Acute Coronary Syndrome
Time is Muscle
Diagnosis & Treatment

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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)
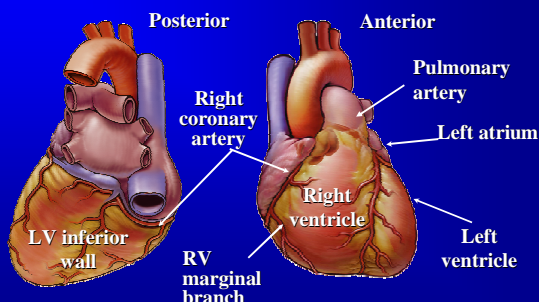 - **Unstable Angina**

Applying Classification of Recommendations and Level of Evidence

Class I	Class IIa	Class IIb	Class III
Benefit >>> Risk	*Benefit >> Risk* Additional studies with focused objectives needed	*Benefit ≥ Risk* Additional studies with broad objectives needed; Additional registry data would be helpful	*Risk ≥ Benefit* No additional studies needed
Procedure/Treatment **SHOULD** be performed/administered	**IT IS REASONABLE** to perform procedure/administer treatment	Procedure/Treatment **MAY BE CONSIDERED**	Procedure/Treatment should **NOT** be performed/administered **SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL**

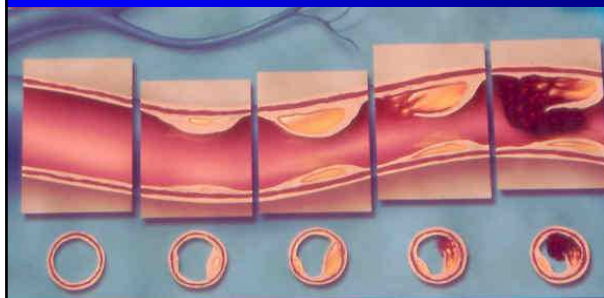|	
Level A:	Recommendation based on evidence from multiple randomized trials or meta-analyses Multiple (3-5) population risk strata evaluated; General consistency of direction and magnitude of effect
Level B:	Recommendation based on evidence from a single randomized trial or non-randomized studies Limited (2-3) population risk strata evaluated
Level C:	Recommendation based on expert opinion, case studies, or standard-of-care Very limited (1-2) population risk strata evaluated

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ACC/AHA 2007 STEMI Guidelines Focused Update Slide Set ©2007, American Heart Association, Inc and American College of Cardiology, Inc
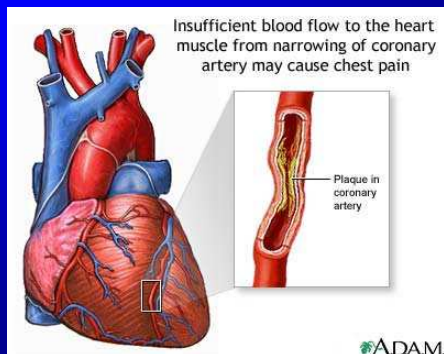
ACC/AHA 2007 STEMI Guidelines Focused Update

Cardiac Anatomy

Posterior — Anterior
Pulmonary artery
Right coronary artery
Left atrium
Right ventricle
LV inferior wall
RV marginal branch
Left ventricle

Atherosclerosis

Insufficient blood flow to the heart muscle from narrowing of coronary artery may cause chest pain
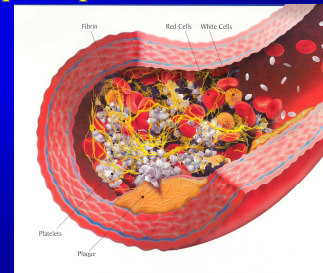
Plaque in coronary artery

Cascade effects of atherosclerotic plaque rupture

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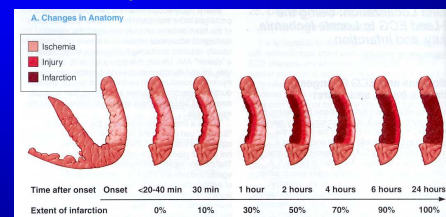- Thrombus formation
- Bleeding into the plaque
- Vasospasm

