Acute Coronary Syndrome
Time is Muscle
Diagnosis & Treatment

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Cardiac Clinical Nurse Specialist

Evidenced based Practice -- Cardiac

- ACC/AHA: American College of Cardiology and American Heart Association
  - 2007 Focused Update Guidelines for Management of STEMI
  - 2007 Guidelines for Management of Patients with Unstable Angina/Non-ST-Elevation Myocardial Infarction
  - www.acc.org

Acute Coronary Syndrome (ACS)
- Umbrella term for a group of thrombotic coronary artery disease conditions that cause myocardial ischemia
- These syndromes represent progression of occlusion in the involved coronary artery
  - STEMI (ST segment Elevation Myocardial Infarction)
  - NSTEMI (Non-ST Segment Elevation Myocardial Infarction)
  - Unstable Angina

Applying Classification of Recommendations and Level of Evidence

<table>
<thead>
<tr>
<th>Class I</th>
<th>Procedure/Treatment SHOULD be performed/administered</th>
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<tr>
<td>Benefit &gt;&gt; Risk</td>
<td>Additional studies with focused objectives needed</td>
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<td>IT IS REASONABLE to perform procedure/administer treatment</td>
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Level A: Evidence from metaanalysis of randomized trials or observational studies with at least moderate evidence for benefit and low burden of harm
Level B: Evidence from metaanalysis of randomized trials or observational studies with at least moderate evidence for benefit and low burden of harm
Level C: Evidence from single randomized trials or nonrandomized studies

Cardiac Anatomy

Atherosclerosis
Cascade effects of atherosclerotic plaque rupture

- Platelet aggregation
- Fibrin accumulation
- Thrombus formation
- Bleeding into the plaque
- Vasospasm

Other Causes of STEMI
- Coronary vessel spasm
- Coronary emboli
- Vasculitis
- Severe chest trauma

Myocardial Ischemia Leads to Myocardial Necrosis

Heart Attack Signs & Symptoms for Males
- Chest Pain
- Pain radiating down arms
- Jaw Pain
- Sweating
- Nausea

Heart Attack Signs & Symptoms for Women
- “Atypical” Chest Pain
- Shortness of Breath/ Trouble Breathing
- Tingling of Fingers
- Extreme Fatigue
- Heartburn / Nausea
- Sweating
- Dizziness
- Feeling of Apprehension or Impending Doom
**Muscle Ejection Fraction**

Ejection Fraction is 
**Quality of Life**

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**CRUSADING towards a GOAL**

**Door to EKG**

10 minutes or less

(Class 1: Level C)

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**EKG Changes with MI**

**Ischemia < 20 Minutes**

Lack of oxygen to the myocardial muscle

- peaked T waves
- inverted T waves
- ST segment depression

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**EKG Changes with MI**

**Injury 20 - 40 minutes**

- When the period of ischemia is prolong more than a few minutes.
- Ischemic areas of the heart become damaged (injured)
  - ST segment elevation

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**Measurement of ST-Segment Deviation**

**Review of Normal QRS complex**

- Q wave is the first negative deflection after the p wave
- R wave is the first positive deflection after the p wave
- S wave is the second negative deflection after the R wave
EKG Changes with MI

Infarction > 1 - 2 hours
- Abnormal Q waves
  - > 1/3 the height of R wave in that lead
  - > 0.03 ms wide

Normal Q wave
1st downward deflection of QRS

Evolution of AMI

EM #1 December 13 at 1701

Hyperacute Phase of MI
- Occurs within minutes to first hour of chest pain
- Very tall ST segment
- Tall peaked symmetrical T waves

EM #2 December 13 at 1823
EM #3 December 14 at 0630
EM #4 December 15 0600
Acute Phase of MI
- Occurs in the first 24 hours
- ST segment elevation returns to baseline within 24 hours
- T wave inversion occurs in 24 - 48 hours and stays for two weeks
- Q wave develops after 48 hours
- R wave decreases

