Acute myocardial infarction

Thomas L McKiernan M.D.

Learning Objectives

1. Recognize the critical importance of early diagnosis and treatment of acute MI.
   A. ST Elevation
   B. Non-ST Elevation
2. Be familiar with the early post MI management and pre-discharge evaluation.

References

- ACC AHA Guidelines for UA/NSTEMI and Guidelines for STEMI
- Published in JACC and Circulation

AMI Stats……

- ***Incidence in the United States****
– Estimated 900,000 will suffer AMI this year
- *~565,000 will be new attacks (avg. age- 65.8yrs/males, 70.4yrs/female)*
- *~300,000 will be recurrent attacks*

– 42% of AMI pts will die within 1 year
– Approximately half of these deaths occur before reaching the emergency department
– Most cardiac deaths are the result of fatal arrhythmias

- ***Types of arrival/discharge AMIs*****

– Upon arrival: STEMI on 1st ECG-26%; STEMI on 1st or subsequent ECG-35%; NSTEMI-65%
– Non-Q-wave: 75% Q-wave: 25%

**American Heart Association. Heart Disease & Stroke Statistics-2004 Update*
***NRMI 4 Quarterly Data Report (Nation). South San Francisco, Calif: Genentech Inc; June, 2004.*

Definition
World Health Organization

1. Clinical history of ischemic type discomfort (70-80%)

2. Changes on serially obtained ECG's (50%)

3. Rise and fall in serum cardiac markers

Criteria for Acute Myocardial Infarction

- Detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit together with evidence of ischemia with at least one of the following:
  - Symptoms of ischemia
  - ECG changes of new ischemia (new ST-T changes or new LBBB)
  - Development of pathological Q waves in the ECG
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

ECG = electrocardiogram; LBBB = left bundle branch block

Jaffe,A Cardiosource Expert Opinion

Clinical Classification of Different Types of Myocardial Infarction

Type 1

Spontaneous myocardial infarction related to ischemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection

Type 2

Myocardial infarction secondary to ischemia due to either increased oxygen demand or decreased supply; e.g., coronary artery spasm, coronary embolism, anemias, arrhythmia hypertension, or hypotension

Type 3

Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischemia, accompanied by presumably new ST-elevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood

Type 4a

Myocardial infarction associated with PCI

Type 4b

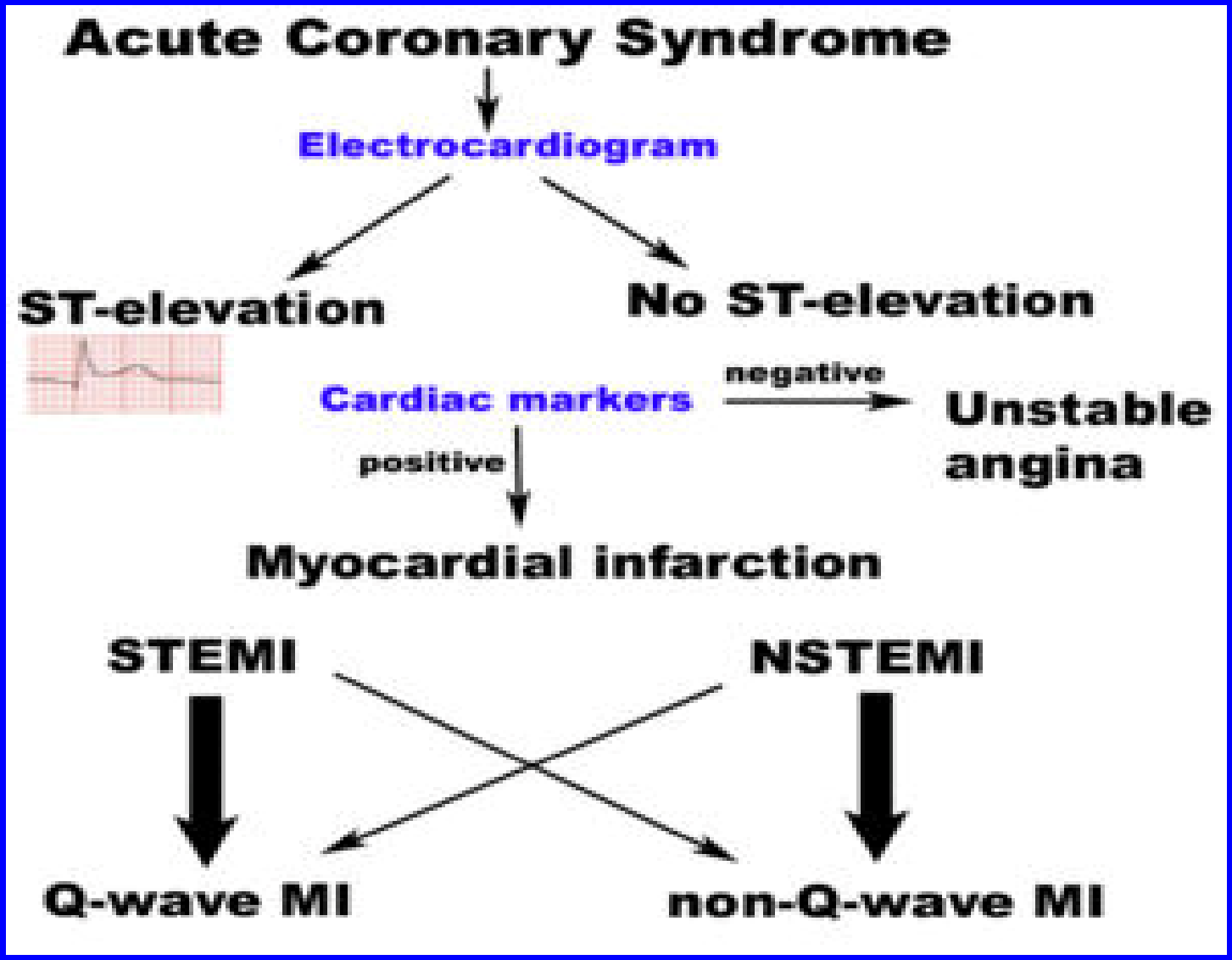
Myocardial infarction associated with stent thrombosis as documented by angiography or at autopsy

Type 5

Myocardial infarction associated with CABG

CABG = coronary artery bypass graft; LBBB = left bundle branch block; PCI = percutaneous coronary intervention.

Jaffe,A Cardiosource Expert Opinion


Acute Coronary Syndrome
Electrocardiogram
ST-elevation
No ST-elevation
Cardiac markers
negative
Unstable angina
positive
Myocardial infarction
STEMI
NSTEMI
Q-wave MI
non-Q-wave MI

Early Reperfusion Improves LV Function And Survival In Patients With ST Elevation or BBB

Achieve Coronary Patency

- **Initial Reperfusion Therapy**
  - Defined as the initial strategy employed to restore blood flow to the occluded coronary artery
- **3 Major Options:**
  - Pharmacological Reperfusion
  - PCI
  - Acute Surgical Reperfusion
- **Under both Pharmacological and PCI are listed several lower recommendations & investigational reperfusion strategies**

Class I All patients should undergo rapid evaluation for reperfusion therapy & have a reperfusion strategy implemented promptly after contact with the medical system

Antman et al. JACC 2004;44:680.

Initial Patient Evaluation

Class I

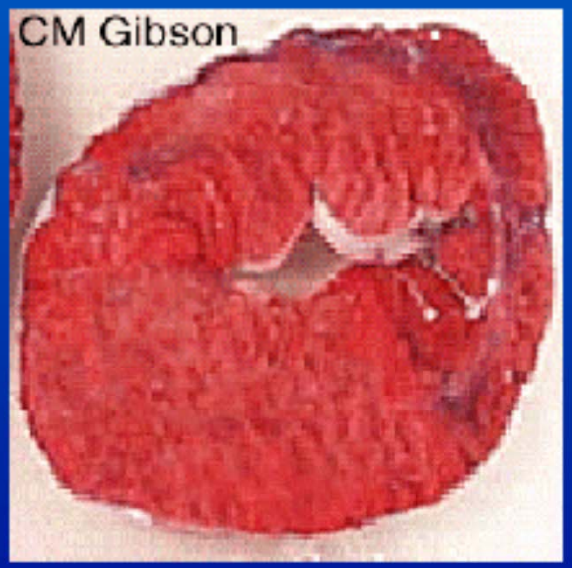
Delay in patient contact (arrival at the ED or contact with paramedics) **to**:

- **fibrinolytic therapy *less than* 30 minutes**
- **PCI *less than* 90 mins**

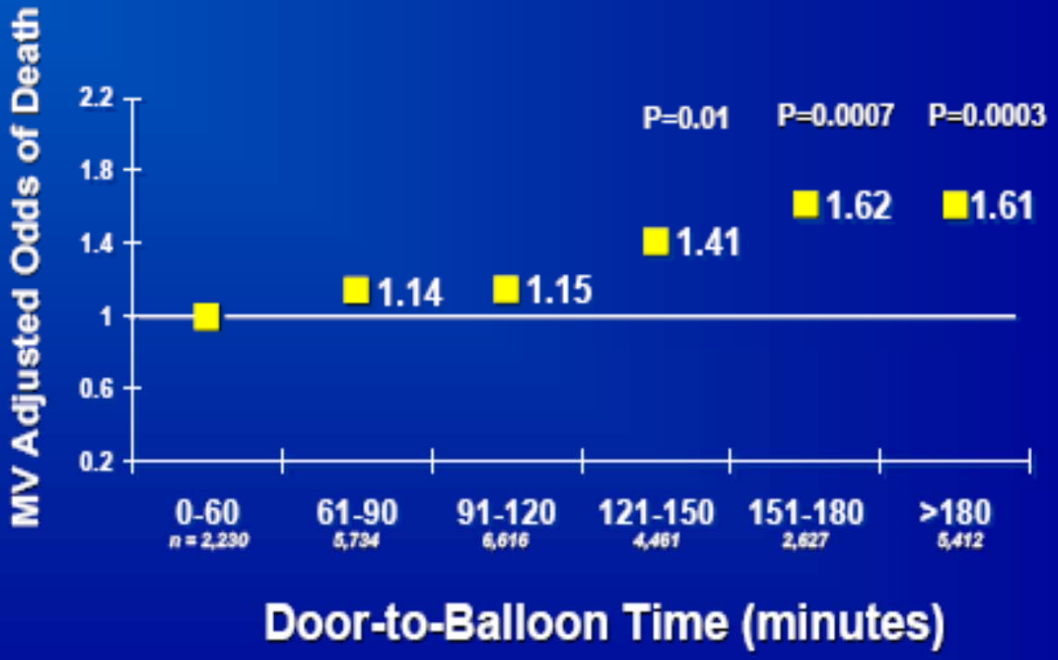
(Level of Evidence: B)

2. The choice of initial STEMI treatment should be made by ED Physician on duty based on a **predetermined, institution-specific, written protocol….** For unclear cases, not covered by the protocol, contact cardiologist immediately.
(Level of Evidence C).

Antman et al. JACC 2004; 44:677-8.



