest pain
- Heart failure
- Delayed onset of presentation
ECG Recognition Challenges in the Elderly

- ST elevation recognition < 65 years = 96%
- ST elevation recognition > 85 years = 70%

- Left bundle branch block
  - > 85 years = 34%
  - < 65 years = 5%

STEMI Systems of Care

- Each community should develop a STEMI system of care consistent with minimum standards of AHA’s Mission: Lifeline
  - ongoing multidisciplinary team meetings (including EMS and referring and receiving hospitals) to evaluate outcomes and quality improvement data;
  - a process for prehospital identification and activation;
  - destination protocols for STEMI receiving centers;
  - transfer protocols for patients who arrive at STEMI referral centers who are primary PCI candidates, are ineligible for fibrinolytic drugs, and/or are in cardiogenic shock.
Options for Transport of Patients With STEMI and Initial Reperfusion Treatment

EMS Transport

Onset of symptoms of STEMI

9-1-1 Call fast

9-1-1 EMS Dispatch

EMS on-scene
- Encourage 12-lead ECGs.
- Consider prehospital fibrinolytic if capable and EMS-to-needle within 30 min.

GOALS

EMS Transport

EMS Prehospital fibrinolysis
EMS transport
EMS-to-needle within 30 min.
EMS-to-balloon within 90 min.
Patient self-transport
Hospital door-to-balloon within 60 min.

Not PCI capable

PCI capable

EMT Trauma Plan

Inter-Hospital Transfer

Hospital fibrinolysis: Door-to-Needle within 30 min.

Golden Hour = first 60 min.
Total ischemic time: within 120 min.

Antman EM, et al. J Am Coll Cardiol 2008. Published ahead of print on December 10, 2007. Available at http://content.onlinejacc.org/cgi/content/full/j.jacc.2007.10.001. Figure 1.

Medical Management of STEMI

- ASA
- Clopidogrel (with or without reperfusion)
- Oxygen
- NTG
- MS (Class I)
- D/C NSAIDS
- Beta-blockers (within 24 hours)
- ACE Inhibitors (within 24 hours with impaired EF, HTN, diabetes or chronic kidney disease)
- Anticoagulants (related to reperfusion strategy)
- Intravenous insulin may be indicated in first 24 to 48 hours after STEMI to tightly control blood sugars.

Reperfusion is primary management strategy.
New 2009 Recommendation for Glucose Control

- It is reasonable to use an insulin-based regimen to achieve and maintain glucose levels less than 180 mg/dL while avoiding hypoglycemia* for patients with STEMI with either a complicated or uncomplicated course.

Treatment of Non STEMI / Unstable Angina: New Guidelines

- **Attacking Platelet is number one treatment strategy**
  - Dual antiplatelet therapy for invasive strategies in medium to high risk patients
    - ASA (and one of following)
    - Clopidogrel (loading)
    - Prasugrel (loading)
    - GP IIb / IIIa Inhibitors (eptifibatide or tirofiban)

- Antiplatelet therapy also in conservative treatment
  - Prasugrel not unless PCI is planned
  - Abciximab not unless PCI is planned
Treatment of Non STEMI / Unstable Angina: New Guidelines

- **Anticoagulation options in NSTEMI:**
  - Unfractionated heparin
  - LMWH (enoxaparin)
  - Factor Xa inhibitor (fondaparinux)
  - Direct thrombin inhibitor (bivalrudin)

- **Duration of anticoagulation**
  - Enoxaparin or fonaparinux for duration (in conservative) or up to 8 days
  - UFH for 48 hours in conservative

**Discussion Points**

- **Class III recommendations**
  - No Abciximab if no PCI Planned
  - No GPIIb/IIIa in low risk patient on dual antiplatelet
    No prasugrel if previous stroke or TiA

- **Oral antiplatelets**
  - Clopidogrel for 1 month preferably a year after conservative treatment
  - Ischemic protection versus bleeding risk
Treatment of NSTEMI / UA: New Guidelines

