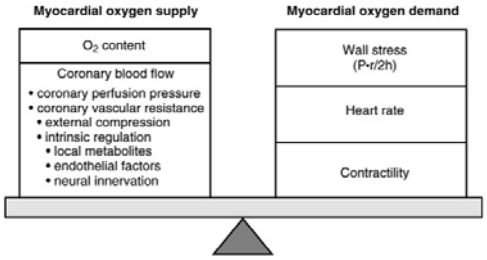
Coronary Artery Pathophysiology ACS / AMI

LeRoy E. Rabbani, MD
Director, Cardiac Inpatient Services
Director, Cardiac Intensive Care Unit
Professor of Clinical Medicine

Major Determinants of Myocardial Oxygen Supply and Demand



MYOCARDIAL OXYGEN DEMAND

1. Wall Tension
2. Heart Rate
3. Contractility (Inotropic State)

WALL TENSION

Wall Tension $\alpha \frac{P \cdot r}{h}$ Formula of Laplace

P = LV Systolic Pressure
r = LV Radius
h = LV Wall Thickness

MYOCARDIAL OXYGEN SUPPLY

1. Diastolic Perfusion Pressure
2. Coronary Vascular Ressitance
3. Oxygen Carrying Capacity

INTRINSIC CONTROL OF CORONARY TONE

1. Heart in basal state consumes as much oxygen as it can (2-3 times as much as most organs)
2. Heart cannot increase oxygen extraction on demand
3. Any additional oxygen requirement must be provided by an increase in blood flow (autoregulation of coronary vascular tone)

CORONARY BLOOD FLOW REGULATION

$$Q \propto \frac{P}{R}$$

Q = Coronary Artery Blood Flow

P = Perfusion Pressure

R = Coronary Vascular Resistance

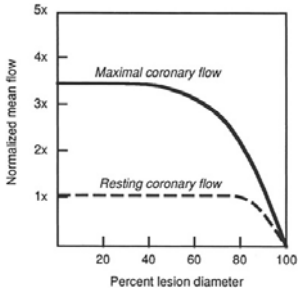
CORONARY VASCULAR RESISTANCE

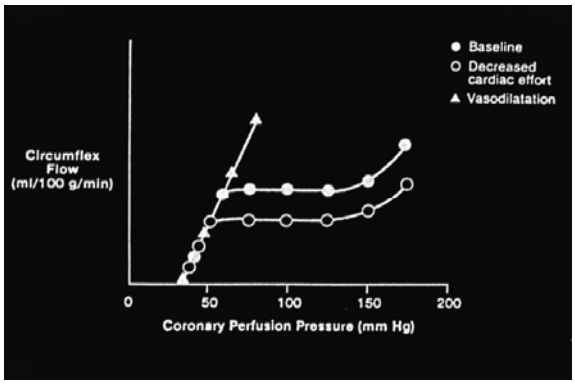
1. External Compression
2. Intrinsic Regulation
   a) Local Metabolites
   b) Endothelial Factors
   c) Neural Innervation

EXTERNAL COMPRESSION OF CORONARIES

1. Greatest in systole
2. Directly related to intramyocardial pressure
3. Subendocardium, adjacent to high intraventricular pressure, is most vulnerable to ischemic damage

Resting and Maximal Coronary Blood Flow





AUTOREGULATION OF CORONARY VASCULAR TONE

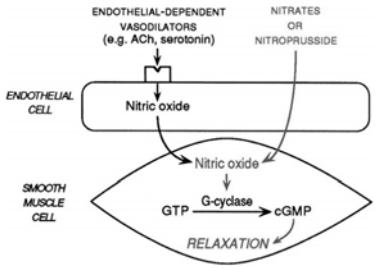
I. Local Metabolites
   a) Oxygen - Vasoconstrictor
   b) Adenosine - Vasodilator
   c) Lactate
   d) Prostaglandins
   e) Hydrogen ions

AUTOREGULATION OF CORONARY VASCULAR TONE

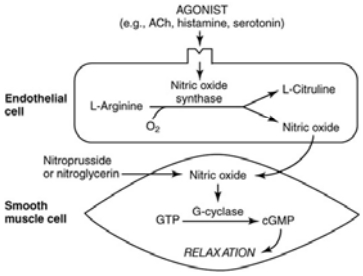
II. Endothelial Factors

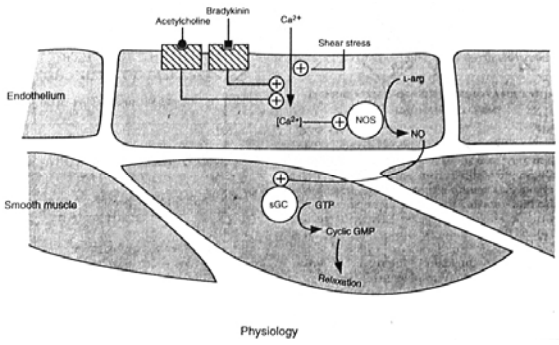
1. Endothelial-dependent vasodilators: (ATP, ADP, bradykinin, histamine, acetylcholine)
2. EDRF (nitric oxide free radical) stimulates SMC guanylate cyclase activity.
3. Increased cGMP mediates vasodilatation through inhibition of calcium release.