NRMI 2: Primary PCI Door-to-Balloon Time vs. Mortality



Patients Transported by EMS After Calling 9-1-1

Call 911
Call Fast

Onset of STEMI Symptoms → 9-1-1 EMS Dispatch → EMS on-scene
- Encourage 12-lead ECG
- Consider prehospital fibrinolytic if capable and EMS-to-needle < 30 min

EMS Triage Plan

Hospital Fibrinolysis: Door-to-needle within<30 min → Not PCI Capable Hospital

Interhospital Transfer → PCI Capable Hospital

Goals

Patient	Dispatch	EMS on scene	EMS transport
5 min after Symptom onset	1 min	Within 8 min	EMS transport:EMS to Balloon within 90 min
			Patient self-transport: Hospital Door-to-Balloon within 90 min

Total ischemic time: Within 120 min*

* Golden hour = First 60 min

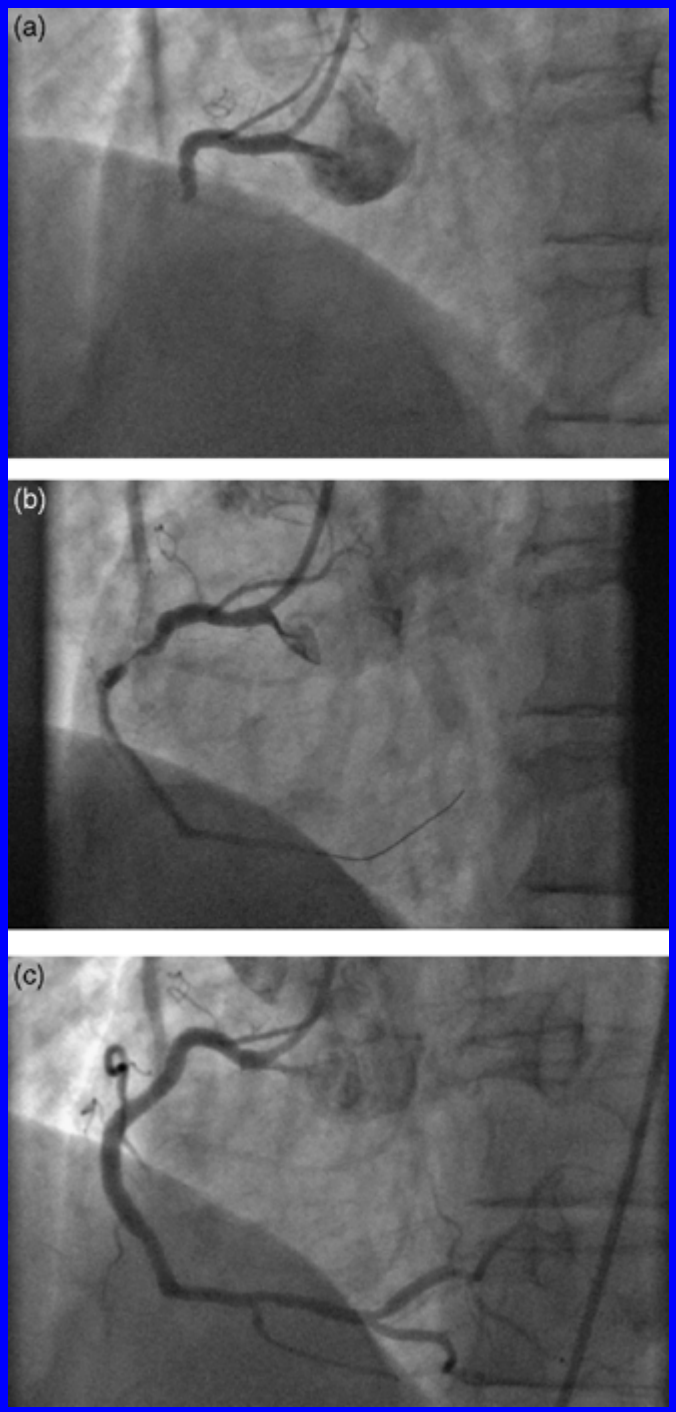
Adapted from Panel A Figure 1
Antman et al. JACC 2004;44:676.

Emergency Department

- Within 10 minutes
  - Oxygen by nasal prongs
  - Sublingual Nitroglycerin unless BP <90 or Heart rate <50 or > 100
  - Adequate Analgesia
  - Aspirin 160-325 mg
  - 12 Lead EKG

TIMI flow

- 0 No flow
- 1 Partial filling of the vessel distal to the obstruction
- 2 Complete filling of the distal vessel but slower than normal
- 3 Normal antegrade filling of the distal vessel

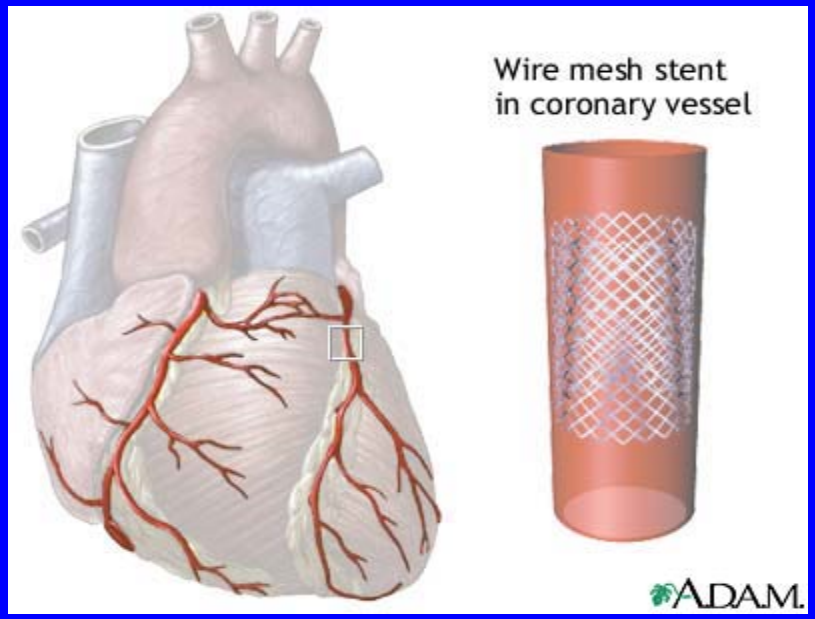

(a)
(b)
(c)

Thrombolytic Therapy

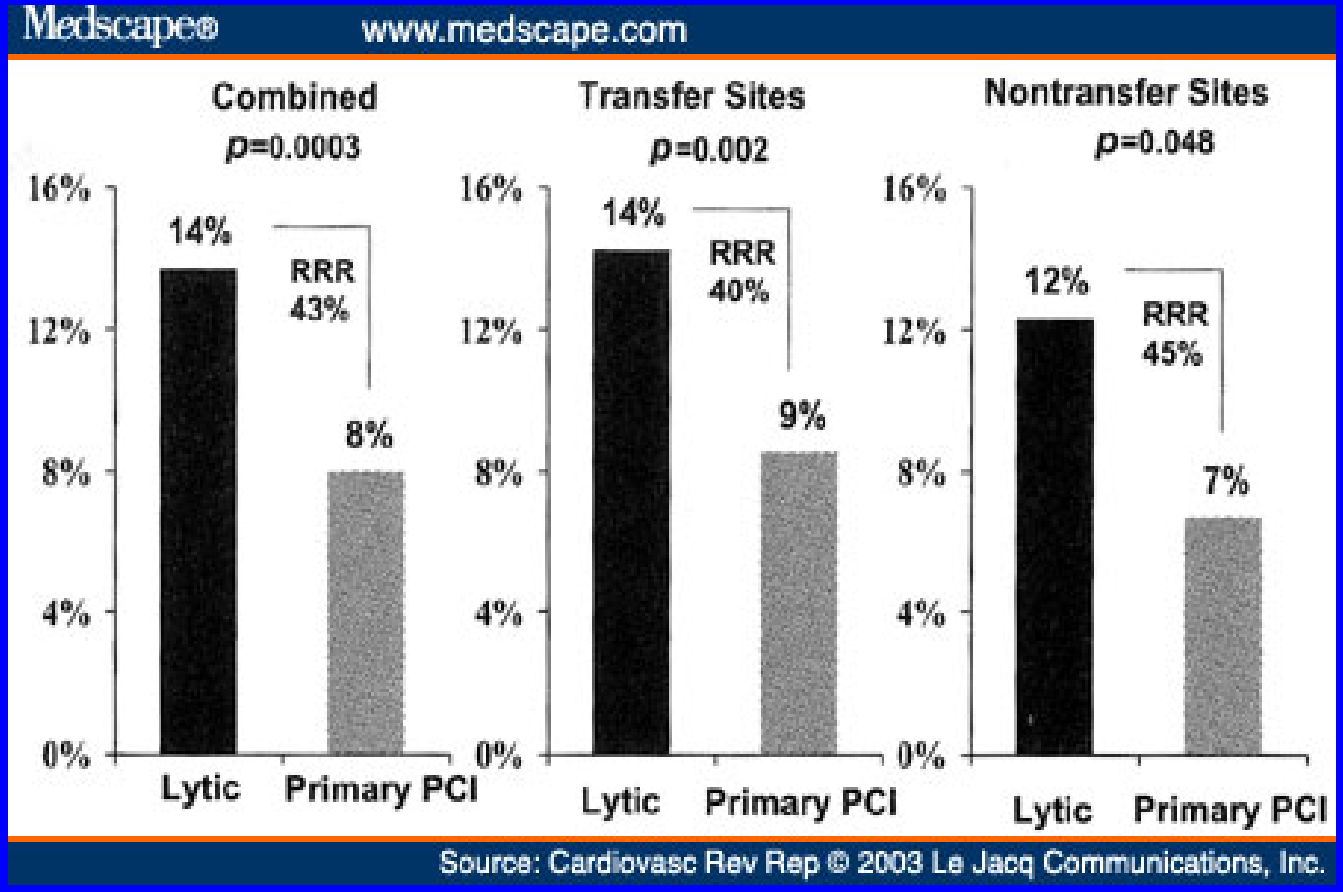
- **Thrombolysis** is the breakdown (*lysis*) of blood clots[1] by pharmacological means. It is colloquially referred to as *clot busting* for this reason. It works by stimulating fibrinolysis by plasmin through infusion of analogs of tissue plasminogen activator (tPA), the protein that normally activates plasmin.

STEMI :patients presenting to a hospital with PCI capability

- Treat with primary percutaneous coronary intervention(PCI) within 90 minutes of first medical contact



STEMI Mortality…… PCI v Lytics



Post PCI STEMI stented patients and ASA

- Assuming no ASA resistance ,allergy ,or bleeding
- ASA 325mg po
- 1 m after BMS,3m after Sirolimus and 6m after Taxus stent placement after which long term ASA at 75-162mg daily should be continued indefinitely.

Fibrinolytic Therapy

Step 2: Determine Whether Fibrinolysis or an Invasive Strategy is Preferred

If presentation is less than 3 hours and there is no delay to an invasive strategy, there is no preference for either strategy.

Fibrinolysis is generally preferred if:

- ***Early presentation*** (3 hours or less from symptom onset & delay to invasive strategy; see below)
- ***Invasive strategy is not an option***
  - Catheterization lab occupied/not available
  - Vascular access difficulties
  - Lack of access to a skilled PCI lab-
    - Operator experience > 75 PPCI cases per year/
    - Team experience >36 PPCI cases per year
- ***Delay to invasive strategy***
  - Prolonged transport
  - (Door-to Balloon) – (Door-to- needle) time is > 1 HR
  - Medical contact-to- balloon time is > than 90 min

An invasive strategy is generally preferred if:

- ***Skilled PCI laboratory available with surgical backup***
  - Medical contact-to- balloon time is > than 90 min
  - (Door-to Balloon) – (Door-to- needle time) is > 1 hr
- ***High risk from STEMI***
  - Cardiogenic shock
  - Killip class greater than or equal to 3
- ***Contraindications to fibrinolysis, including increased risk of bleeding and ICH***
- ***Late presentation***
  - Symptom onset was more than 3 hours ago
- ***Diagnosis of STEMI is in doubt***

Adapted from Figure 3; Antman et al. *JACC* 2004;44:682.