Evolving Phase of MI
- Occurs in the first week
- ST segment returns to normal
- T wave is deeper and inverted
- Q wave deepens
- R wave decreases more

Resolving Phase of MI
- Occurs in second week
- ST segment returns to normal
- T wave stops inverting and stays that way for 2 weeks and then resolves and will be low voltage
- Q wave stops deepening and stays due to dead tissue
- R wave stops decreasing and stays due to dead tissue

Positive EKG
- ST elevation > 0.1mV (1 mm) in at least 2 contiguous precordial leads or at least 2 adjacent limb leads (STEMI) (Class 1, Level A)
- Transient ST Elevation > 0.5 mm
- ST depression > 0.5 mm (NSTEMI)
- T wave inversion > 0.2 mV (2 mm)
- New LBBB (Class 1, Level A)

BBB = QRS > 0.12ms
- LBBB = QRS > 0.12, Negative QRS in V1 (carrot)
- RBBB = QRS > 0.12; Positive QRS in V1 (rabbit ears)

We Can STOP Heart Attacks!
- Goal: OPEN THE ARTERY!
**Time Is Muscle**

Muscle is Ejection Fraction

Ejection Fraction is Quality of Life

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**PreHospital: Routine Cardiac Protocol**

**Priorities of care**

- Assessing and securing ABCs
- Determining the quality and severity of the patient’s distress
- Identifying contributing factors of the event
- Obtaining a medical history
- **Timely transportation** to the emergency department is an important factor in patient outcomes

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**Prehospital: Routine Cardiac Protocol**

**BLS Care**

- Oxygen 15 L/min via non-rebreather mask.
- EKG monitor
- Aspirin -- 324 mg po (4 tablets of 81 mg chewable aspirin)
- Nitroglycerin -- 0.4 mg SL (1 tablet or 1 metered spray dose sublingually) May repeat every 3 – 5 minutes to a total of 3 doses if systolic BP remains > 100 mmHg
- Initiate ALS intercept if necessary & transport ASAP
- Contact receiving hospital ASAP

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**Assessment Questions (OPQRST)**

- Onset
- Provocation
- Quality
- Radiation
- Severity
- Time

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**Target:**

**Door to Balloon < 90 minutes**

(Class 1, Level A)

**Door to Needle < 30 minutes**

(Class 1, Level B)

ACC/AHA 2007 Focused Update
Guidelines for Management of STEMI

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**Goals**

- At least 75% of STEMI patients have door to balloon within 90 minutes of presentation. 2007 STEMI guidelines
- The overarching goal is to keep total ischemic time within 120 minutes (ideally within 60 minutes) from symptom onset to initiation of reperfusion treatment.

Source: 2007 ACC/AHA STEMI Guidelines
Open the Artery

Goal: Have artery open < 90 minutes after arrive at ED

Reaching this target….

• How do we do it?

Treatment as getting ready for PCI

• Oxygen (Class 1, Level B)
• ASA 162 mg if not given in ambulance (Class I, Level C)
• Betablocker: Metoprolol 5 mg IV q 5 min x 3 doses.
  Hold if SBP < 90, notify MD if held. (Class I, Level A)
• Nitroglycerin: NTG 0.4 mg SL x 3 or IV NTG (Class I, Level C)
• Morphine 2 - 4 Mg IV q 5 - 15 min for pain relief (Class I, Level C)

What if PCI is not available?

• Treat with fibrinolytic therapy within 30 minutes of hospital presentation
  – If unable undergo PCI within 90 minutes of first medical contact
  – unless fibrinolytic therapy is contraindicated.

Door to Cath Lab Door < 30 minutes

• If unable to occur, TPA or TNKase needs to be considered.