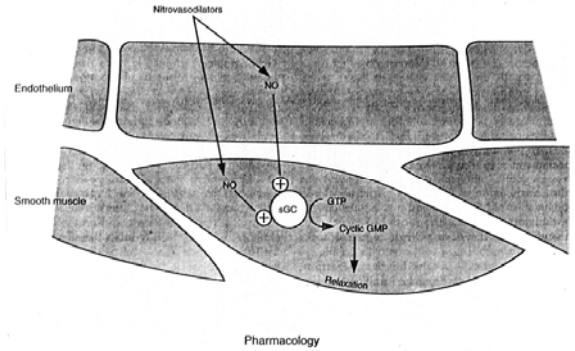
Organic Nitrates



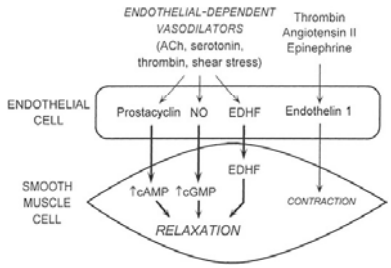
EDRF







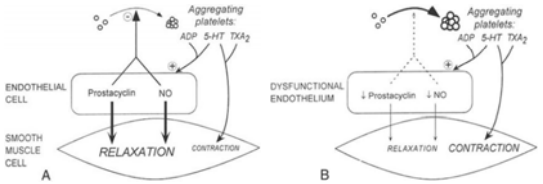
Endothelium-derived vasoactive substances and their regulators.



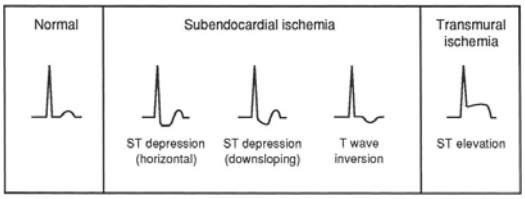
The interaction between platelets and endothelial cells

Normal Endothelium

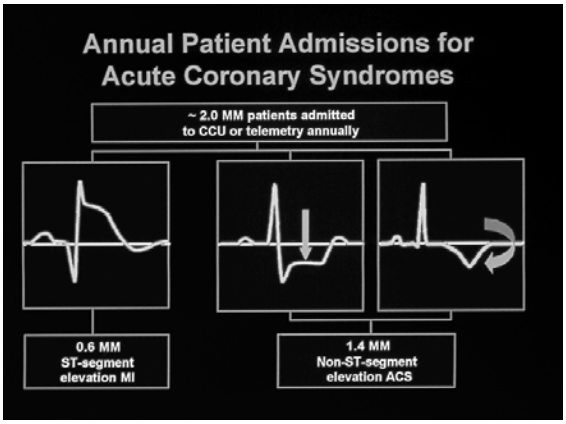
Dysfunctional Endothelium



Common transient ECG abnormalities during ischemia

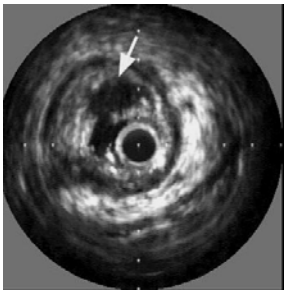


Annual Patient Admissions for Acute Coronary Syndromes



ST-segment Depression: Indicates Increased Risk for Long-term Mortality

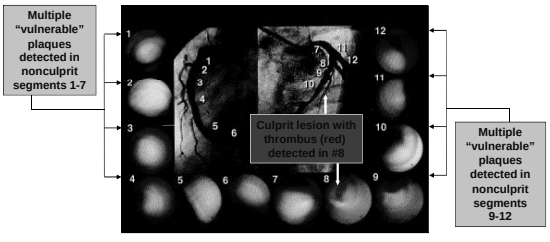
IVUS of Ruptured Plaque



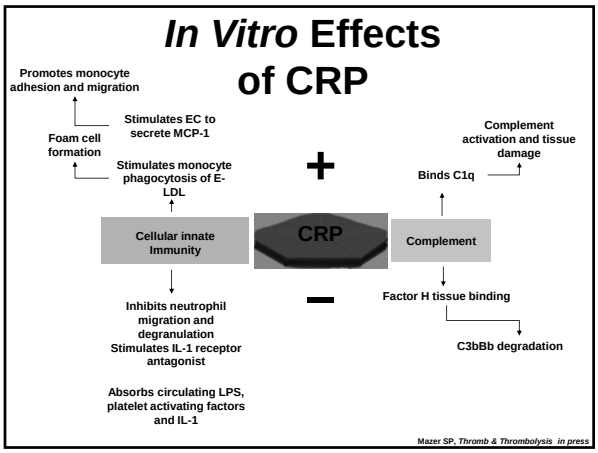
Ziada K, Bhatt DL. *Essential Concepts in Cardiovascular Intervention*. ReMEDICA Publishing; 2004.

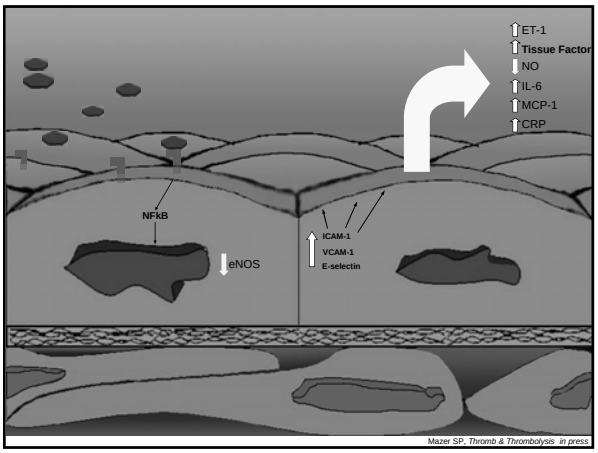
Evidence of Multiple "Vulnerable" Plaques in ACS