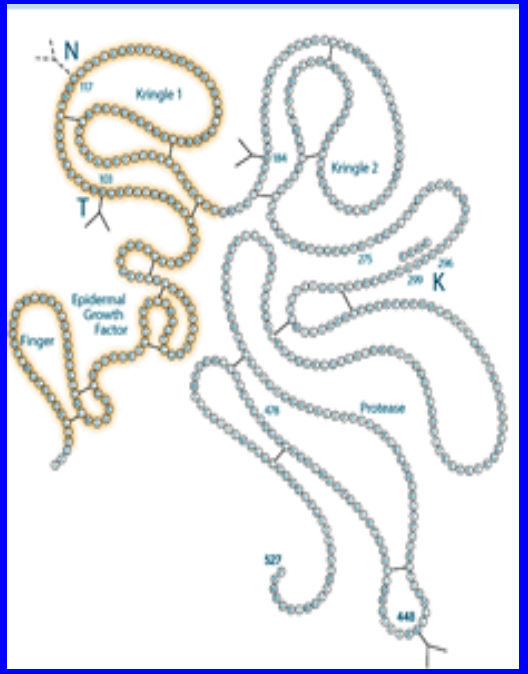
Comparison of Approved Fibrinolytic Agents

	Streptokinase	Alteplase	Reteplase	Tenecteplase
•Dose	1.5 MU over 30-60 min	Up to 100mg in 90 min (wt-based)	10U x 2 each over 2 min	30-50mg based on weight
•Bolus Admin.	No	No	Yes	Yes
•Antigenic	Yes	No	No	No
•Allergic React	Yes	No	No	No
•Systemic Fibrinogen Depletion	Marked	Mild	Moderate	Minimal
• ~90-min patency rates (%)	50	75	75?	75
•TIMI grade 3 flow, %	32	54	60	63

Adapted from Table 15, pg 53.Accessed on August 6, 2004 http://www.acc.org/clinical/guidelines/stemi/index.pdf.

TNKase….. For thrombolytic therapy

Investigate the Molecule



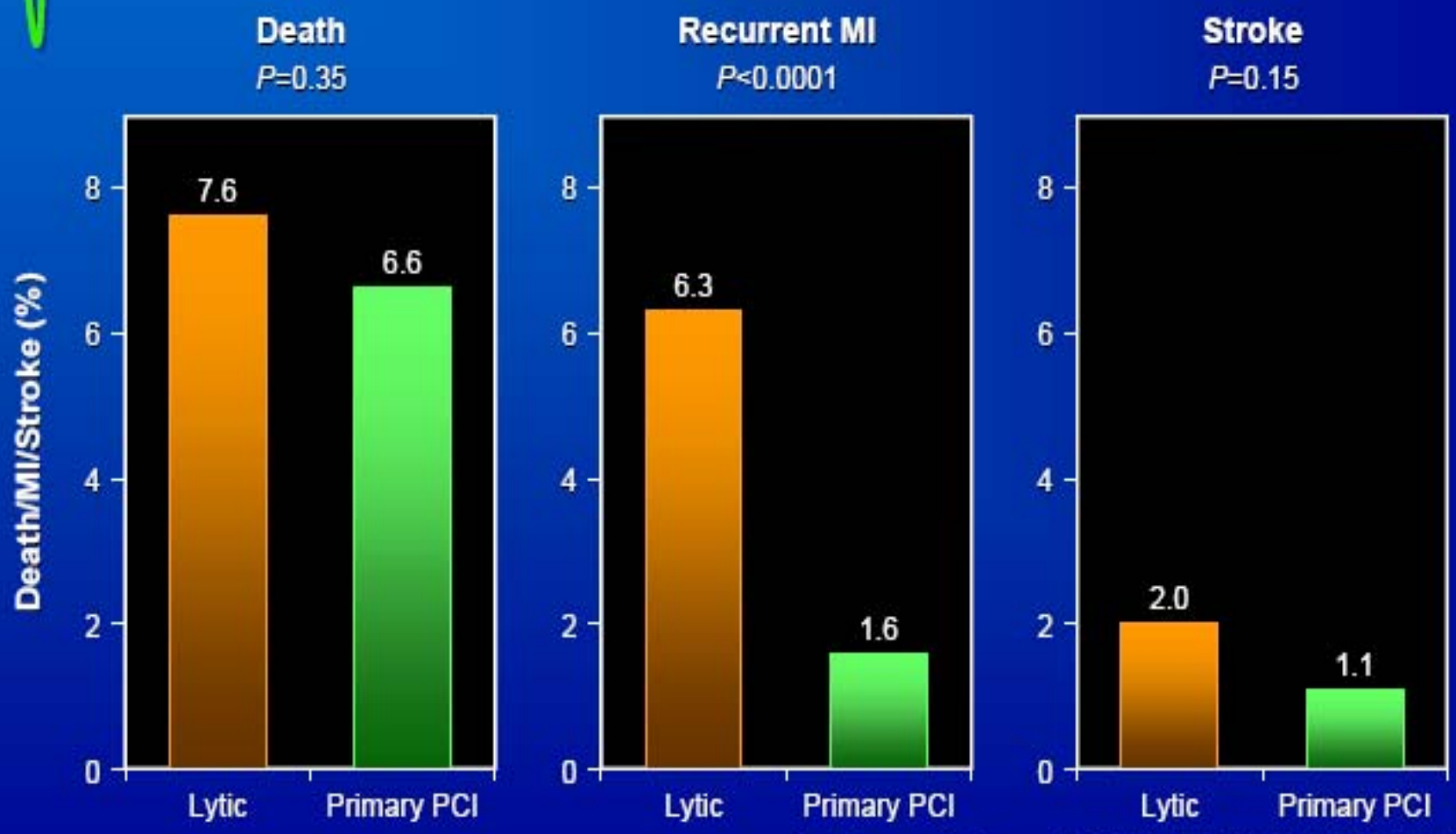
An Advanced Lytic by Design

Genentech's goal in developing TNKase was to create a thrombolytic with refined clinical properties relative to wild-type recombinant tissue plasminogen activator (rt-PA). TNKase incorporates three targeted alterations, each of which contributes to the pharmacological improvements that are seen in TNKase relative to rt

Patients undergoing reperfusion with fibrinolytics should receive anti coagulant therapy for 48 hrs

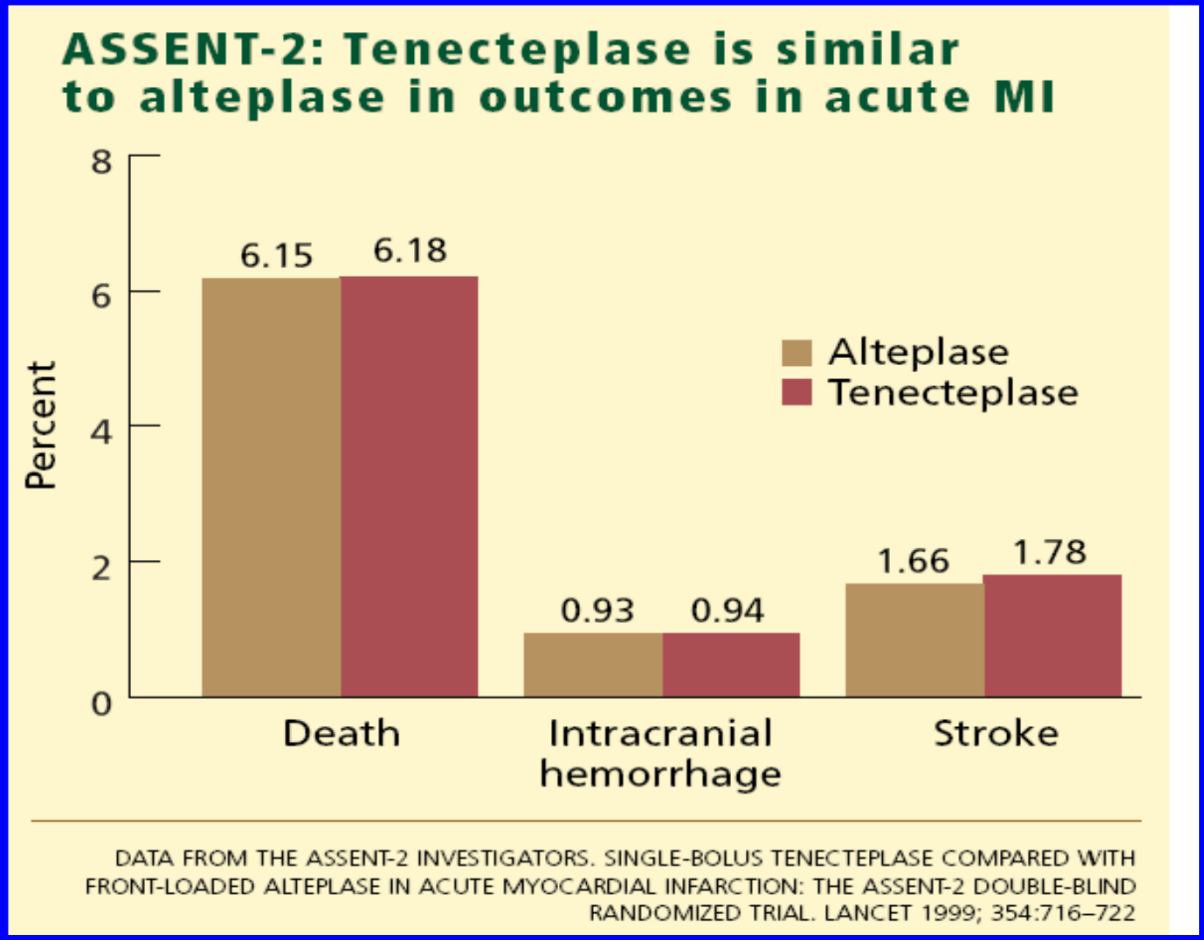
- UHF (IV bolus 60U/kg,IV infusion of 12U /Kg/h, max 4000u…1000u,to PTT~50-70s
- ENOXAPARIN (if crt <2.5mg men,<2.0mg
- Women)and < 75 yrs of age .30mg IV bolus then 1mg/kg every 12 hrs. Crt clearance < 30,the subcutaneous regimen is 1mg/kg every 24 hrs .Coninue for index hospitalization for up to 8 days.
- FONDAPARINUX initial 2.5mg IV,2.5mg once daily for up to 8 days (crt < 2.5mgs)

DANAMI-2: Results



Anderson HR, et al. *NEJM* 2003;349:733-42

Fibrinolytic therapy has been shown to reduce the 30-day mortality rate by approximately 30% in patients presenting with ST-elevation MI or bundle branch block.30



Summary: Selection of the Optimal Reperfusion Options for the STEMI Patient: 2004

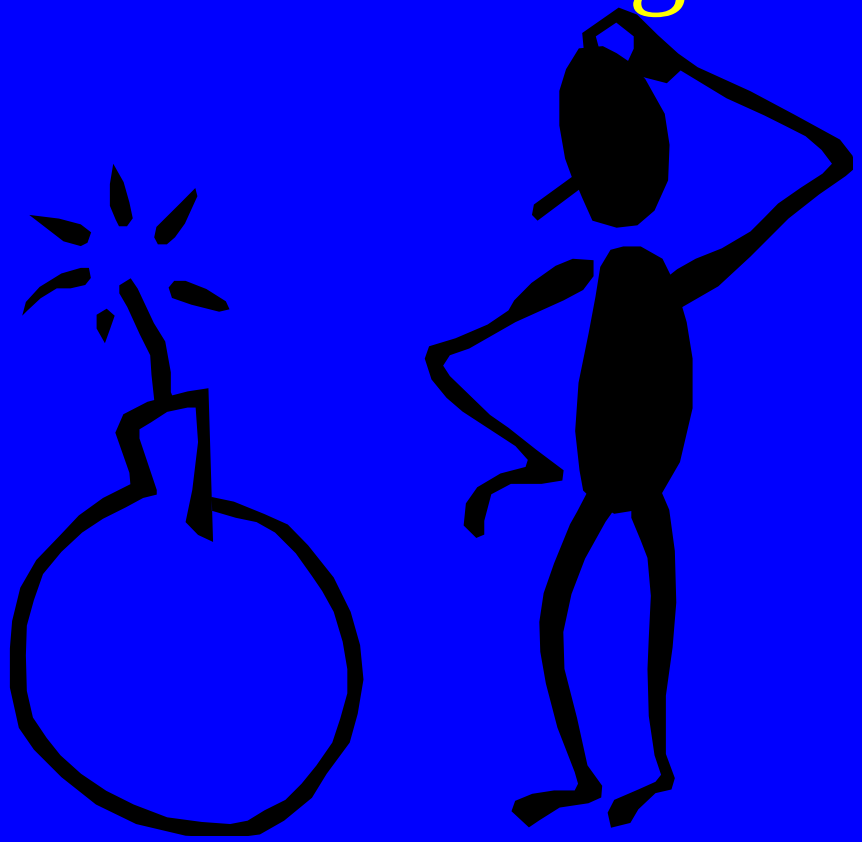
Full Dose Fibrinolytic Monotherapy

- Door to balloon (D-B) > 90 min
- Lack of access to skilled PCI center
- (D-B) – (D-N) > 1 h
- < 3 h from symptom onset