- **ASA**
- **Oxygen** (1st 6 hours)
- **NTG**
  - IV in first 48 hours for persistent ischemia, HTN, HF
  - Should not interfere with mortality reducing beta blockers or ace inhibitors
- **MS** (if NTG unsuccessful and other anti ischemic drugs on board)
- **Beta Blockers** (within 24 hours)
  - Start PO when hemodynamically stable
  - May use IV if hypertensive
- **ACE Inhibitors** (within 24 hours)
  - In select patients — pulmonary congestion or LVEF ≤ 40% — may also be used in other patients
- **DC — NSAIDS**

Medical Supportive Therapy:
Similar to STEMI

Early Invasive Option in UA / NSTEMI

- Not waiting for failed medical treatment
- Not waiting for + noninvasive test
- Angiography with intent of revascularization
- Better outcomes with GP IIb/IIIa inhibitors
- Excluded: very frail elderly, severe hepatic, pulmonary or renal failure (CKD IV or V), active or inoperable cancer

Overall reduction in mortality and increased quality of life.

- Initial conservative (selective invasive) is always an alternative option
- Initial conservative strategy recommended in low risk women
Early Invasive Indications

- Refractory angina or hemodynamic or electrical instability
  - Without serious co-morbidities or contraindications to such procedures
  - May be reasonable in patients with chronic renal insufficiency
- Initially stabilized with high risk for clinical events

High Risk Features in UA / NSTEMI

- Recurrent angina / ischemia
  - Rest or low level activity with medical treatment
- Troponin +
- New or presumed new ST depression
- S&S HF or worsening mitral regurgitation
- High risk findings on noninvasive testing
  - EF < 35%, large anterior perfusion defect, multiple perfusion defects
- Hemodynamic instability
- Sustained VT
- PCI within 6 months
- Prior CABG
- Reduced LV Function
- High risk TIMI or GRACE Score

**Elderly**: cancer, renal insufficiency, lung disease, anemia, and heart failure are common co-morbid conditions
Risk Assessment in UA / NSTEMI

- **TIMI Risk Score**
  - Age > 65
  - 3 or > risk factors for CAD
  - Prior 50% or > stenosis
  - ST deviation on ECG
  - 2 or > anginal events in previous 24 hours
  - Use of ASA in prior 7 days
  - Elevated cardiac biomarkers

- **GRACE**
  - Older age
  - Killip class
  - Systolic BP
  - Cardiac arrest during presentation
  - Serum creatinine
  - Positive initial cardiac markers
  - HR

Markers of Risk: Specific to Elderly

- Mobility and function
  - Activities of daily living
  - Strength
- Physiological reserves
  - Frailty
- Poor Nutrition Status
  - Albumin
  - Weight loss
- Cognitive Impairment
- Hearing Alterations
- Vision Alterations
- Isolation
- Resources / Education
- Socioeconomic
Risk of In Hospital Death Specific to Age

- < 65 years: 1 in 100
- > 85 years: 1 in 10

1 year mortality rate:
- 75 years: 1 in 5
- 85 years: 1 in 4

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Short-Term Risk of Death/Nonfatal MI in Patients With UA/NSTEMI

<table>
<thead>
<tr>
<th>Feature</th>
<th>High Risk</th>
<th>Intermediate Risk</th>
<th>Low Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≥ 1 of the features below must be present:</td>
<td>No high-risk features, but must have 1 of the following:</td>
<td>No high- or intermediate-risk features but may have any features below:</td>
</tr>
<tr>
<td>History</td>
<td>Accelerating tempo of ischemic sx in preceding 48 h</td>
<td>Prior MI, peripheral or cerebrovascular disease, or CABG; prior ASA use</td>
<td></td>
</tr>
</tbody>
</table>
| Character of pain   | Prolonged ongoing (> 20 min) rest pain | • Prolonged (> 20 min) rest angina, now resolved, w/ moderate/high likelihood of CAD  
• Rest angina (> 20 min) or relieved with rest or sublingual NTG  
• Nocturnal angina  
• New-onset or progressive CCS class III/IV angina in past 2 wks w/o prolonged (> 20 min) rest pain but with intermediate/high likelihood of CAD | • † Angina frequency, severity or duration  
• Angina provoked at lower threshold  
• New onset angina with onset 2 wks to 2 mos prior to presentation |
### Short-Term Risk of Death/Nonfatal MI in Patients With UA/NSTEMI, Continued