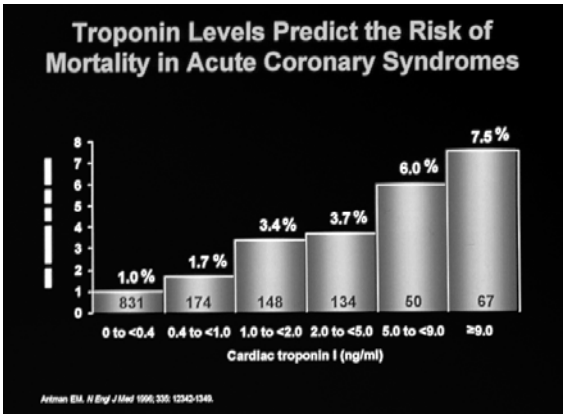
Angiographic and angioscopic images of a 58-year-old man with anterior myocardial infarction



Reprinted with permission from Asakura M, et al. *J Am Coll Cardiol.* 2001;37:1284-1288.







Evolution of serum biomarkers in acute MI

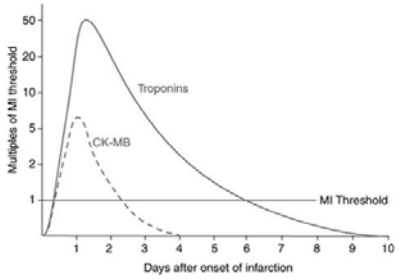


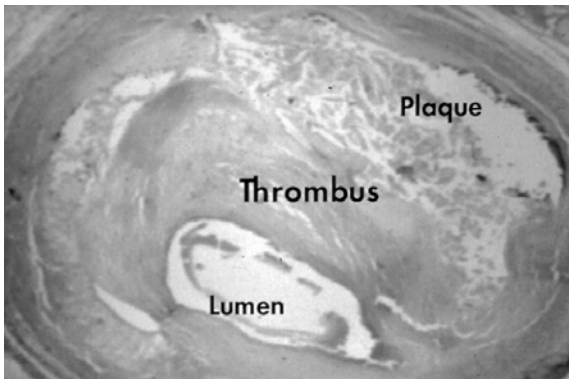
TABLE 4. Canadian Cardiovascular Society Angina Classification

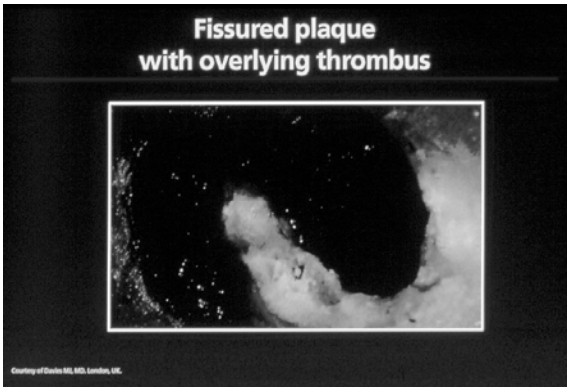
Class	Activity Evoking Angina	Limits to Normal Activity
I	Prolonged exertion	None
II	Walking >2 blocks	Slight
III	Walking <2 blocks	Marked
IV	Minimal or rest	Severe

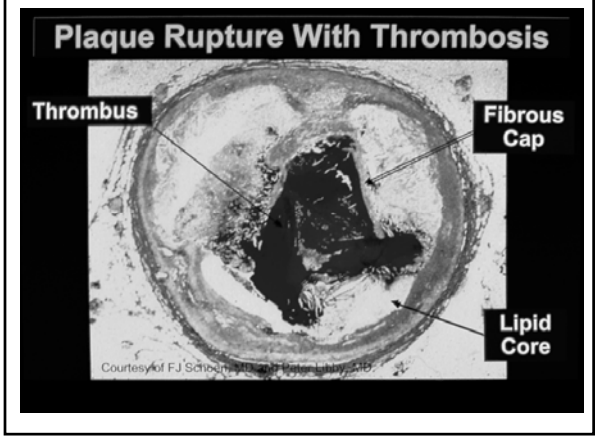
Adapted from: Campeau L. Grading of angina pectoris [letter]. *Circulation*. 1976;54:522-523. Copyright 1976, American Heart Association, Inc. Used with permission.

TABLE 1. Unstable Angina Presentations

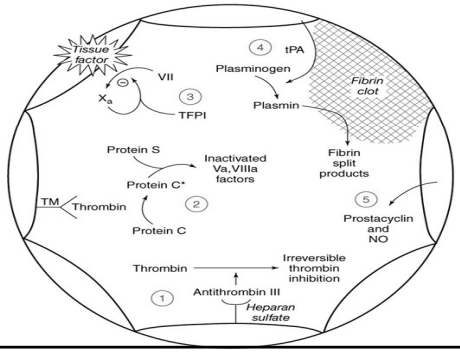
- Rest angina within 1 week of presentation
- New onset angina of Canadian Cardiovascular Society Classification (CCSC) class III or IV within 2 months of presentation (see Table 4)
- Angina increasing in CCSC class to at least CCSC III or IV
- Variant angina
- Non–Q-wave myocardial infarction
- Post–myocardial infarction angina (>24 hours)