Invasive Strategy

- Cardiogenic shock (age < 75)
- Bleeding risk
- Diagnosis in doubt (pericarditis/aneurysm)
- Door to balloon < 90 min
- Skilled PCI center available, defined by:
  - Operator experience > 75 cases/yr
  - Team experience > 36 primary PCI/yr
- Age > 75
- Symptoms > 2-3 h

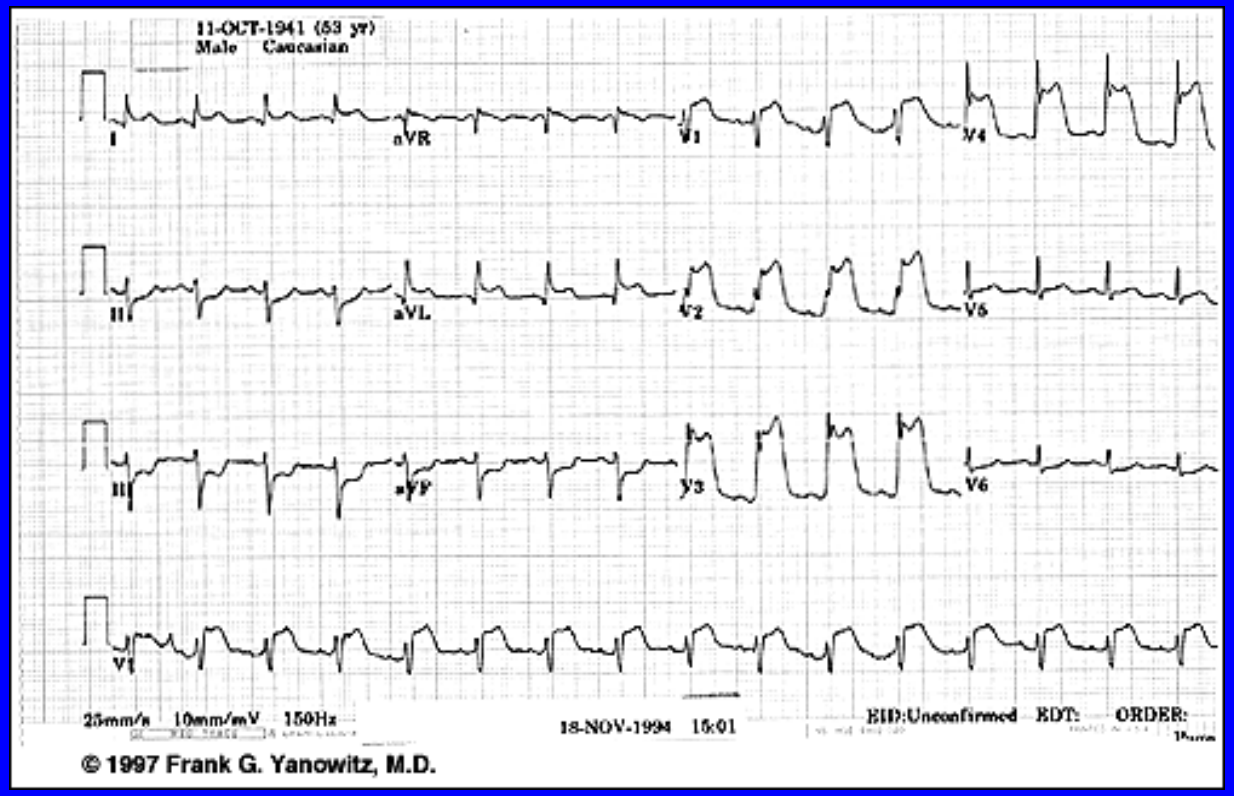
Back to the Diagnosis!



Detection

- History
- History
- History
- History
- History
- Exam
- ECG
- “Enzymes”

STEMI Acute Anterior MI…….



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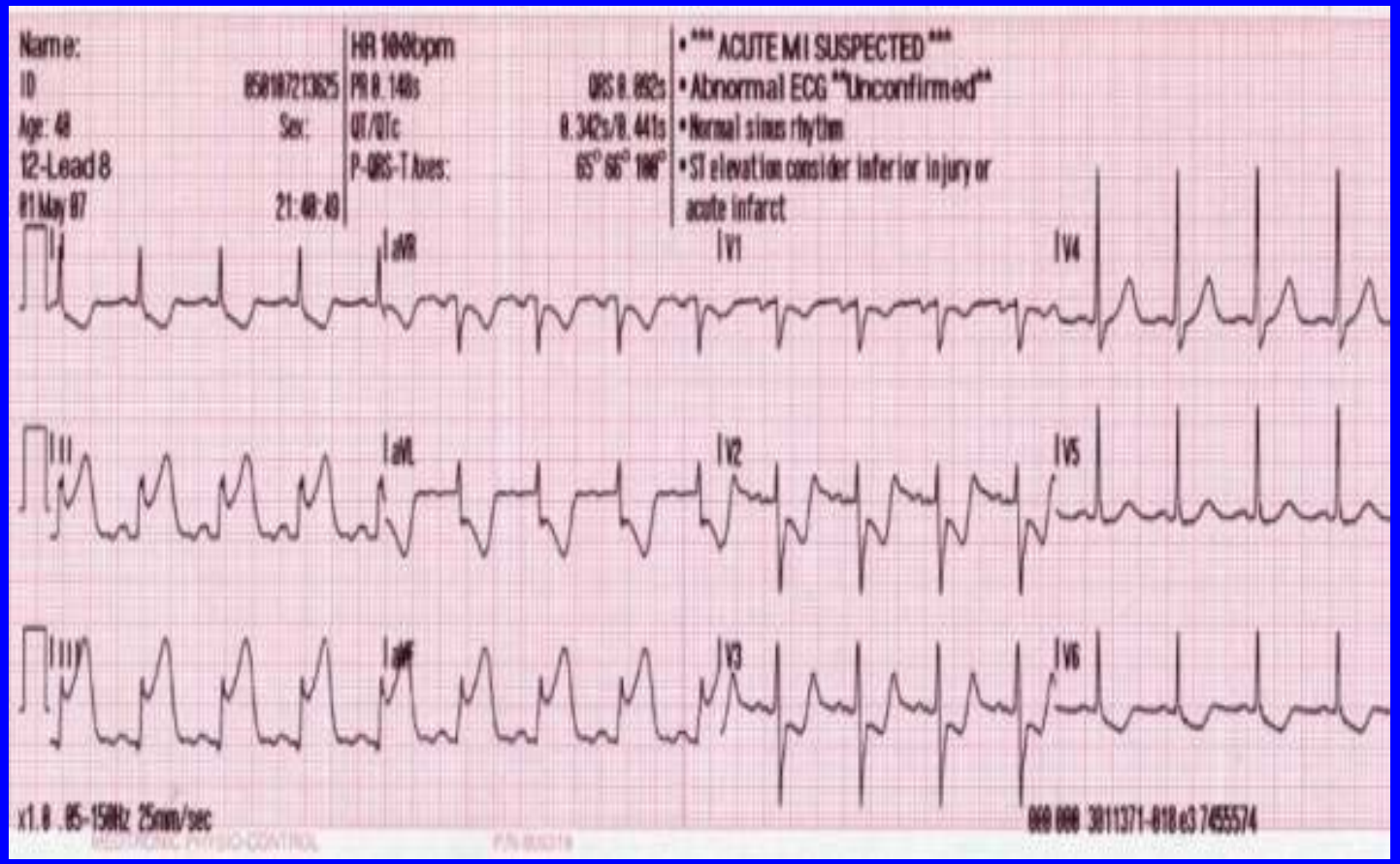
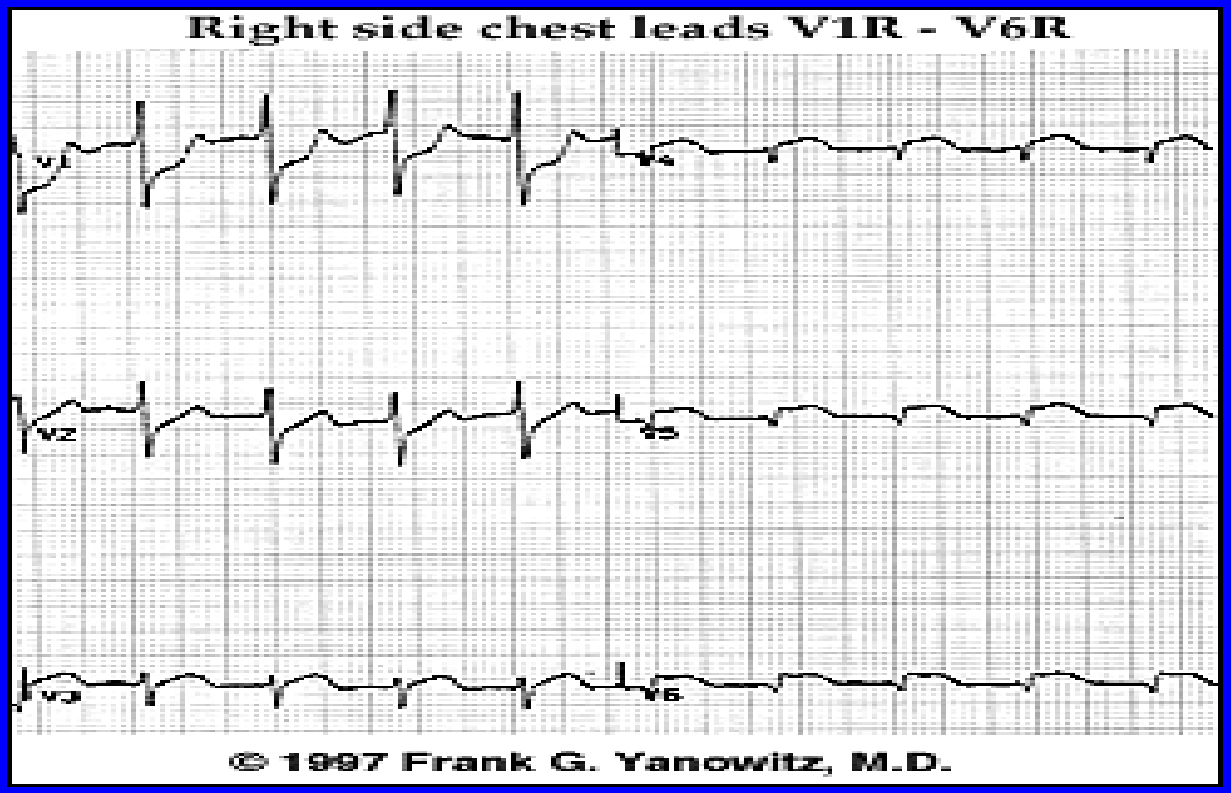
Jaffe,A Cardiosource Expert Opinion