ARTERY OCCULUSION

Other Causes of STEMI

- Coronary vessel spasm
- Coronary emboli
- Vasculitis
- Severe chest trauma

Myocardial Ischemia Leads to Myocardial Necrosis

Source: AHA ACLS book 1999

♥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

Time Is Muscle

Muscle is Ejection Fraction

Ejection Fraction is Quality of Life

CRUSADING towards a GOAL

Door to

EKG

10 minutes or less

(Class 1: Level C)

EKG Changes with MI

Ischemia < 20 Minutes
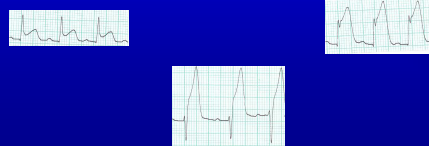**Lack of oxygen to the myocardial muscle**
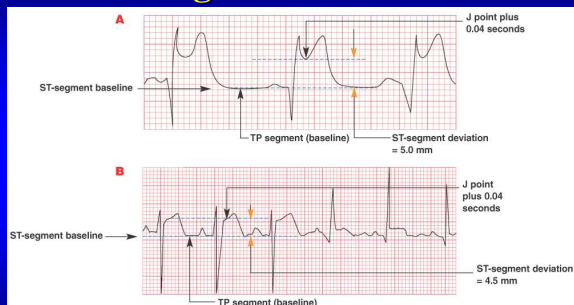- ♥ Peaked T Waves
- ♥ Inverted T Waves
- ♥ ST Segment Depression

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

## Measurement of ST-Segment Deviation

A
J point plus 0.04 seconds
ST-segment baseline
TP segment (baseline)
ST-segment deviation = 5.0 mm

B
J point plus 0.04 seconds
ST-segment baseline
ST-segment deviation = 4.5 mm
TP segment (baseline)

STEMI: ≥1 mm ST-segment elevation in 2 leads.*
NSTEMI/UA: ≥0.5 mm ST-segment ischemic depression in 2 leads.*
*Anatomically (regionally) contiguous leads.
Source: AHA ACLS EP

Review of Normal QRS complex

qRs   QS   Rs   qR   rSR'
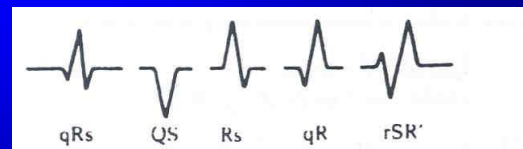
- Q wave is the first negative deflection after the p wave
- R wave is the first positive deflection after the p wave
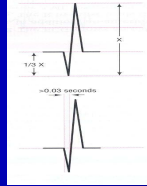- S wave is the second negative deflection after the R wave

EKG Changes with MI

Abnomormal (significant Q waves
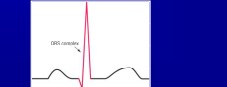
Infarction > 1 - 2 hours
- ♥ **Abnormal Q waves**
  - > 1/3 the height of R wave in that lead
  - or
  - > 0.03 ms wide

Normal Q wave
1st downward deflection of QRS
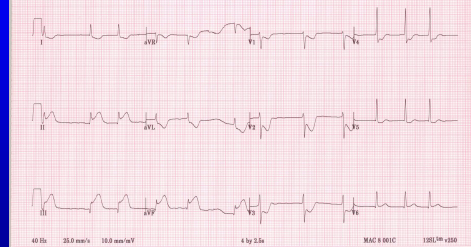
Evolution of AMI