<table>
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<tr>
<th>Feature</th>
<th>High risk</th>
<th>Intermediate risk</th>
<th>Low risk</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical findings</strong></td>
<td>• Pulmonary edema, most likely due to ischemia</td>
<td>Age &gt; 70 y</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• New/worsening MR murmur</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>• S₃ or new/worsening rales</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Hypotension, bradycardia, tachycardia</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Age &gt; 75 y</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ECG</strong></td>
<td>• Angina @ rest with transient ST-segment changes &gt; 0.5 mm</td>
<td>• T-wave changes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• BBB, new/presumed new</td>
<td>• Pathological Q-waves/resting ST-depression &lt; 1 mm in multiple lead groups (anterior, inferior, lateral)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Sustained VT</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cardiac markers</strong></td>
<td>↑ Cardiac TnT, TnI, or CK-MB (e.g., TnT/TnI &gt; 0.1 ng/mL)</td>
<td>Slightly ↑ cardiac TnT, TnI, or CK-MB (e.g., TnT &gt; 0.01, but &lt; 0.1 ng/mL)</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Estimation of the short-term risk of death and nonfatal cardiac ischemic events in UA/NSTEMI is a complex multivariable problem that cannot be fully specified in a table such as this; this table is meant to offer general guidance & illustration rather than rigid algorithms. Braunwald E, et al. AHCPR Publication No. 94-0602:1–154. Anderson JL, et al. J Am Coll Cardiol 2007;50:e1–e157, Table 7.

### Beta Blockers Considerations

- **Oral Beta Blockers**
  - Within 24 hours
- **IV Beta Blockers**
  - Reasonable in patients who are hypertensive
  - May be harmful in patients with high risk for cardiogenic shock
- **Beta blockers have greater benefit in elderly for reduction of future MI and death than in younger patient populations**
- **Contraindications**
  - Signs of HF
  - Low cardiac output state
  - Increased risk for cardiogenic shock
    - Age > 70 years is a risk factor
  - Relative contraindications
    - PR > .24 seconds
    - 2nd or 3rd degree block
    - Active asthma
    - Reactive airway disease
Nitrate Considerations

Contraindications
- Systolic BP < 90 mm Hg or ≤ 30 mm Hg below baseline
- Bradycardia < 50 BPM
- Tachycardia > 100 BPM (in absence of clinical HF)
- Right ventricular infarct
- Within 24 hours of sildenafil
- Within 48 hours of tadalafil

Other Medication Considerations
- Hold ace inhibitors for BP < 100 mm Hg systolic or < 30 mm Hg below baseline
- No IV ace inhibitor within 24 hours due to risk of hypotension
- No immediate release dihydropyridine calcium channel blockers without beta blockade on board
- NSAIDS (except for ASA), whether nonselective or COX-2–selective agents increase risk of mortality, reinfarction, hypertension, HF, and myocardial rupture
- Proton Pump Inhibitors should be prescribed to patients at risk for GI bleed – However: Caution with Clopidogrel
Stepped Care Approach To Pharmacologic Therapy for Musculoskeletal Symptoms with Known Cardiovascular Disease or Risk Factors for Ischemic Heart Disease

- Acetaminophen, ASA, tramadol, narcotic analgesics (short term)

- Nonacetylated salicylates

- Non COX-2 selective NSAIDs

- NSAIDs with some COX-2 activity

- COX-2 Selective NSAIDs

Select patients at low risk of thrombotic events
Prescribe lowest dose required to control symptoms
Add ASA 81 mg and PPI to patients at increased risk of thrombotic events *

* Addition of ASA may not be sufficient protection against thrombotic events


New Antiplatelet

- Ticagrelor (BRILINTA)
  - oral, reversible, direct-acting adenosine diphosphate receptor P2Y12 inhibitor
  - more rapid onset and more pronounced platelet inhibition than clopidogrel.
  - 2 x daily dosing
  - Maintenance doses of aspirin above 100 mg decreases the effectiveness of Ticagrelor.
  - Metabolized by CYP3A4/5.
  - Contraindicated in severe hepatic failure.
  - Hold 5 days prior to CABG
  - Most common SE: Dyspnea (14%), Bleeding (11.6%), and ventricular pauses (6.0%) - dyspnea and ventricular pauses were dose related
More on Ticagrelor