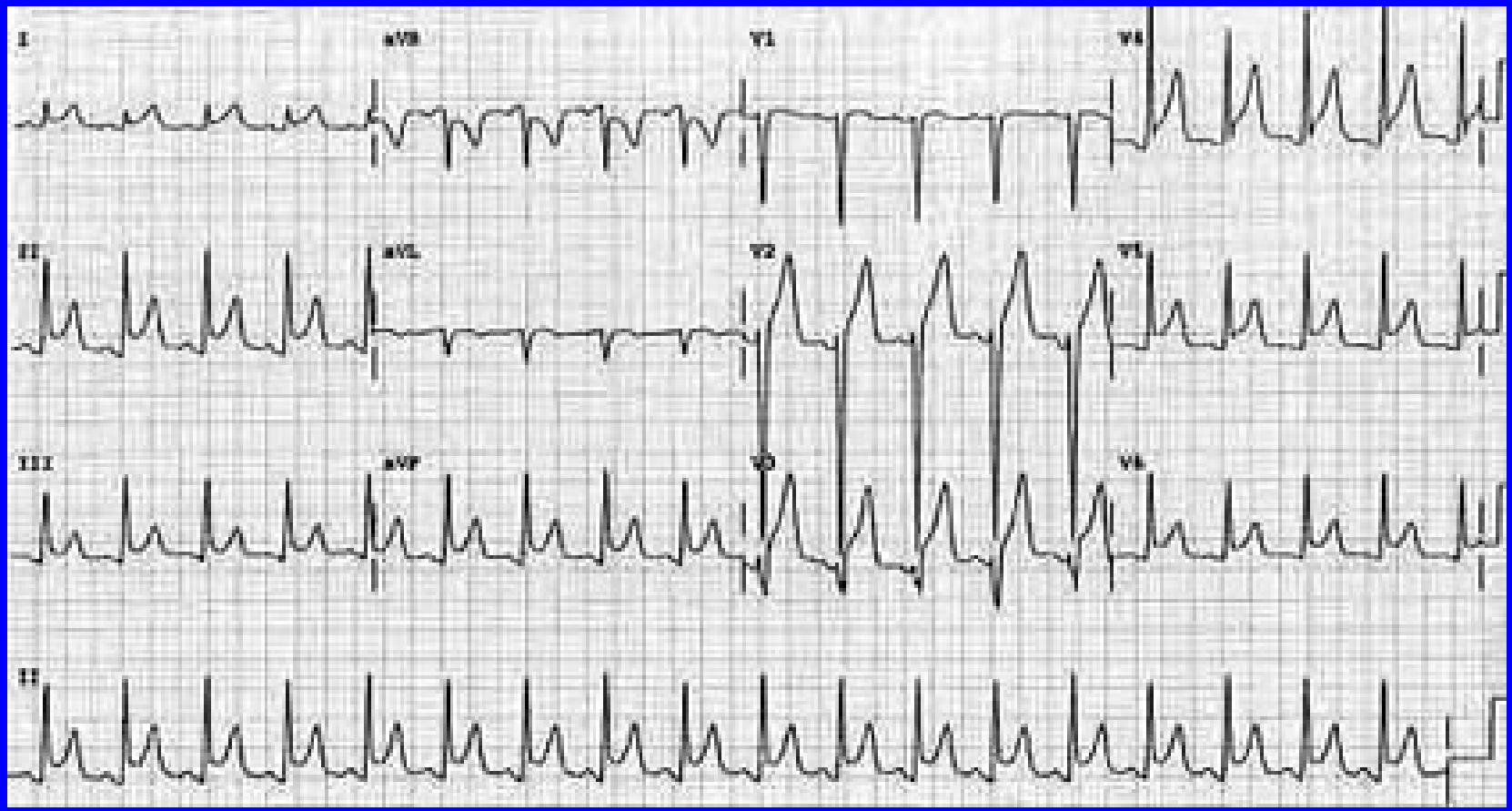
STEMI inferior mi



RV Infarction…….



Pericarditis



Myoglobin

- Not cardiac specific
- **Released earlier but still only about 60 % at 3 hours**

"Enzymes" = Serum Markers (Intracellular Macromolecules)

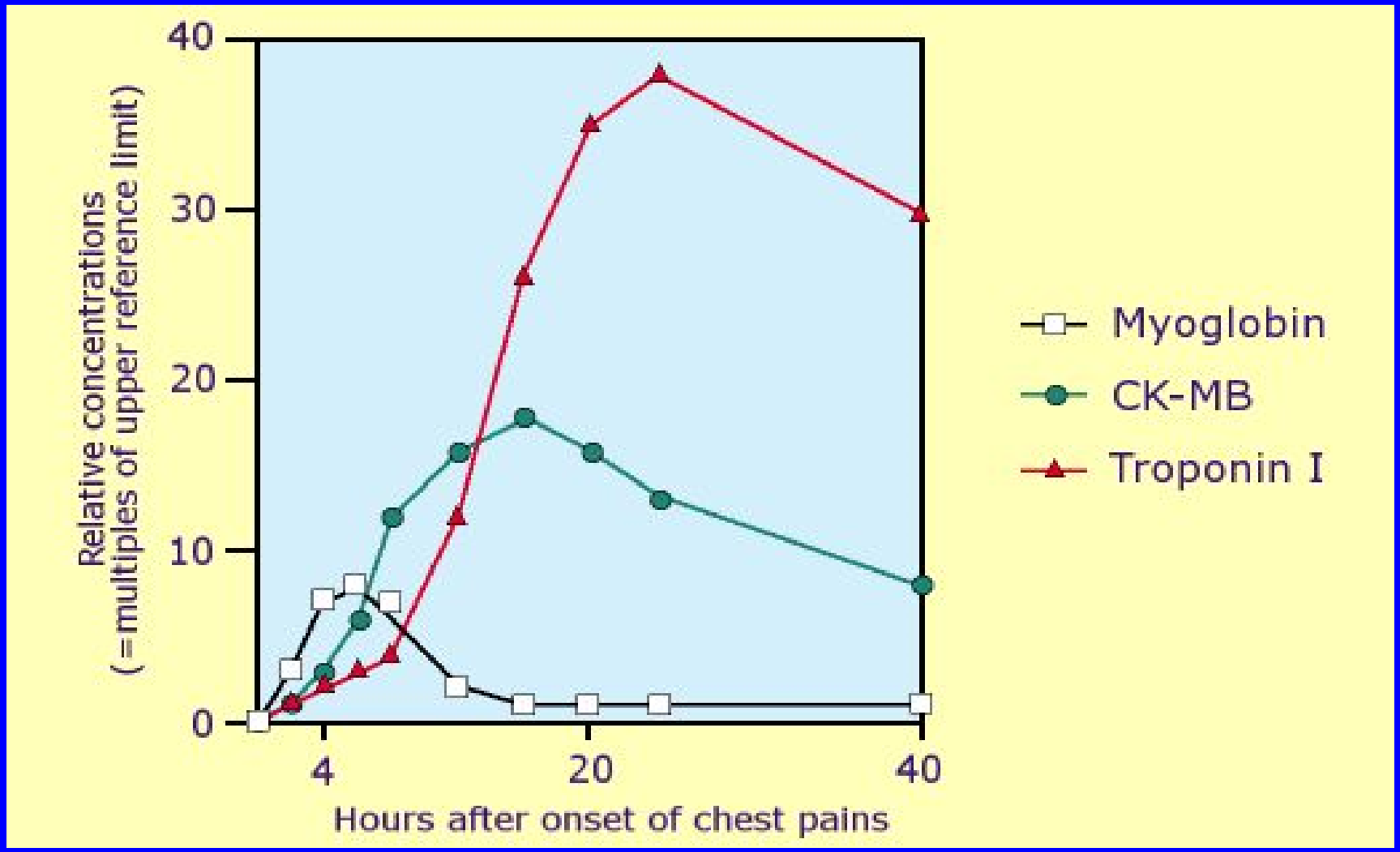
Troponin

- Troponin I : binds to actin and inhibits actin-myosin interaction
- Troponin C : responsive to changes to intracellular calcium (Not used)
- Troponin T : Binds the troponin to tropomyosin

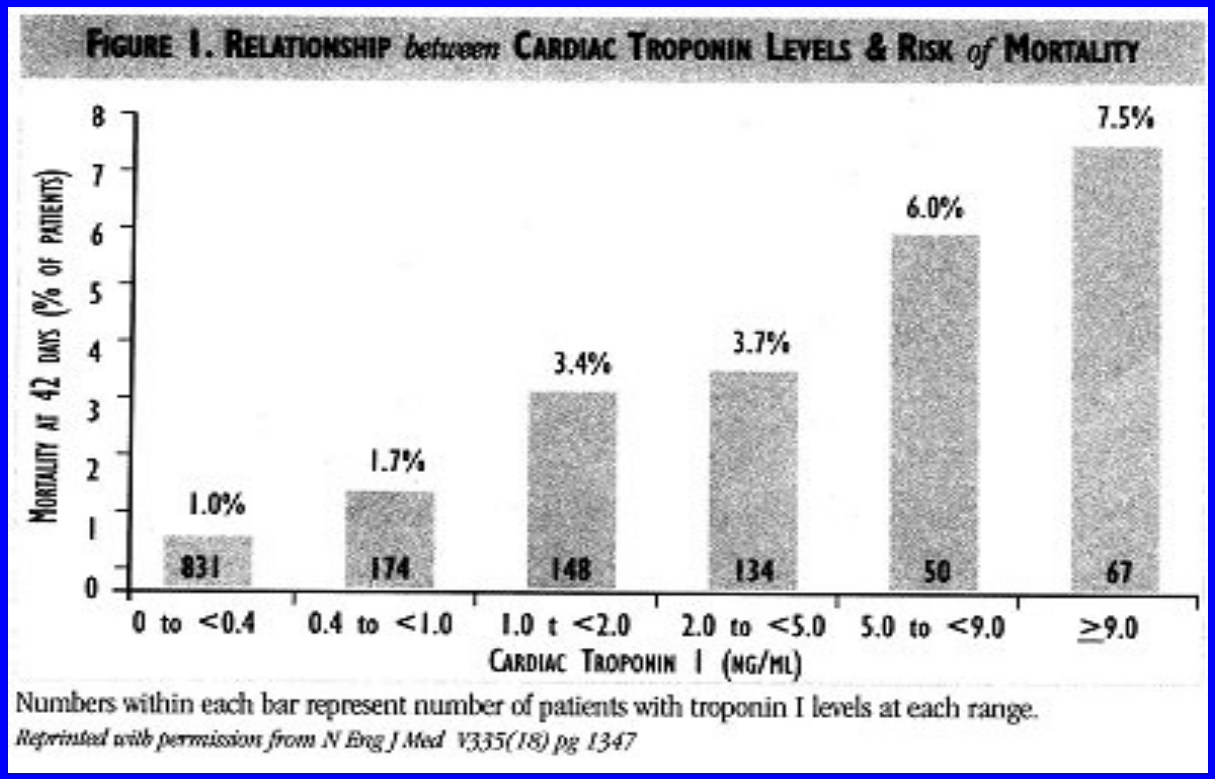
I and T are cardiac specific. C is not

Troponin I & T

- Specific for cardiac injury
- Most sensitive when you take the diagnostic window out to 24 hours
- It is not an earlier marker!
  - The majority of Troponins are not elevated in the first few hours
- It remains elevated for many days!
- When elevated is a marker for increased risk with non-ST elevation Acute Coronary Syndrome


40
30
20
10
0
Relative concentrations
(=multiples of upper reference limit)
4
20
40
Hours after onset of chest pains
Myoglobin
CK-MB
Troponin I

Troponin relates to Risk of Mortality…….