EM #1 December 13 at 1701

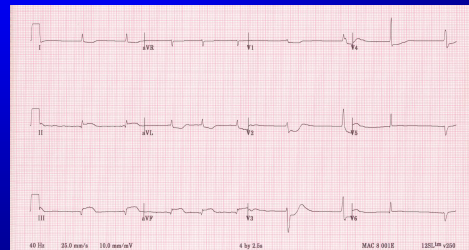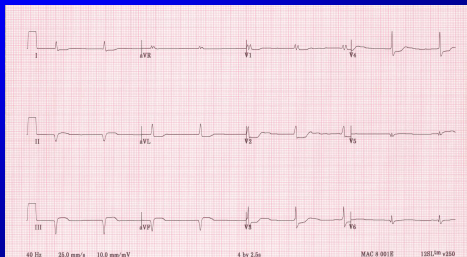
Evolution of AMI
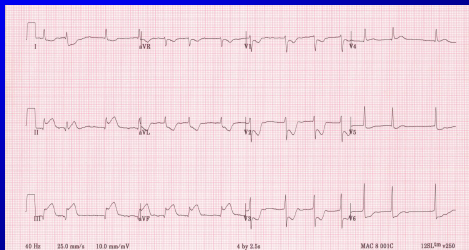
EM # 2 December 13 at 1823

Evolution of AMI

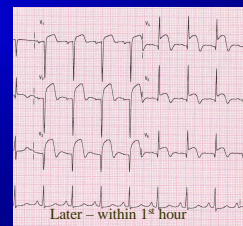**EM #3 December 14 at 0630**

Evolution of AMI

**EM # 4 December 15 0600**

Hyperacute Phase of MI

- ♥ **Occurs within minutes to first hour of chest pain**
- ♥ **Very tall ST segment**
- ♥ **Tall peaked symmetrical T waves**

Within minutes   Later – within 1st hour

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**
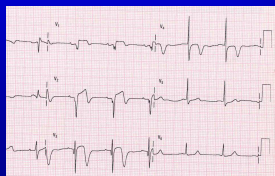
Evolving Phase of MI

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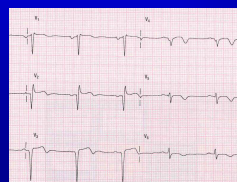
Resolving Phase of MI

- ♥ **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**
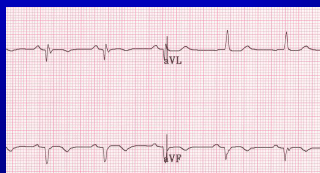
Positive EKG

- ST elevation > O.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)
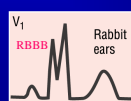
We Can STOP Heart Attacks!

- **Goal: OPEN THE ARTERY!**

Time Is Muscle

Muscle is Ejection Fraction

**Ejection Fraction is
Quality of Life**

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

Assessment Questions (OPQRST)

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

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

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**
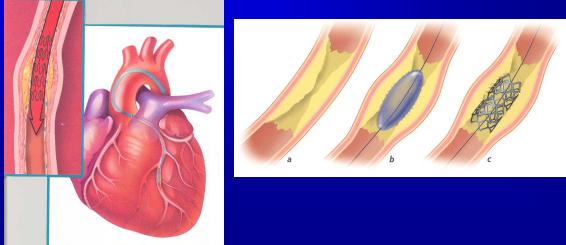
Reaching this target….

- 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 1, 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

  - DeLuca, Circulation 2004;109:1223-1225.
  - Berger, Circulation 1999;100:14-20.

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 II a: 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

Time	Troponin I	CK	CKMB	Myoglobin
1625	<0.35	55	2.2	133.8 ↑
2100	31.9 ↑	767 ↑	47.2 ↑	368 ↑
2230	42.4 ↑	853 ↑	99.5 ↑	-----
0400 - 12 hours later	63.0 ↑	1091 ↑	104 ↑	-----
0400 - 36 hours later	14.9 ↑	277 ↑	12.5	-----

81 y/o female presents with c/o of chest discomfort over the weekend

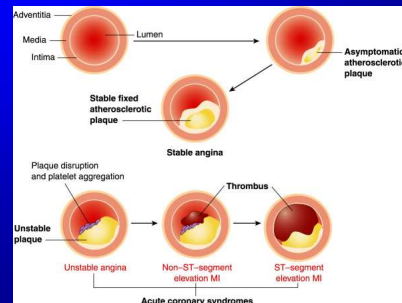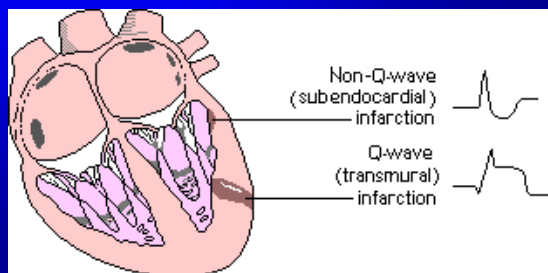| Time	Troponin I	CK	CKMB	Myoglobin
1700	6.9 ↑	198	7.2 (s)	66.1
1800	5.7 ↑	163	6.8 (s)	61.7
2330	6.4 ↑	146	6.0 (s)	-----
0500 - next day	6.0 ↑	120	5.4 (s)	-----