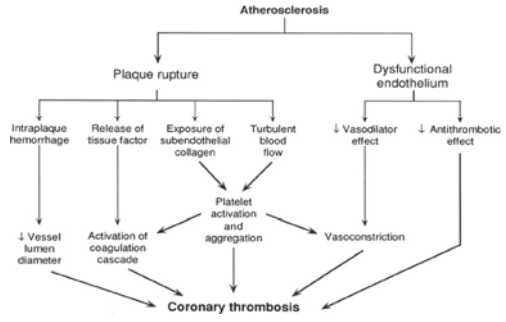




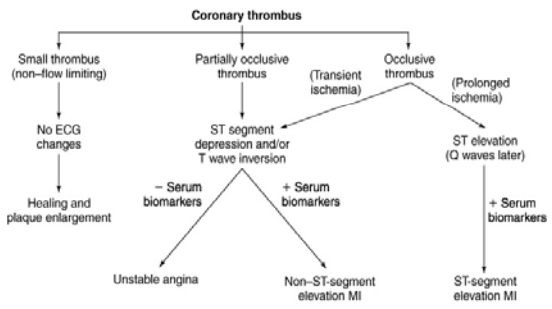
Endogenous Protective Mechanisms



Mechanisms of Coronary thrombus formation



Consequences of coronary thrombosis



Mechanisms of cell death in myocardial infarction

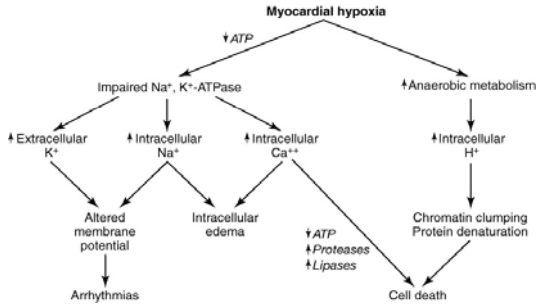


Table 1. Angiographic Evolution Acute Coronary Syndromes

	Initial Stenosis		
Syndrome	<50%	50% to 70%	>70%
Unstable angina, % (n=25)[49]	72	16	12
Myocardial infarction, %			
n=23[41]	48	30	22
n=41[50]	66	31	3
n=92[52]	78	9	13
n=39[51]	59	15	26
Average	65	20	15

*Ambrose et al.[49] †Ambrose et al.[41] ‡Little et al.[50] §Giroud et al.[52] ||Nobuyoshi et al.[51]

Table 1. Biochemical Analysis of Protein and Extracellular Lipid Content of Ulcerated and Intact Human Aortic Plaque Caps

	Ulcerated Plaques (n = 24)	Nonulcerated Plaques (n = 31)
Total protein (% dry weight)	54.8 ± 1.2	57.2 ± 2.2
Collagen	35.4 ± 8.4	56.8 ± 1.4*
Elastin	0.87 ± 0.87	1.17 ± 0.31
Glycosoaminoglycan	0.9 ± 0.20	1.9 ± 0.2*
Extracellular lipid (% plaque volume)	54.9 ± 3.8	22.1 ± 2.4†

*p < 0.05. †p < 0.001. Values presented are mean value ± SEM. Modified, with permission, from Davies et al. (30).

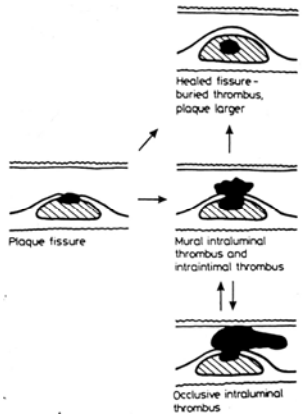


Table 2. Cellular Content of Ulcerated and Intact Human Aortic Plaque Caps

	Ulcerated Plaques (n = 24)	Nonulcerated Plaques (n = 31)
Density of SMC	65.2 ± 13.2	174.0 ± 11.9*
Density of MO	122.1 ± 13.3	62.2 ± 8.8*
SMC/MO ratio	1.2	5.8*

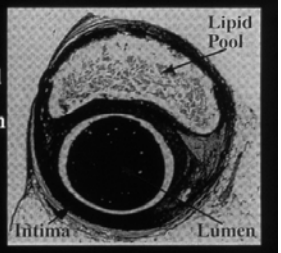
*p < 0.001. Values are presented as mean value (SEM). MO = monocytes; SMC = smooth muscle cells. Modified, with permission, from Davies et al. (30).

$$t = p \cdot r \qquad \sigma = \frac{p \cdot r}{h}$$

Figure 4. Circumferential tension on the fibrous cap of an atherosclerotic plaque containing a lipid pool **(hatched area)** is determined by the law of Laplace. This relates tension (t) to the intralumen pressure (p) and the lumen radius (r). The mean circumferential stress (σ) on the fibrous cap is related to circumferential tension and cap thickness (h).

Pathological View of Plaque Rupture

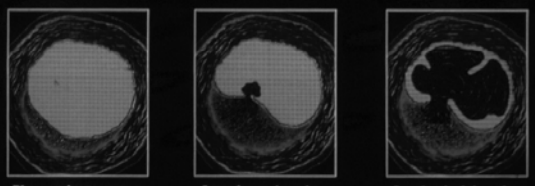
- Plaques with increased lipid content appear more prone to rupture, particularly when the lipid pool is localized eccentrically within the intima



Davies, MJ, et al., Circulation, 82(Suppl II); II38-II46, 1990

Plaque Rupture of Lipid-Rich Plaques

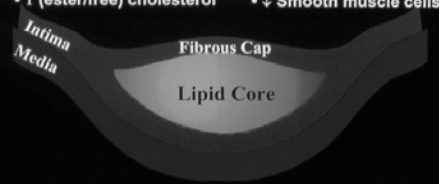
Mild-to-Moderate Lesions that Rupture are the Most Common Cause of Cardiac Events



Plaque prior to rupture | Intraplaque thrombus postrupture | Intraluminal thrombus postrupture