Numbers within each bar represent number of patients with troponin I levels at each range.

Reprinted with permission from N Eng J Med V335(18) pg 1347

In the first few hours you are on your own with your history, exam and ECG!

Diagnostic and Treatment Measures for Patients with ST Elevation or Bundle Branch Block

- Initial
  - Monitoring, targeted H&P, Start IV, ECG,CXR
- General
  - ASA 160-325 Chew and swallow, SL NTG, O2 adequate analgesia
- Specific Treatment
  - Reperfusion Lytic or Angioplasty
  - Heparin, NTG-IV, Beta Blocker

SHould We Emergently Revascularize **O**ccluded Coronaries of **C**ardiogenic Shoc**K**

SHOCK

n=152 early revascularization 150 medical stabilization with 86% in both groups getting an IABP (Intraaortic Balloon Pump)

30 day mortality

Early revascularization 46.7%

Medical stabilization 56%

p=0.11

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Cardiogenic Shock Complicating Acute Myocardial Infarction—Etiologies, Management and Outcome: A Report from the SHOCK Trial Registry

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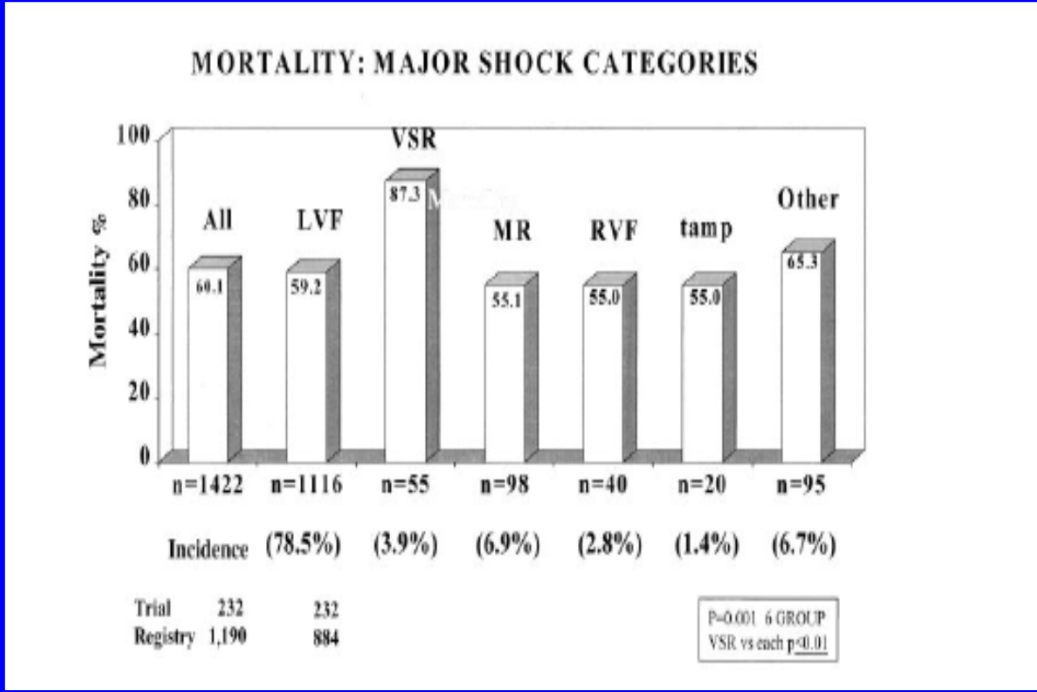
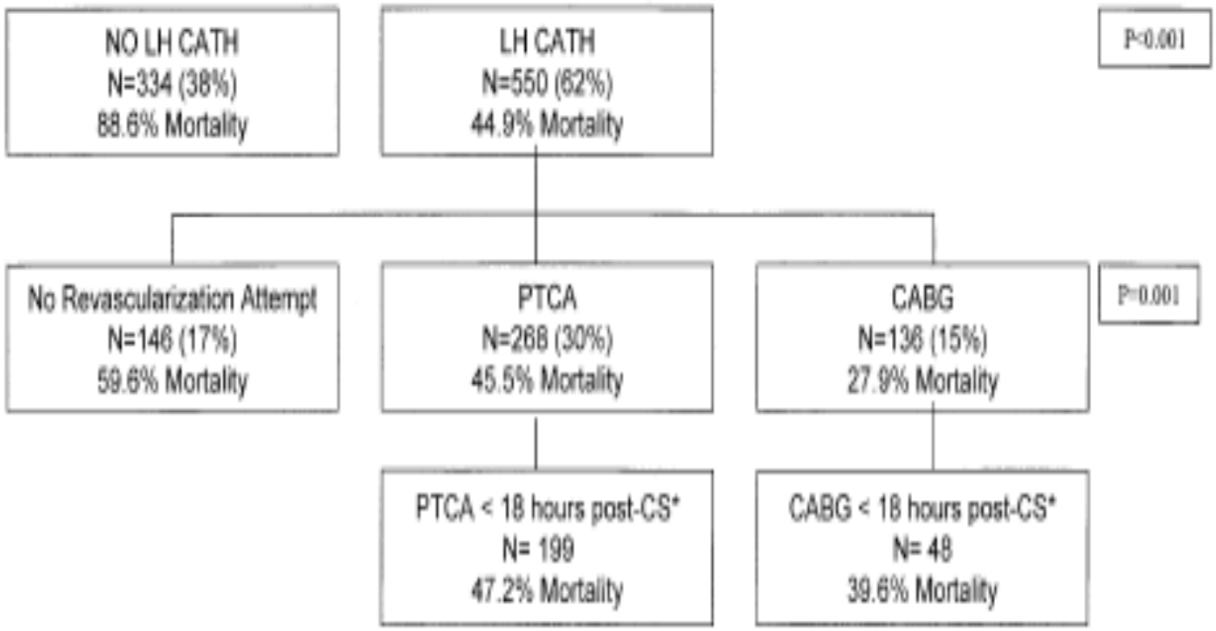
New York, New York; Vancouver and Edmonton, Canada; Watertown, Massachusetts; Liege, Belgium; Auckland, New Zealand



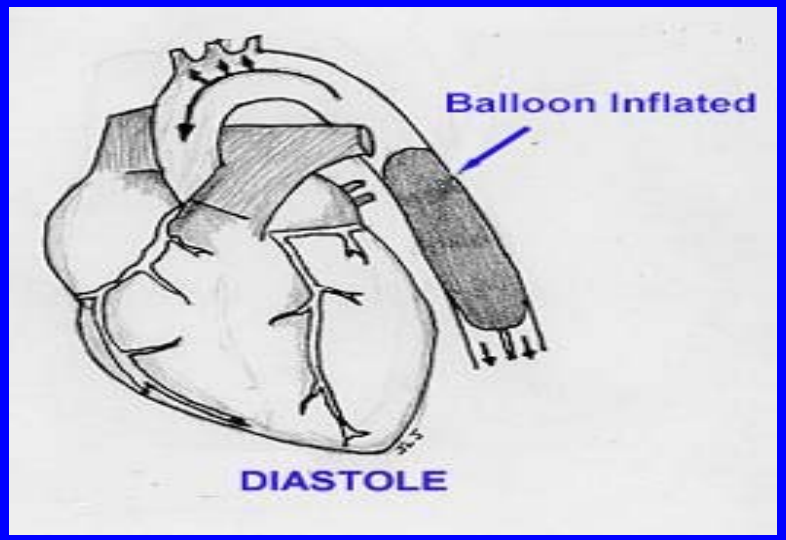
MORTALITY BY REVASCULARIZATION STATUS



Acute MI + Shock =

IABP

Equipment



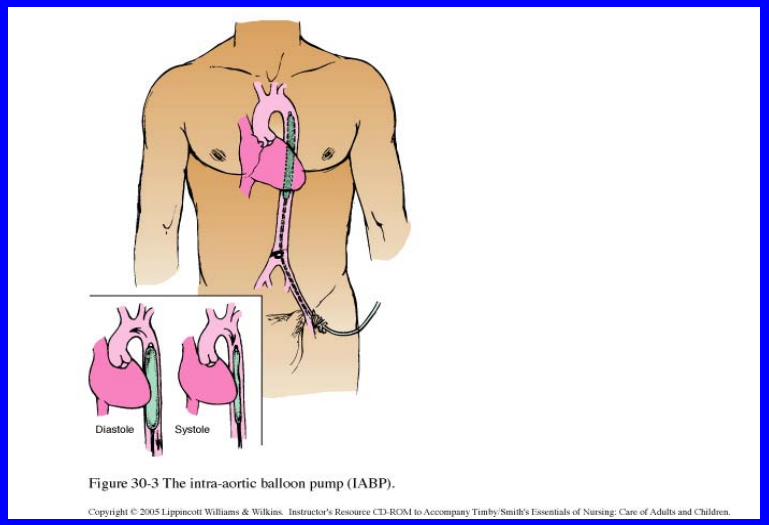


Figure 30-3 The intra-aortic balloon pump (IABP).

- Single-chambered balloon
  - Assembled on a 12 Fr double-lumen catheter
  - 1 lumen opens into the balloon and is used to deliver gas (CO2 or He)
  - Other lumen opens at the catheter tip and is used to monitor aortic pressure
  - When inflated, it displaces blood volume retrograde to the aortic arch and antegrade, perfusing areas distal to the balloon

Hemodynamics

- Improvement is usually seen within the first hour or two
  - Increased MAP
  - Increased coronary/peripheral perfusion
  - Decreased mental confusion
  - Increased urinary flow
  - Increased CO
  - Decreased PAP
  - Decreased PCWP
- Optimal duration hasn’t been established…same say no longer than 48 hours use

Complications

- Aortic dissection
- Perforation of the common iliac artery
- Thrombus
- Sepsis
- Vascular insufficiency of the catheterized limb (most common)

First 24 hours

- Continuous ECG monitoring
- Limit Activities for at least 12 hours
- Have available but not routinely used:
  - Atropine Lidocaine Epinephrine
  - Defibrillator Transcutaneous pacing patches
- Use: Heparin, Aspirin, IV Nitroglycerin
- Beta Blocker, ACE Inhibitor

Routine Measures

Class I

Oxygen

1. Supplemental oxygen should be administered to patients with arterial oxygen desaturation (SaO_2 less than 90%).

(Level of Evidence: B)

Analgesia

1. Morphine sulfate (2 to 4 mg IV with increments of 2-8 mg IV repeated at 5-15 minute intervals) is the analgesic of choice for management of pain associated with STEMI.

(Level of Evidence: C)

Antman et al. *JACC* 2004;44:679.

Routine Measures

Nitroglycerin

Class I

. Patients with ongoing ischemic discomfort should receive sublingual nitroglycerin (0.4 mg) every 5 minutes for a total of 3 doses, after which an assessment should be made about the need for intravenous nitroglycerin. *(Level of Evidence:* C)

2. Intravenous nitroglycerin is indicated for relief of ongoing ischemic discomfort, control of hypertension, or management of pulmonary congestion. *(Level of Evidence:* C)

Class III

1. Nitrates should not be administered to patients with systolic blood pressure less than 90 mm Hg or greater than or equal to 30 mm Hg below baseline, severe bradycardia (less than 50 bpm), tachycardia (more than 100 bpm), or suspected RV infarction. *(Level of Evidence:* C)

2. Nitrates should not be administered to patients who have received a phosphodiesterase inhibitor for erectile dysfunction within the last 24 hours (48 hours for tadalafil). *(Level of Evidence:* B)

Antman et al. *JACC* 2004;44:679.