Target:

Door to Balloon < 90 minutes
(Class 1, Level A)

or

Door to Needle < 30 minutes
(Class 1, Level B)

ACC/AHA 2007 Focused Update Guidelines for Management of STEMI
Absolute Contraindications for Fibrinolysis in STEMI

- Any prior intracranial hemorrhage
- Known structural cerebral vascular lesion (e.g., arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head or facial trauma within 3 months

Findings

- Primary PTCA better than thrombolytic therapy at reducing
  - Short term death (7% vs. 9%) p=0.0002
  - Non-fatal reinfarction (3% vs. 7%) p<0.0001
  - Stroke/ICH (1% vs. 2%) p<0.0001
  - 43% relative reduction of combined endpoints
- PTCA superior during long-term follow-up, independent of thrombolytic used and whether or not patient was transferred for primary PTCA.
  (Keeley, Boura, & Grines, 2003)

Take away message:

- Primary PCI is superior to fibrinolysis
  - In high volume PCI centers
  - If performed in a timely manner:
    - <120 min, possibly longer

Time is Muscle!
And Mortality!

- Each 30 minute delay in reperfusion with PCI increases 1 yr mortality 7.5%
- Door to balloon <60 min, 1% 30 day mortality; Door to balloon >90 min, 6.4% mortality


The artery is open….
Now what ???

Reperfusion characteristics

- Relief of symptoms
- Maintenance or restoration of hemodynamic or electrical stability
- Reduction of at least 50% of the initial ST segment elevation injury pattern on a follow-up EKG 60 - 90 minutes after initiation of therapy (Class IIa: Level B)
Anticoagulant Therapy post PCI or Fibrinolysis

- Anticoagulant regimens with established efficacy include:
  - UFH (Unfractionated Heparin) (LOE: C)
  - Enoxaparin (LOE:A)
  - Fondaparinux (LOE:B)

- Clopidogrel 75 mg per day orally and continued for at least 14 days (Class 1, A)

Source: 2007 ACC/AHA STEMI Guidelines

Troponin I
Normal: 0 – 1.2 ng/ml

- Elevates = 4 – 6 hours
- Peak = 24 hours
- Returns to baseline = 6 – 7 days
- Most sensitive & specific serum marker to detect myocardial injury
- Remains elevated up to 5 or more days so can diagnose MI for several days after the event.

Creatine Kinase (CK)
Normal: 12 – 108 ng/ml
CK MB Normal: 0 – 5 ng/ml

- Elevates = 4- 6 hours
- Peak = 24 hours
- Returns to baseline = 3 days
- Somewhat less specific for myocardial injury than troponin
- More sensitive to recurrence of a second or third event
- Assists in assessment of reperfusion following thrombolytic therapy.

Myoglobin
Normal: 0 – 86 ng/ml

- Elevates = 1 – 2 hours
- Peak = 6 hours
- Returns to baseline = 24 hours
- Fast biomarker to rule out MI
- If it does not elevate, there is no cardiac muscle damage.
- If it does elevate, there is muscle damage somewhere in the body. It may or may not be cardiac muscle damage
- Used in clients with normal 12 Lead EKG and nonspecific chest pain. If myoglobin is normal can send client home and know it is not a cardiac event.

49 y/o female presents with chest pain

<table>
<thead>
<tr>
<th>Time</th>
<th>Troponin I</th>
<th>CK</th>
<th>CKMB</th>
<th>Myoglobin</th>
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<tbody>
<tr>
<td>1625</td>
<td>&lt;0.35</td>
<td>55</td>
<td>2.2</td>
<td>133.8 †</td>
</tr>
<tr>
<td>2100</td>
<td>31.8 †</td>
<td>767 †</td>
<td>47.2 †</td>
<td>368 †</td>
</tr>
<tr>
<td>2230</td>
<td>42.4 †</td>
<td>853 †</td>
<td>90.5 †</td>
<td>-----</td>
</tr>
<tr>
<td>0400 - 12 hours later</td>
<td>63.0 †</td>
<td>1091 †</td>
<td>104 †</td>
<td>-----</td>
</tr>
<tr>
<td>0400 - 36 hours later</td>
<td>14.9 †</td>
<td>277 †</td>
<td>12.5 †</td>
<td>-----</td>
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81 y/o female presents with c/o of chest discomfort over the weekend