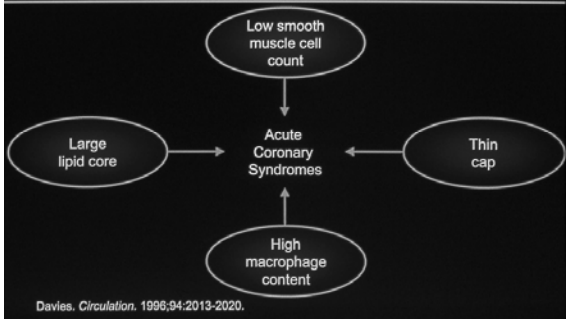
Dynamics of Atherosclerotic Plaque Stability

Factors increasing stress	Factors weakening the cap
• Thin fibrous cap	• ↓ Collagen synthesis
• Large lipid pool	• ↑ Collagen degradation
• Less stenotic lesions	• ↑ Macrophages, T cells
• ↑ (ester/free) cholesterol	• ↓ Smooth muscle cells

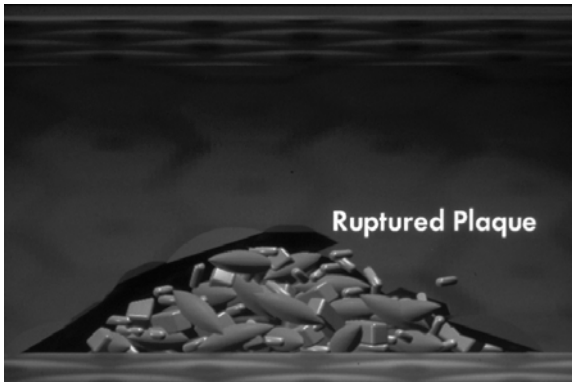


Reproduced with permission from Lee and Libby. *Arterioscler Thromb Vasc Biol.* 1997;17:1859.

Conditions Leading to Plaque Vulnerability

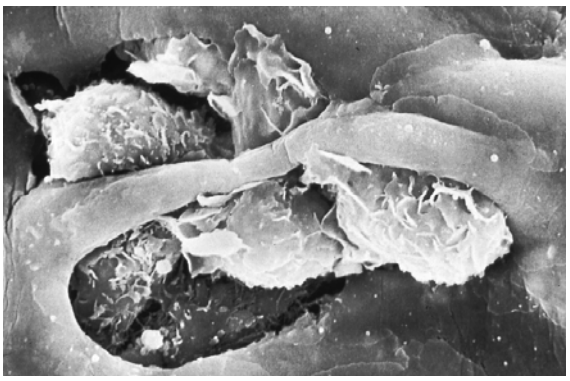


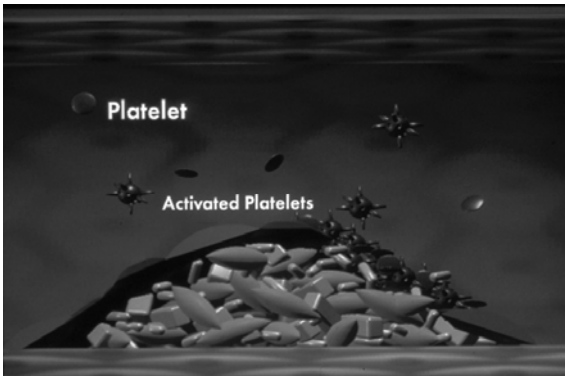
Davies. *Circulation.* 1996;94:2013-2020.

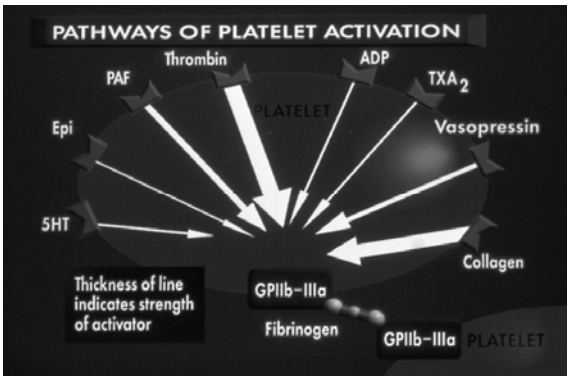


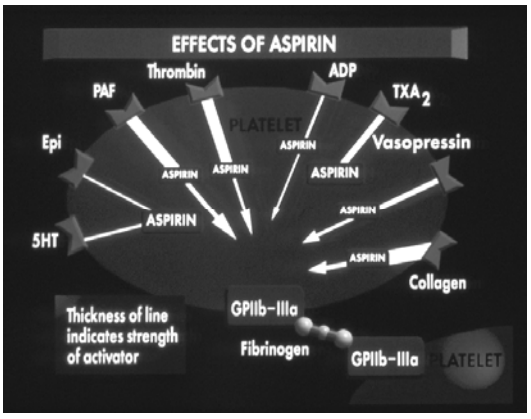
Macrophage Foam Cells

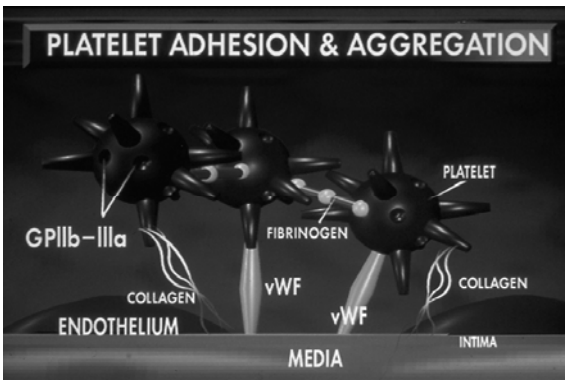
- Matrix Metalloproteinases
  - Collagenase
  - Stromelysin
  - Gelatinase
  - Elastase
- Tissue Factor
- CRP
- Myeloperoxidase