Routine Measures

Aspirin

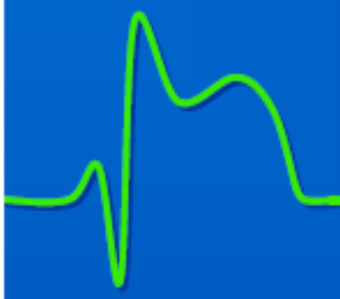
Class I

1. Aspirin should be chewed by patients who have not taken aspirin before presentation with STEMI. The initial dose should be 162mg (*Level of Evidence: A) to 325 mg (Level of Evidence: C*). Although some trials of have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non-enteric-coated aspirin formulations.

Antman et al. *JACC* 2004;44:680.

CAPRIE
Lancet 1996;348:1329-1339

- Clopidogrel versus Aspirin in Patients at Risk of Ischemic Events
  - Relative risk reduction in vascular death, MI or stroke of 8.7% in favor of clopidogrel
  - 0.10% incidence of a significant reduction in neutrophils
  - TTP has not been reported with clopidogrel like has been reported with Ticlopidine



Routine Measures

ß-blocking agents

Class I

1. **Oral beta-blocker** therapy should be administered **promptly** to those patients without a contraindication, irrespective of concomitant fibrinolytic therapy or performance of primary PCI. *(Level of Evidence:* A)

Class IIa

1. It is **reasonable to administer IV beta-blockers** promptly to STEMI patients without contraindications, **especially if a tachyarrhythmia or hypertension** is present. *(Level of Evidence:* B)

Antman et al. *JACC* 2004;44:680. [57]

Relative contraindications

- HR < 60
- Systolic BP < 100
- Moderate or severe LV failure
- Signs of peripheral hypoperfusion
- PR > .24
- 2nd or 3rd degree AV block
- Severe COPD
- History of asthma
- Severe PVD
- Insulin-dependent diabetes

Inhibition of Renin-Angiotensin-Aldosterone System

Class I

1. An angiotensin converting enzyme (ACE) inhibitor should be administered orally within the first 24 hours of STEMI to patients with anterior infarction, pulmonary congestion, or LVEF less than 0.40, in the absence of hypotension (systolic blood pressure less than 100 mm Hg or less than 30 mm Hg below baseline) or known contraindications to that Class of medications. *(Level of Evidence:* A)

2. An angiotensin receptor blocker (ARB) should be administered to STEMI patients who are intolerant of ACE inhibitors and who have either clinical or radiological signs of heart failure or LVEF less than 0.40. Valsartan and candesartan have established efficacy for this recommendation. *(Level of Evidence:* C)

Class IIa

1. An ACE inhibitor administered orally within the first 24 hours of STEMI can be useful in patients without anterior infarction, pulmonary congestion, or LVEF less than 0.40 in the absence of hypotension (systolic blood pressure less than 100 mm Hg or less than 30 mm Hg below baseline) or known contraindications to that class of medications. The expected treatment benefit in such patients is less (5 lives saved per 1000 patients treated) than for patients with LV dysfunction. *(Level of Evidence:* B)

Antman et al. *JACC* 2004;44:690.

Non Steroidal anti-inflammatory drugs

- Pts routinely taking these drugs , non-selective as well as cyclooxygenase-2 selective agents,before STEMI should have these agents discontinued at the time of presentation with STEMI
- Non-steroidals increase risk of mortality,reinfarction,hypertension,heart failure, and myocardial rupture with their continued use.

Divergent Effects of Hormone Therapy on Serum Markers of Inflammation in Postmenopausal Women With Coronary Artery Disease on Appropriate Medical Management
JACC 2000;36:1797-1802

- HRT has reduced the risk of coronary heart disease events in observational studies of healthy postmenopausal women
  - favorable effects are seen on lipoproteins, increased nitric oxide bioactivity, enhanced fibrinolysis and reduced levels of soluble cell adhesion molecules.
  - Unfavorable increase in C-reactive protein
- Studies have shown an increased risk early (first year) and benefit late (year 4-5) like the HERS trial and Nurses Health Study
- Inflammatory cells and activated smooth muscle cells secrete matrix metalloproteinases and weaken the fibrous cap leading to rupture
- Increased levels of MMP-2(gelatinase A) and MMP-9(gelatinase B) are seen in serum of women with CAD

NEW: STRICT GLUCOSE CONTROL DURING STEMI

Class I

1. An **insulin infusion** to normalize blood glucose is recommended for patients with STEMI and complicated courses. *(Level of Evidence:* B)

Class IIa

1. During the acute phase (first 24 to 48 hours) of the management of STEMI in patients with hyperglycemia, it is **reasonable to administer an insulin infusion to normalize blood glucose, even in patients with an uncomplicated course.** *(Level of Evidence:* B)

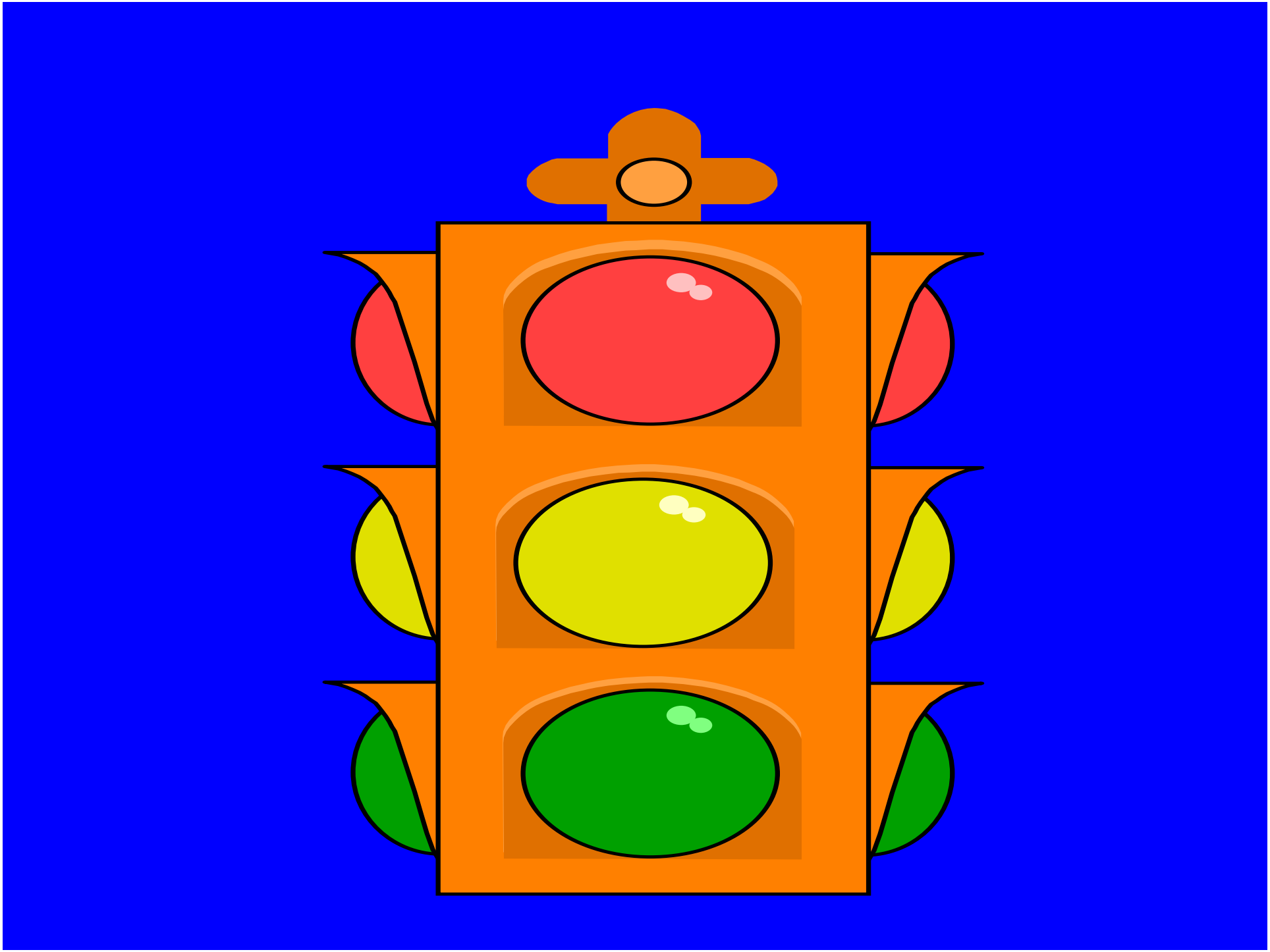
Antman et al. *JACC* 2004;44:690.

Calcium Channel Blockers

Class IIa

1. It is reasonable to give verapamil or diltiazem to patients in whom **beta-blockers are ineffective or contraindicated** (eg, bronchospastic disease) for relief of ongoing ischemia or control of a rapid ventricular response with **atrial fibrillation** or flutter after STEMI in the absence of CHF, LV dysfunction, or atrioventricular (AV) block. *(Level of Evidence:* C)

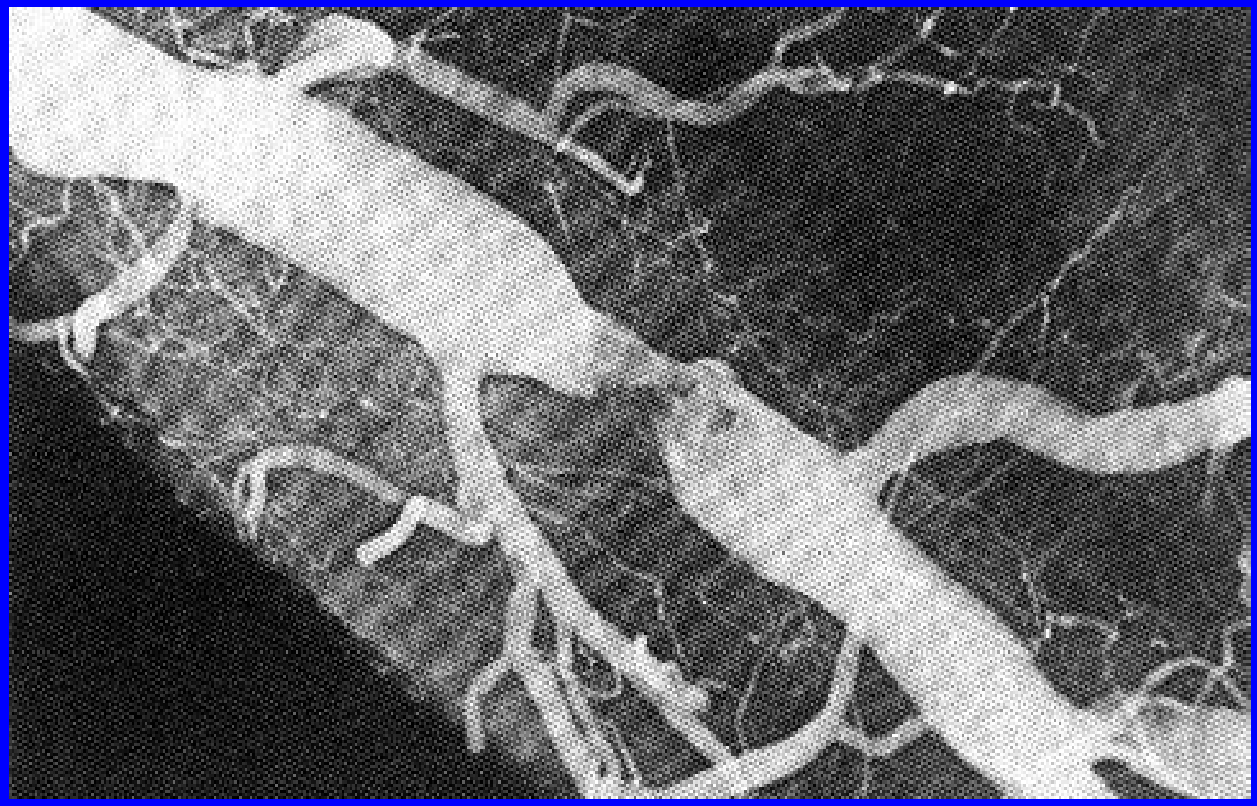
Antman et al. *JACC* 2004;44:691.