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<thead>
<tr>
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<th>Troponin I</th>
<th>CK</th>
<th>CKMB</th>
<th>Myoglobin</th>
</tr>
</thead>
<tbody>
<tr>
<td>1700</td>
<td>6.9 †</td>
<td>198</td>
<td>7.2 (s)</td>
<td>66.1</td>
</tr>
<tr>
<td>1800</td>
<td>5.7 †</td>
<td>163</td>
<td>6.8 (s)</td>
<td>61.7</td>
</tr>
<tr>
<td>2330</td>
<td>6.4 †</td>
<td>146</td>
<td>6.0 (s)</td>
<td>-----</td>
</tr>
<tr>
<td>0500 - next day</td>
<td>6.0 †</td>
<td>120</td>
<td>5.4 (s)</td>
<td>-----</td>
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AMI probably > 24 hours ago
NSTEMI
Non ST Segment Elevation MI
- No ST segment Elevation
- ST segment depression
- New LBBB

STEMI vs NSTEMI
Both elevated Troponins

STEMI
PCI < 90 minutes

NSTEMI
PCI within 24-48 hours
Inversion of the T wave Depression of ST segment

If the initial ECG is not diagnostic but the patient remains symptomatic and there is high clinical suspicion for ACS
- Do serial ECGs, initially at 15- to 30-min intervals, to detect the potential for development of ST-segment elevation or depression. (Class I, A)

12-Lead ECG Findings

1. ST-segment elevation or new LBBB strongly suspicious for injury
2. ST-segment depression/dynamic T wave inversion; strongly suspicious for ischemia
3. Normal or nondiagnostic or ECG; chest pain strongly suspicious for ischemia

Reperfusion
Lytics—PCI
Antithrombin Therapy
Risk Stratification
TIMI Risk Score of NSTEMI/UA

<table>
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<tr>
<th>Historical</th>
<th>Points</th>
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<tr>
<td>• Age &gt; 65</td>
<td>1</td>
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<tr>
<td>• 3 or &gt; cardiac risk factors</td>
<td>1</td>
</tr>
<tr>
<td>(age, male, family history, hyperlipidemia</td>
<td></td>
</tr>
<tr>
<td>diabetes, smoking, hypertension, obesity</td>
<td></td>
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<tr>
<td>• Documented prior coronary artery stenosis &gt; 50 %</td>
<td>1</td>
</tr>
<tr>
<td>• Use of ASA in last 24 hours</td>
<td>1</td>
</tr>
<tr>
<td>• 2 or &gt; anginal events in past 24 hr</td>
<td>1</td>
</tr>
<tr>
<td>• Elevated Cardiac Markers</td>
<td>1</td>
</tr>
<tr>
<td>• ST segment deviation</td>
<td>1</td>
</tr>
<tr>
<td>• Transient elevations or persistent depression</td>
<td>1</td>
</tr>
<tr>
<td>• Risk score = total points (0 - 7)</td>
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High-Risk for death or MI.

Must include at least one of the following features to be present:

• > 20 minutes of rest pain
• ST depression (> 0.5mm)
• Markedly elevated cardiac markers
• Transient ST elevation (> 0.5mm)
• Accelerating tempo of ischemic symptoms in preceding 48 hours
• Signs of CHF (rales or new S3)
• New murmur
• Hypotension (SBP < 100)
• Tachycardia (pulse > 100)
• Bradycardia (pulse < 60)
• Note if medication induced
• Sustained ventricular arrhythmias
• Age > 75 yrs
• TIMI risk score > 3

Treatment for NSTEMI/ACS – patient with high-risk features:

Strategy A: Early Invasive Protocol

Intermediate/Moderate-risk for death or MI:

Must demonstrate no high-risk features but must have one of the following:

• > 10 minutes of rest pain now resolved, with moderate to high likelihood of CAD
• T-wave inversion ≥ 0.2mV
• Slightly elevated cardiac markers (Troponin I > 0.1 mg/ml)
• Prior MI, CABG or PCI
• Age > 70 yrs
• TIMI risk score > 3