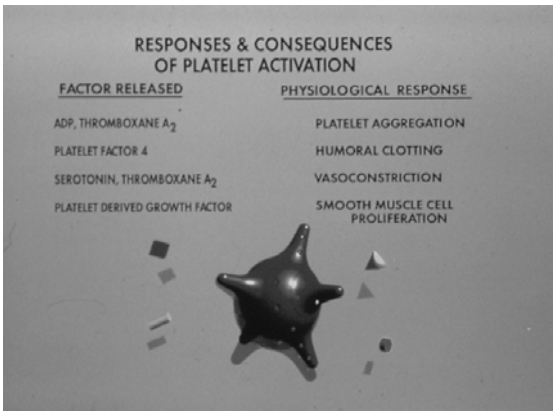


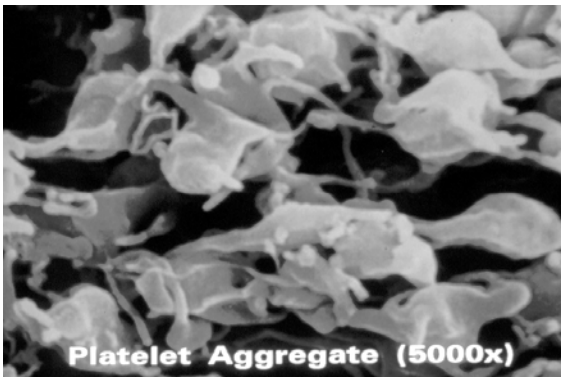


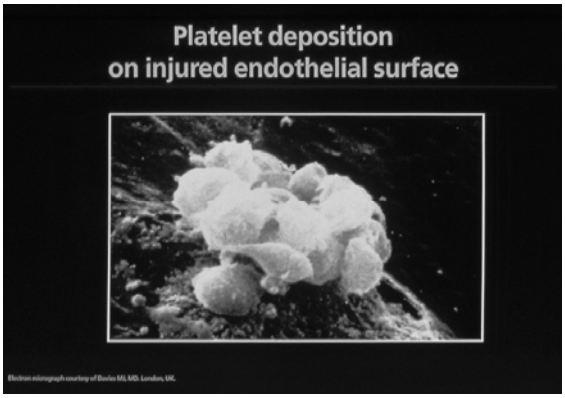


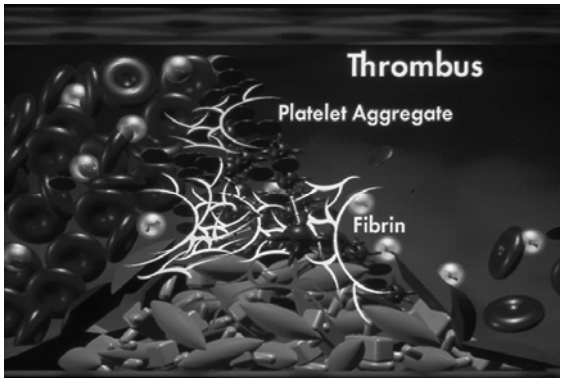


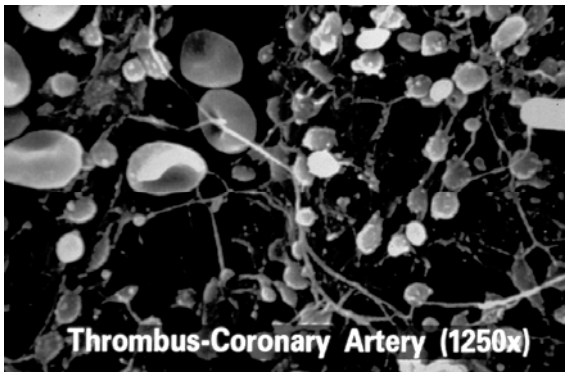


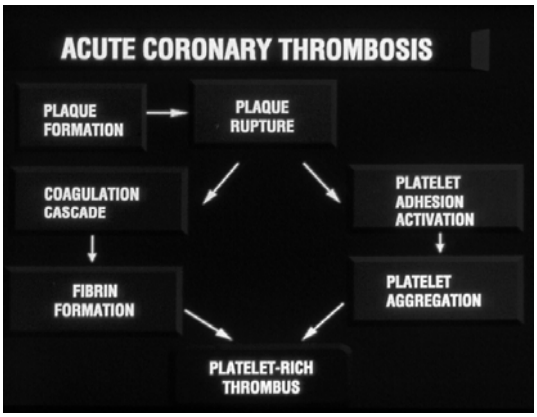

RESPONSES & CONSEQUENCES
OF PLATELET ACTIVATION
FACTOR RELEASED
PHYSIOLOGICAL RESPONSE
ADP, THROMBOXANE A_2
PLATELET AGGREGATION
PLATELET FACTOR 4
HUMORAL CLOTTING
SEROTONIN, THROMBOXANE A_2
VASOCONSTRICTION
PLATELET DERIVED GROWTH FACTOR
SMOOTH MUSCLE CELL PROLIFERATION


Platelet Aggregate (5000x)

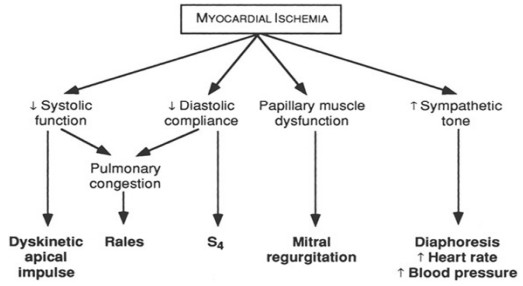

Platelet deposition
on injured endothelial surface


Thrombus
Platelet Aggregate
Fibrin


Thrombus-Coronary Artery (1250x)