AMI probably > 24 hours ago

NSTEMI
Non ST Segment
Elevation MI

- ⊙ No ST segment Elevation
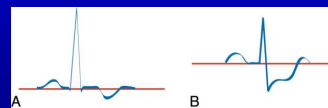- ⊙ ST segment depression
- ⊙ New LBBB

STEMI vs NSTEMI

STEMI vs NSTEMI

Non-Q-wave (subendocardial) infarction

Q-wave (transmural) infarction

Both elevated Troponins

STEMI

PCI < 90 minutes

NSTEMI

PCI within 24 - 48 hours

A   B

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

Antiplatelet Antithrombin Therapy

Risk Stratification

TIMI Risk Score of NSTEMI/UA

Historical	Points
•Age > 65	1
•3 or > cardiiac risk factors	1
(age, male, family history, hyperlipidemia diabetes, smoking, hypertension, obesity)	
•Documented prior coronary artery stenosis > 50 %	1
•Use of ASA in last 24 hours	1
•2 or > anginal events in past 24 hr	1
•Elevated Cardiac Markers	1
•ST segment deviation	1
•(Transient elevationn or persistent depression	1
•_____	
•Risk score = total points (0 - 7)	

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 OR
  – 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
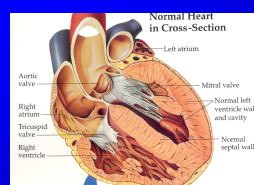
Heart Failure is the nation's most rapidly growing cardiac problem.

- **About 22% males & 46% female MI patients will be disabled with Heart Failure within 6 years.**

50% Heart Failure Patients die within 5
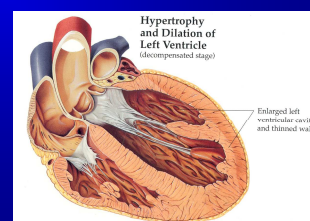
Pharmacologic Management of AMI– *ABC's*

- **A**SA
- **B**eta-Blocker
- **C**irculation (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

ß-Blockers

Within 24 hours & at discharge (Class 1,B)

Limit the donkey's speed, thus saving energy
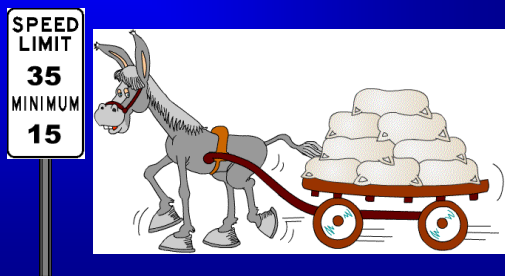
Beta Blocker "Olols"

Beta Blockade of the Sympathetic Nervous System

- Decrease oxygen demand
  - ↓ HR & contractility
  - Vasodilate
  - ↓ Afterload
  - ↓ O_2 wasteage
- Antiarrhythmic effect
- Increase oxygen supply
  - Increased diastolic perfusion
  - Less exercise vasocontriction

  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 B/P <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)
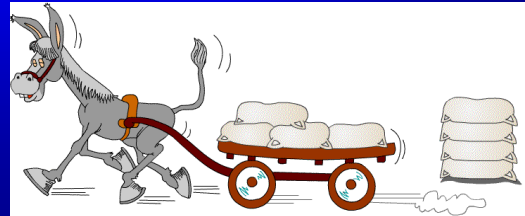 - consider LDL-C < 70 mg/dL (Class IIa, Level A)