- **PLATO**: Multicenter, double-blind, randomized trial
  - Ticagrelor (180-mg loading dose, 90 mg twice daily thereafter) to clopidogrel (300-to-600-mg loading dose, 75 mg daily thereafter)
  - 18,624 patients admitted to the hospital with an acute coronary syndrome, (with or without ST-segment elevation).
  - Primary end point — a composite of death from vascular causes, myocardial infarction, or stroke (9.8% to 11.7%; \( p < 0.001 \))
  - No significant difference in rates of major bleeding
  - Ticagrelor was associated with a higher rate of major bleeding not related to coronary-artery bypass grafting

- **Indications**
  - Reduce rate of thrombotic cardiovascular events in ACS.
  - Reduce rate of stent thrombosis post PCI.

Long Term Management of ACS

*Medications to improve prognosis*

- **Aspirin**
  - ASA benefits > in those \( \geq 65 \) years
  - Generally no dose adjusting

- **Clopidogrel / Prasugrel**
  - Caution age > 75 with prasugrel
  - Higher risk of bleeding dual antiplatelet therapy
    - No elderly subgroup data for clopidogrel

- **Beta-blockers**

- **ACE inhibitors** (in select patients)
  - ARBs (may be used with ACE-I in systolic dysfunction)
  - Aldactone (EF \( \leq 40 \) with HF or diabetes)

- **Lipid-lowering drugs** (statins)
  - Have greater benefit in elderly for reduction of future MI and death than in younger patient populations

* Beta blockers and ACE inhibitors impact long term ventricular remodeling
SL NTG Instruction

- No more than 1 dose of SL NTG
  - If chest discomfort is unimproved or is worsening 5 min after 1 NTG call 9-1-1 immediately before taking additional NTG.
  - May take additional NTG while waiting EMS.
  - Chew ASA while waiting EMS.

- In chronic stable angina if symptoms are significantly improved by 1 dose of NTG may repeat NTG every 5 min for a maximum of 3 doses and call 9-1-1 if symptoms have not resolved completely.

Medical Therapy Issues in the Elderly

- Altered responses and vulnerability to drugs with:
  - Hypotensive action (nitrates, calcium blockers)
  - Cerebral effects (beta blockers)
- Caution with renally cleared drugs
- START LOW and GO SLOW!!
## Treatment of Stable Angina

- Medical Treatment
  - Lipid-lowering therapy
  - Antiplatelet therapy
  - Antianginal therapy
- Revascularization (* if criteria are met)
  - Primary Coronary Intervention
  - Coronary Artery Bypass Graft
- Aggressive risk factor modification

## Secondary Prevention: ACS and Stable CAD

- Smoking cessation
- Reduction of hyperlipidemia
  - LDL < 100 mg/dL or < 70 mg/dL (optimal)
- Hypertension control
  - <130/80 for kidney disease or diabetes
- Diabetes control Hb A1c < 7
- Physical activity minimum of 5 days / per week
  - 7 days recommended
- BMI 18.5 – 24.9 kg/m²
- Phase II Cardiac Rehab
- Influenza vaccine
Key Nursing Care Considerations

- Assess response to beta-blocker therapy
  - HR / BP
  - Arrhythmia control
  - Need for higher / lower dose
- Reassess oxygen saturation after 6 hours and discontinue O₂ if saturation is more than 90%
- Assess for complications related to specific type of MI
  - Assess heart sounds for new holosystolic murmurs
    - Risk for myocardial rupture
  - Observe for signs of left ventricular dysfunction, including hypotension or clinical signs of heart failure.
  - Monitor ECG for conduction disturbances and arrhythmias
  - Assess for presence of RV infarct