Treatment for Unstable Angina patient with moderate-risk features:

- Strategy A: Early Invasive Protocol
- Strategy B: Early Conservative Protocol

Low risk for death or MI

Must have no high or intermediate risk features but may have any of the following features:

• New onset of chest pain or progressing angina without prolonged (> 10 minutes) of rest pain
• Normal or unchanged ECG
• Normal cardiac markers

Treatment for Chest pain patient with low-risk features:

- Strategy B: Early Conservative Protocol

Other Risk Stratifications

- GRACE risk score
- PURSUIT risk model

Call elevated Troponins

• Troponin I > 0.1 mg/ml makes a person at intermediate/moderate-risk for death or MI
• Elevates in 4 hours -- stays elevated for days (Class 1; Level C)
• For patients with STEMI on 12 Lead EKG, reperfusion therapy should be initiated as soon as possible and is not contingent on a biomarker assay! (Class 1; Level C)
Bedside ST segment Monitoring

Ventricular Remodeling after AMI
- Changes in the cardiac architecture after infarction that affect infarcted and noninfarcted areas of the heart

Ventricular remodeling in the infarcted area
- Dilation & ventricular wall thinning
- Increased wall stress on the healthy myocardium
- Sets the stage for Heart Failure
- ACE Inhibitors reduce remodeling & prevent the progression of heart failure

Heart Failure is the nation’s most rapidly growing cardiac problem.
- About 22% males & 46% female MI patients will be disabled with Heart Failure within 6 years.
- 50% Heart Failure Patients die within 5 years of MI diagnosis

Pharmacologic Management of AMI—ABC’s
- ASA
- Beta-Blocker
- Circulation (PCI) and Cholesterol Lowering Medicine
Also:
- Oxygen
- Nitroglycerin

Goals After Myocardial Infarction
- Reducing the risk of another heart attack
  - ASA
  - Antithrombotic therapy
  - Beta-blockers
  - Statins
  - ACE inhibitors
- Reducing the risk of heart failure
  - ACE inhibitors
  - Aldosterone antagonists
  - Beta-blockers
- Reducing the risk of sudden cardiac death
  - ICD therapy
Drug Eluting Stents (DES)

AMI Evidence Based Practice

- Aspirin
  - Decrease Vascular deaths from 11.8% to 9.4% (2.4%)
- Beta Blockers
  - 46% risk reduction
  - 23% Reduction all cause mortality
  - 30% Reduction in risk of sudden cardiac death
  - 26% Reduction in nonfatal reinfarction
- Statins
  - Reduce infarction size

β-Blockers
Within 24 hours & at discharge (Class 1,B)
Limit the donkey’s speed, thus saving energy

Beta Blocker “Olols”
Beta Blockade of the Sympathetic Nervous System

- Decrease oxygen demand
  - ↓ HR & contractility
  - Vasodilate
  - ↓ Afterload
  - ↓ O₂ wasteage
- Antiarrhythmic effect
- Increase oxygen supply
  - Increased diastolic perfusion
  - Less exercise vasoconstriction
Side effect: May promote spasm in vasospastic angina

Contraindications to Beta Blockers

- Bradycardia less than 60 bpm within 24hrs of DC
- 2nd or 3rd degree heart block on EKG
- Systolic BP <90 mmHg
- Allergy
- Signs of heart failure
- Evidence of a low output state
- Increased risk for cardiogenic shock

Lipid Lowering Medication if LDL > 100

- Statin goal:
  - LDL-C < 100 mg/dL (Class I, Level A)
  - consider LDL-C < 70 mg/dL (Class Iia, Level A)
Lipid – Lowering Agents

- **Statins**
  - atorvastatin (Lipitor)
  - cerivastatin (Baychol)
  - fluvastatin (Lescol)
  - lovastatin (Mevacor)
  - pravastatin (Pravachol)
  - simvastatin (Zocor)