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)
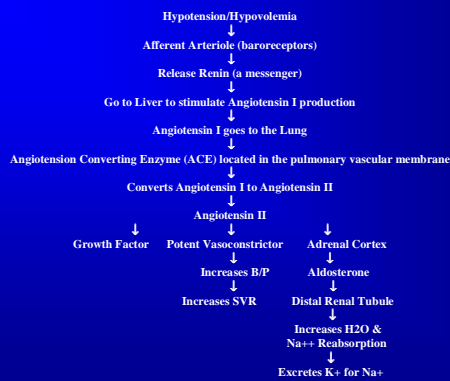 - colesevelam (Welchol)
- Niacin (Niaspan and other various brands)

ACE Inhibitors

Within 24 hours if EF < 40% (Class 1; Level A)

Reduce the number of sacks on the wagon

Renin-Angiotensin-Aldosterone System (RAAS)

Hypotension/Hypovolemia
↓
Afferent Arteriole (baroreceptors)
↓
Release Renin (a messenger)
↓
Go to Liver to stimulate Angiotensin I production
↓
Angiotensin I goes to the Lung
↓
Angiotension Converting Enzyme (ACE) located in the pulmonary vascular membrane
↓
Converts Angiotensin I to Angiotensin II
↓
Angiotensin II
↓

Growth Factor	Potent Vasoconstrictor	Adrenal Cortex
	↓	↓
	Increases B/P	Aldosterone
	↓	↓
	Increases SVR	Distal Renal Tubule
		↓
		Increases H2O & Na++ Reabsorption
		↓
		Excretes K+ for Na+

Renin-Angiotensin-Aldosterone System (RAAS)

Hypotension/Hypovolemia
↓
Afferent Arteriole (baroreceptors)
↓
Release Renin (a messenger)
↓
Go to Liver to stimulate Angiotensin I production
↓
Angiotensin I goes to the Lung
↓
Angiotension Converting Enzyme (ACE) located in the pulmonary vascular membrane
↓
Converts Angiotensin I to Angiotensin II
↓
Angiotensin II
↓

Dilatation & Diuresis
ACE Inhibitors "Prils"

Angiotensin II receptor Blockers "ARBs" "Sartans"

Growth Factor	Potent Vasoconstrictor	Adrenal Cortex
	↓	↓
	Increases B/P	Aldosterone
	↓	↓
	Increases SVR	Distal Renal Tubule
		↓
		Increases H2O & Na++ Reabsorption
		↓
		Excretes K+ for Na+

ACE Inhibitors "Prils"

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

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)

ACC/AHA 2007 STEMI Guidelines Focused Update

Psychosocial Aspect AMI

CARDIAC REHABILITATION

JOSIE HARRINGTON, RN
LEAD REGISTERED NURSE

Cardiac Rehab

- 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)
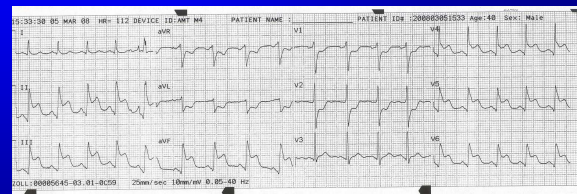- Reduced risk of blood clots, cardiac work, and oxygen requirement