Patients without ST Elevation

- A mixed bag with
  - Non-cardiac
  - Unstable without necrosis
  - With necrosis “Small MI’s”
  - Posterior MI
  - Patients with high risk multivessel CAD and LV dysfunction

What Do You Think Is Going On In There?



The pathology is the acute disruption of an atherosclerotic plaque

- The difference is that there is adequate perfusion (Subtotal occlusion or collaterals) as compared to AMI where there is adequate tissue perfusion to maintain cell life.

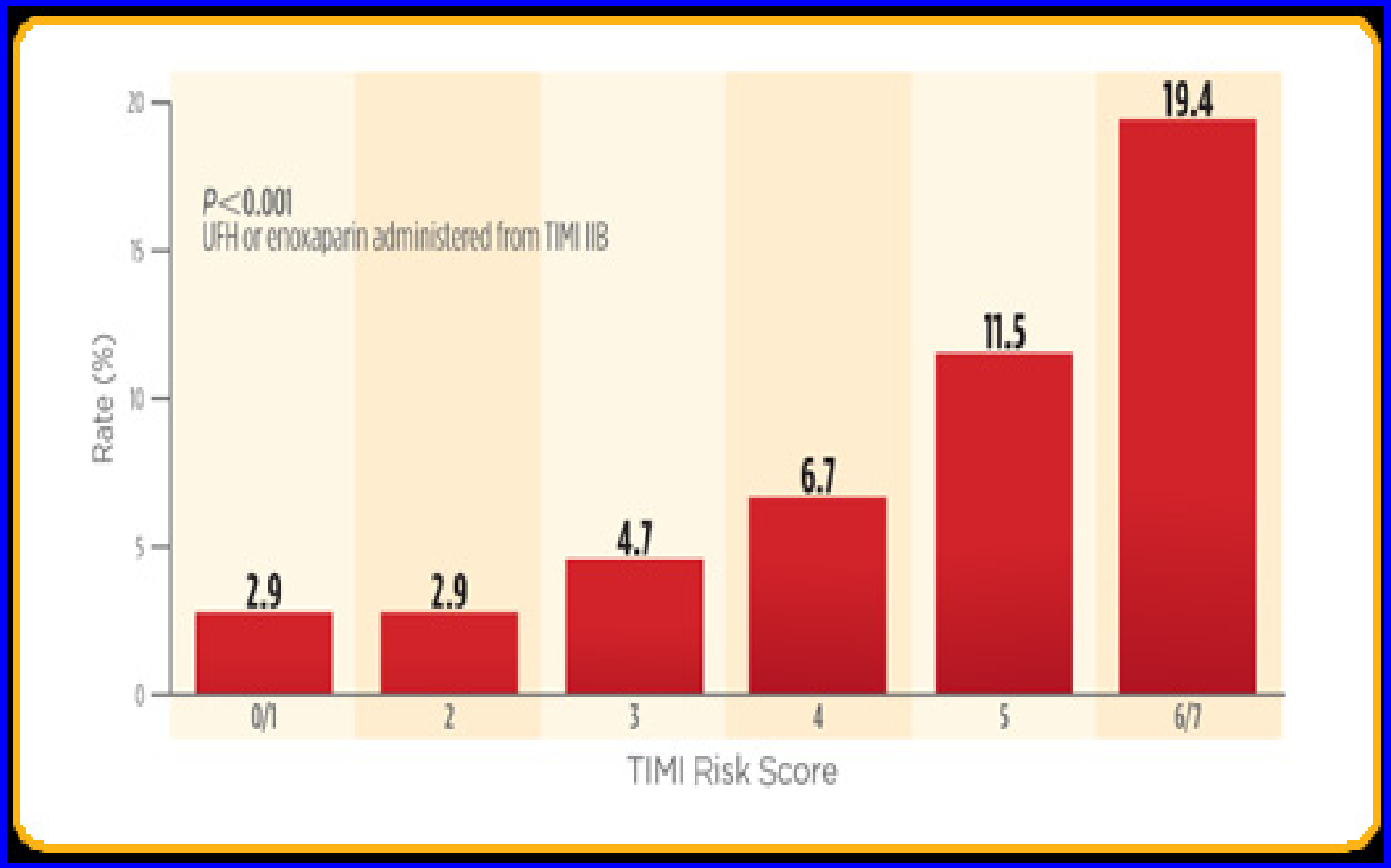
TIMI Risk Score

- Age>65
- >3 CAD risk factors (FH,HTN,Chol,DM,active smoker)
- Known CAD (stenosis > 50%)
- ASA use in past 7 days
- Recent (<24h) severe angina
- Increased cardiac markers
- ST deviation > 0.05mV

Risk score =Total pts (0-7)

Antman EM JAMA 2000;284:835

Rate of Death or MI by 14 days v. TIMI risk score



Antman EM JAMA 2000;284:835

Cornerstone of Therapy

Aspirin

Heparin

Nitrates for recurrent angina

Acute Coronary Syndromes

- Antithrombotic Therapy
  - Low-molecular-weight heparin versus unfractionated
    - ESSENCE Trial (Benefit)
      - NEJM 1997;337: 447-452
    - JACC 26: 313-318, 1995 (Better than placebo)
    - FRIC
      - Eur Heart J 1996;17 Suppl: 306 (Equal)
    - TIMI 11A
      - JACC 1997 29: 1474-1482 (Benefit)

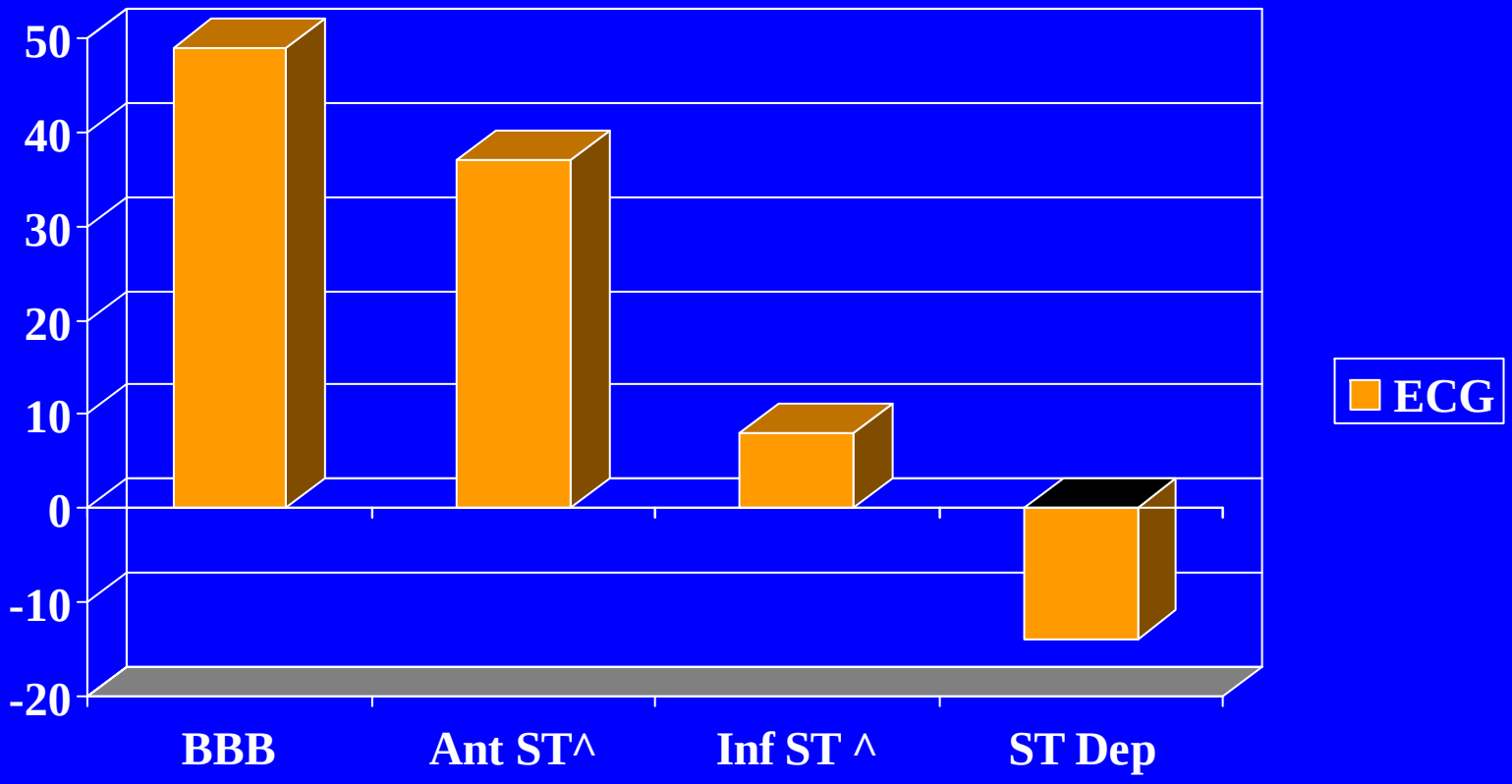
Glycoprotein IIB/IIIa inhibitors

- Abciximab (REOPRO)
  - CAPTURE Lancet 1997;349:1429-1435
- Eptifibatide INTEGRELIN
  - PURSUIT NEJM 1998;339:436-443
- Tirofiban AGGRASTAT
  - PRISM-PLUS NEJM 1998;338: 1488-1497

Non-ST Elevation Cohort

- Early Intervention?
  - TIMI - IIIB
    - n=1473 randomized
    - Death, MI or + exercise test at 42 day
      - 16.2% versus 18.1%
    - By 42 days 64% of the conservative group had been cathed due to spontaneous or induced ischemia with 55% being done before discharge.

ECG and Mortality Effect
Lives saved Per Thousand



Early Intervention
VANQUISH

- NEJM 1998;338:1785-1192
  - n=920 with no Q waves and no complications within 24-72 hours
  - Aggressive therapy with a cath and revascularization of significant lesions.
  - Follow-up 12-44 months had a 28% rate of events
    - combined endpoint death or nonfatal MI were no different in either group at an average of 23 months (138 invasive;123 conservative)
    - higher death rate at hospital discharge 21 versus 6 (p=0.007) and at one year 58 versus 36 (p=0.025)

FRISC Trial II
March 1999 ACC

- Early invasive strategy + LMWH dalteparin may reduce early events in patients with unstable coronary artery disease.
- N 2267 open acute phase received 120 IU/kg q 12 hours for 5-7 days then double blind n = 2015 versus placebo for 3 months.

	45 days	90 days	6 months
	p=0.0003	No diff	p=0.045
Active	3.7%		Invasive 9.5%
Placebo	6.5%		Non-Inv. 12%

Preparation for discharge

- Submaximal exercise test (5 METS) at 4-7 days Class I
- Predischarge EF (Echo, Nuclear)
- Decision about need for coumadin made based upon size/site of infarct and echo
- At 2 month follow up visit ,repeat the Echo and if EF < 30% ,evidence from MADIT trial favors AICD placement

Long Term

- Don’t smoke
- Symptom limited treadmill at 10-14 days
- Aspirin & Beta-Blockers & Statin“forever”
- ACE Inhibitors for minimum of 6 weeks and then look at LV
- AHA Step II diet (<7% sat fat & < 200mg Chol)
- LDL < 100 , High dose statin supported by PROVE IT TIMI 22 trial . High risk patients can have LDL of < 70 as target
- 30 minutes exercise 5 times/week