ACUTE CORONARY THROMBOSIS
PLAQUE FORMATION
PLAQUE RUPTURE
COAGULATION CASCADE
PLATELET ADHESION ACTIVATION
FIBRIN FORMATION
PLATELET AGGREGATION
PLATELET-RICH THROMBUS

Pathophysiology of Physical Signs during Acute MI



ECG abnormalities in unstable angina and non-ST elevation MI

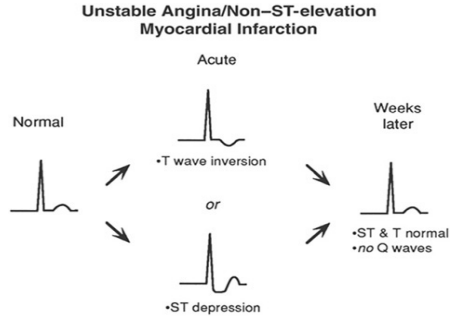
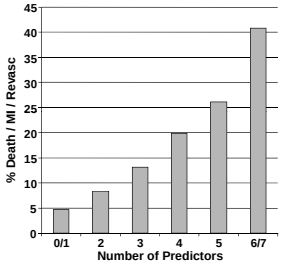


Figure 3: TIMI risk score for UA/NSTEMI – 7 independent predictors[3]

- Age ≥65 years
- ≥3 CAD Risk Factors (↑ chol, FHx, HTN, DM, smoking)
- Prior CAD (cath stenosis >50%)
- ASA in last 7 days
- ≥2 anginal events ≤24 hours
- ST deviation
- Elevated cardiac markers (CK-MB or troponin)

TIMI Risk Score for UA/NSTEMI: 7 Independent Predictors

1. **Age ≥65 y**
2. **≥3 CAD risk factors (high cholesterol, family history, hypertension, diabetes, smoking)**
3. **Prior coronary stenosis ≥50%**
4. **Aspirin in last 7 days**
5. **≥2 anginal events ≤24 h**
6. **ST-segment deviation**
7. **Elevated cardiac markers (CK-MB or troponin)**



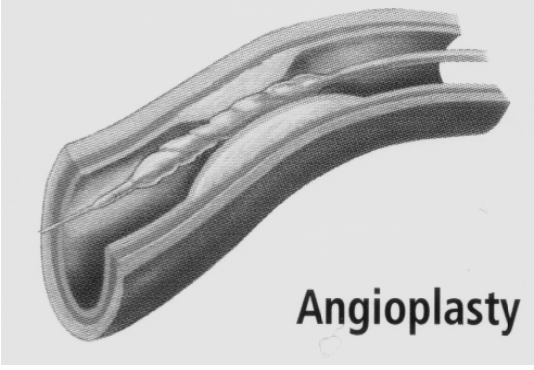
TIMI = Thrombolysis in Myocardial Infarction.
Antman EM, et al. *JAMA*. 2000;284:835-842.

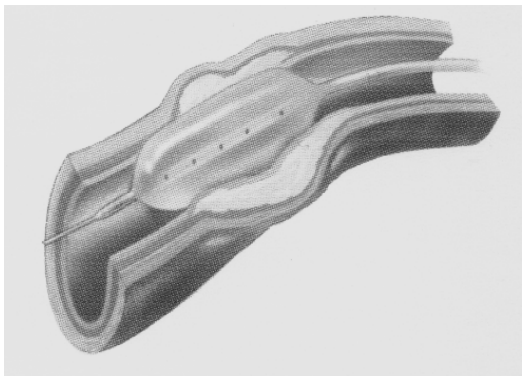
ACC/AHA Guidelines Recommendations: NSTE ACS Patients at High Risk of Death or MI

At least one of the following features must be present:

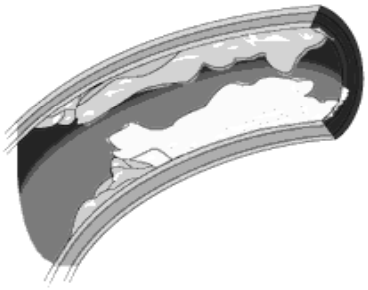
- **Prolonged ongoing rest pain > 20 minutes**
- **Elevated cardiac troponin (TnT or TnI > 0.1 ng/mL)**
- **New or presumably new ST-segment depression**
- **Sustained ventricular tachycardia**
- **Pulmonary edema, most likely due to ischemia**
- **New or worsening mitral regurgitation (MR) murmur**
- **S_3 or new/worsening rales**
- **Hypotension, bradycardia, tachycardia**
- **Age > 75 years**

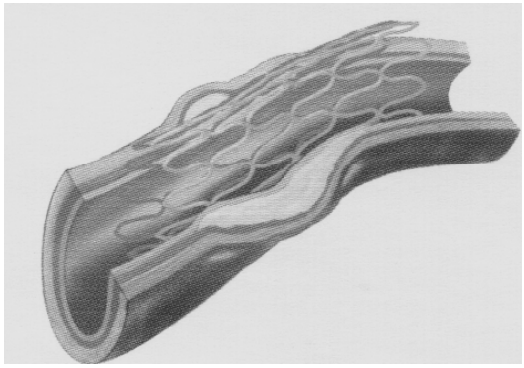
Braunwald E, et al. 2002. http://www.acc.org/clinicalguidelines/unstable/unstable.pdf.