Implantable Cardioverter-Defibrillators

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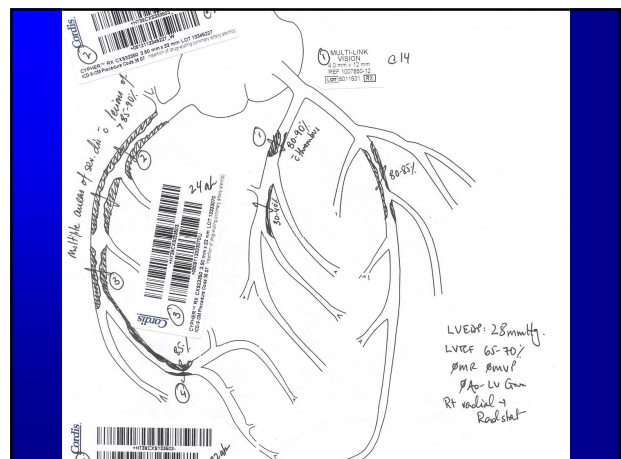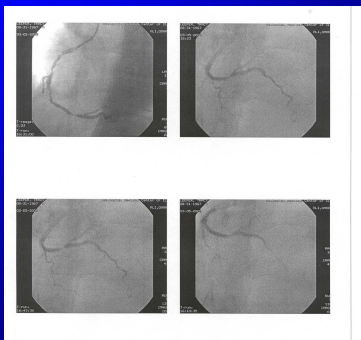- NYHA functional Class II or III
  (Class I, A)
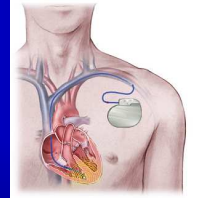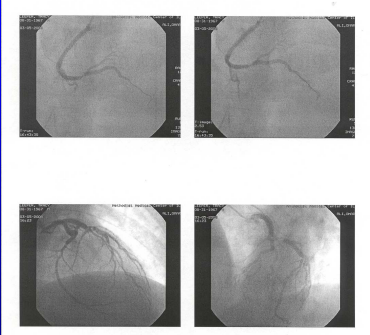

Inferior Lateral AMI
Case Study

48 y/o male has crushing chest pain Calls 911

Top left photo --- three stenosis in RCA

Top right photo – RCA after 3 stents
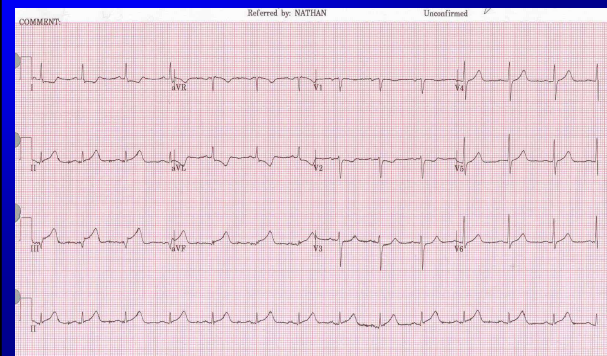## Bottom right – Cx

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
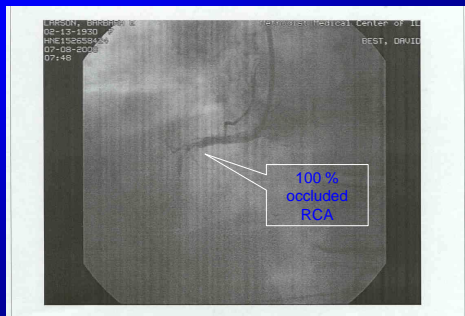- 12 hours later CK MB = 3.3 ng/ml, Troponin = 0.51ng/ml
- EF 55 – 65%

MINIMAL MYOCARDIAL DAMAGE
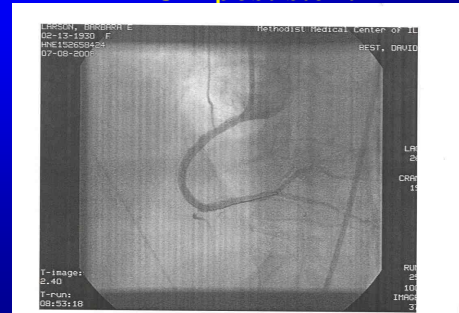
RCA Case Study

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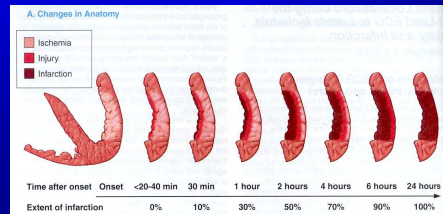## Troponin 3.73 CK 153

100% occluded RCA

100 % occluded RCA

RCA post stent

In Summary….

Time Is Muscle
Saving Time Saves Lives

A. Changes in Anatomy							
Ischemia							
Injury							
Infarction							

Time after onset	Onset	<20-40 min	30 min	1 hour	2 hours	4 hours	6 hours	24 hours
Extent of infarction		0%	10%	30%	50%	70%	90%	100%

We Can STOP Heart Attacks!

- Goal: OPEN THE ARTERY!