- **Fibric Acid Derivatives**
  - gemfibrozil (Lopid)
  - micronized fenofibrate (Tricor)
  - clofibrate (Atromid-S)

- **Bile Acid Resins**
  - colestipol (Colestid)
  - cholestyramine (Questran, Questran Light, Prevalite, LoCholest)
  - colesevelam (Welchol)

- **Niacin** (Niaspan and other various brands)

ACE Inhibitors

Within 24 hours if EF < 40% (Class I; Level A)

Reduce the number of sacks on the wagon

ACE Inhibitors “Prils”

- Benazepril Lotensin
- Captopril Capoten
- Lisinopril Zestril Prinivil
- Quinapril Accupril
- Ramipril Altace

Angiotensin II receptor Blockers “ARBs” “Sartans”

- Candesartan Atacand
- Irbesartan Avapro
- Losartan Cozaar
- Valsartan Diovan
- Telmisartan Micardis
- Eprosartan Teveten
### AMI CORE Measure
- ASA & Beta Blocker within 24 hours of admission
- PCI time < 90 minutes
- Smoking Cessation Counseling
- Prescribed Discharge
  - ASA
  - Beta Blocker prescribed at discharge
  - ACEI or ARB if EF < 40%
  - Lipid Lowering Medication if LDL > 100

### Secondary Prevention
- Ask, advise, assess, and assist patients to stop smoking – I (B)
- Clopidogrel 75 mg daily:
  - PCI – I (B)
  - no PCI – IIa (C)
- Statin goal:
  - LDL-C < 100 mg/dL – I (A)
  - consider LDL-C < 70 mg/dL – IIa (A)
- Daily physical activity 30 min 7 d/wk, minimum 5 d/wk – I (B)
- Annual influenza immunization – I (B)

### Psychosocial Aspect AMI
- No Smoking?!?
- Cardiac Rehab
  - Exercise?
- Who will pay the bills?
- Will it stop next time?
- French fries?
- What about relations with my wife?
- I don’t want to take medicine
- I’m only 55
- We’ll stop next year?

### Cardiac Rehabilitation
- Began to take shape in the 1950s
- A 1995 study demonstrated that participation in CR programs resulted in a 20-25% reduction in mortality from CHD
- It also determined that CR programs are critically important to overall recovery and long term outcomes

### Cardiac Rehab
- An evolving process that helps people:
  - Change lifestyle behaviors
  - Reduce risk factors for disease progression
  - Decrease impact of disease on quality of life
  - Decrease morbidity
  - Decrease mortality
Benefits of Exercise Training

Specific benefits include:
- Improved functional capacity
- Improved blood vessel function
- Improvement in cardiovascular risk factors
- Improved coronary blood flow
- Improved electrical stability of the heart muscle (thus reducing the risk of a fatal heart rhythm disturbance)
- Reduced risk of blood clots, cardiac work, and oxygen requirement

Implantable Cardioverter-Defibrillators

- ICD therapy is indicated with LVEF < 35% due to a prior MI
- Wait at least 40 post-MI
- NYHA functional Class II or III (Class I, A)

Inferior Lateral AMI Case Study

48 y/o male has crushing chest pain
Calls 911

Top left photo --- three stenosis in RCA
RCA Case Study

Door to PCI time = 49 minutes
Ambulance EKG to PCI time = 66 minutes!
- Initial CK = 72 IU/L, CK MB = 1.0 ng/ml
  Troponin = < 0.4 ng/ml
- 8 hours later CK MB = 2.8 ng/ml, Troponin = 0.58 ng/ml
- 12 hours later CK MB = 3.3 ng/ml, Troponin = 0.51 ng/ml
- EF 55 – 65%

MINIMAL MYOCARDIAL DAMAGE

Chest pain for 8 hours prior to coming to ED
Troponin 3.73  CK 153

100% occluded RCA

RCA post stent
In Summary….

Time Is Muscle
Saving Time Saves Lives

We Can STOP Heart Attacks!

• Goal: OPEN THE ARTERY!