Key Nursing Care Considerations

- Restrict activity for at least the first 12 hours, and then begin Phase I Cardiac Rehabilitation
  - Referral to Phase II Cardiac Rehabilitation
- Utilize cardiac monitoring
  - ST-segment monitoring
  - Uninterrupted monitoring for first 24-48 hours
- Focus on holistic approach to anxiety reduction
  - Include the family. Family visits do not have a negative impact on vital signs or cardiac rhythm
- Address addiction to nicotine
  - Consideration for nicotine withdrawal
  - Specific smoking cessation plan
Complications of MI

- Hemodynamic Alterations
- Ventricular Arrhythmias
- Atrial Arrhythmias
- Pericarditis
- Ventricular Aneurysms
- Mechanical Complications
  - Myocardial Rupture (free wall or VSD)
  - Papillary Muscle Rupture
- Long Term: Ventricular Remodeling

Complications Specific to Elderly

- STEMI 30 day mortality
  - < 65 years = 3%
  - > 85 years = 30%
  - Death related to electrical and mechanical (free wall or papillary muscle rupture) catastrophes
- Reinfarction
- HF development
- Need for transfusion
  - PCI – Elderly women and groin complications
Left Ventricular Remodeling Following Myocardial Infarction

Acute Infarction, hours

Infarct Expansion, hours to days

Global Remodeling, days to months

Progressive adverse LV remodeling

Acute MI → At one year

Alteration in Myocyte architecture, Size, Shape and Contractility

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Ventricular Aneurysm

- Persistent ST elevation after AMI (anterior)
- Anatomic LV aneurysm
  - Myocardial thinning and bulging
- Use of echo in the reperfusion decision
- Risk of fibrinolytics with ventricular aneurysm
- Embolization of thrombus

- Most common in V1-V3
- Usually less than 3 mm elevation
- Relatively unchanged from previous ECGs
- Q waves are deep and well formed
  - QS pattern in V1-V3 or very minimal r
  - QR pattern common in inferior aneurysm / RBBB

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Myocardial Rupture

- Incidence
  - 10% MI deaths

- Definition
  - Myocardial leakage – hemipericardium – tamponade
  - Perceived sudden; often slow tear

- Associated Factors
  - Late fibrinolytics
  - Delayed hospital admission

- Free wall rupture = tamponade
- Septal involvement = VSD
- Posterior wall = Risk for papillary muscle rupture
Myocardial Rupture

- Post-infarction regional pericarditis precedes rupture (94% of the time)

T Wave Patterns in Post-infarction Regional Pericarditis

- Persistently positive T waves 48 hours after an MI
- Premature reversal of T wave inversion to positive
- ST segment reelevation
ST Segment Monitoring

A SUCCESS Story!!

The next 2 slides show the following:
1. Admission ECG for a patient with an anteroseptal / lateral wall STEMI.
2. ECG post intervention for same patient.
   1. Note: The T waves have not yet inverted post intervention. Ideally T waves will begin to invert after an intervention showing evidence of reperfusion.

REMEMBER: T wave must invert within 48-72 hours after a STEMI (the sooner the better). Failure of T waves to invert after a STEMI is indicative of post infarction regional pericarditis and the patient is at higher risk for myocardial rupture.
The strip below assessing ST segments in V3 was done 48 hours post STEMI (same patient as previous 2 ECGs). The failure of the T waves to invert is indicative of post infarction regional pericarditis with increased risk of myocardial rupture. The patient was hypotensive, which raises the concern for cardiac tamponade as the etiology of the hypotension. This assessment finding was communicated to the cardiologist.

The patient’s echocardiogram showed a large pericardial effusion and the patient subsequently underwent a surgical pericardial window.
Cardiac Tamponade

- Clinical syndrome caused by accumulation of fluid in the pericardial space
- Same causes as pericarditis / pericardial effusion
- Increase capillary permeability due to inflammation may cause fluid leak into pericardial space
  - >120cc can cause tamponade if rapid
  - 2 Liters may not cause tamponade if slow
- Results in reduction in ventricular filling and ultimately hemodynamic compromise
- Differentiation between pericardial effusion and tamponade is hemodynamic status.

Practice EKG 1of 3
Practice EKG 2 of 3

Practice EKG 3 of 3
Papillary Muscle Rupture

Final Quote:
Our grand business in life
is not to see what lies
dimly at a distance,
but to do what lies clearly at hand.

Thomas Carlyle (1795-1881)