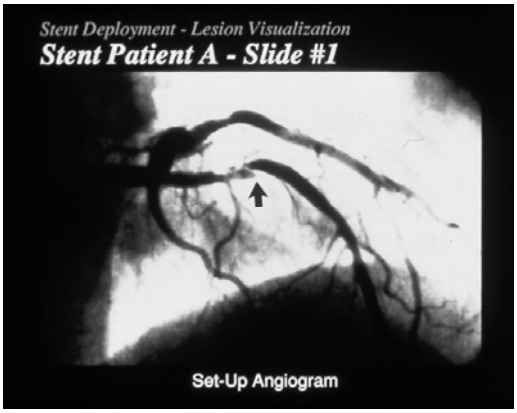


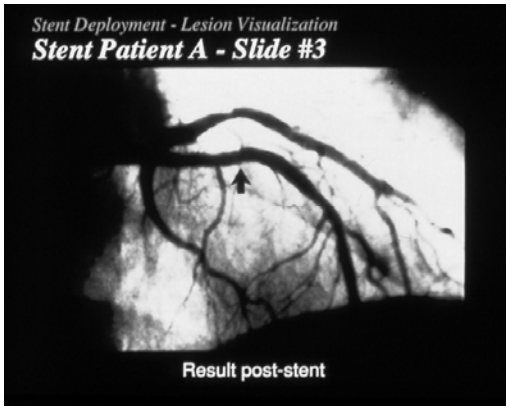


Balloon

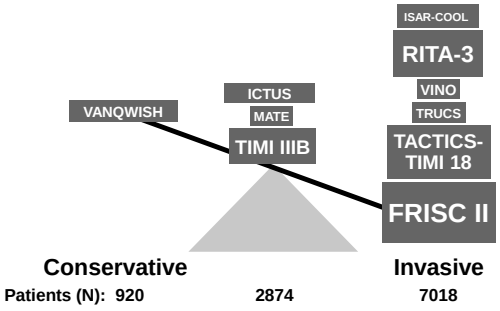




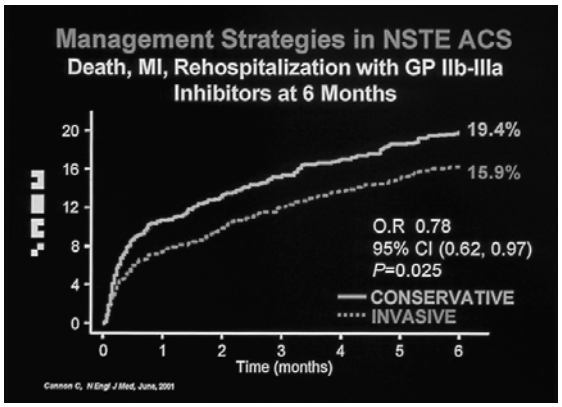


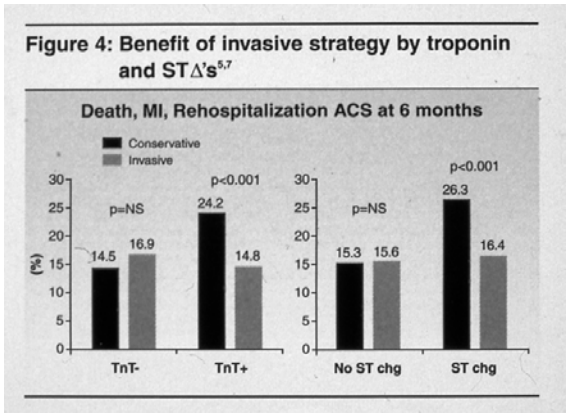


Optimal Strategy for UA/NSTEMI



Adapted with permission from Cannon CP, Turpie AG. *Circulation.* 2003;107:2640-2645.





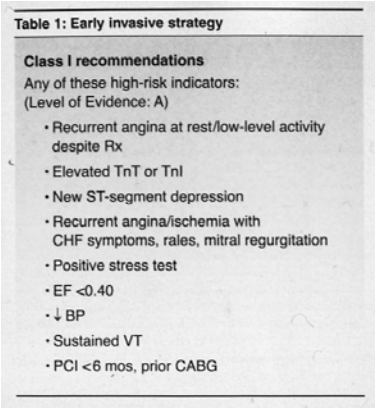
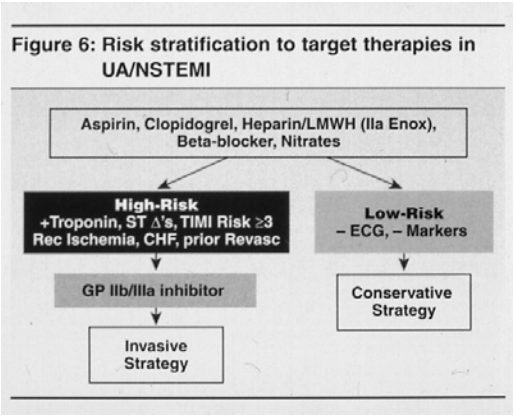


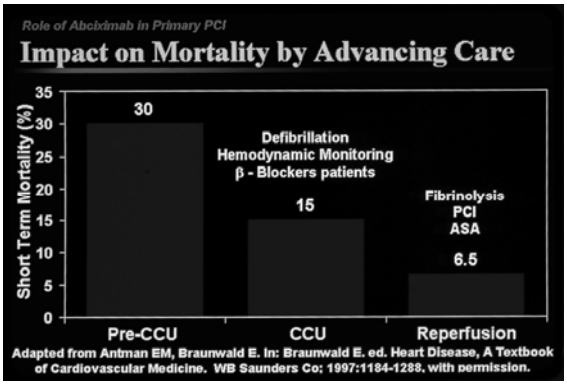
Table 1: Early invasive strategy

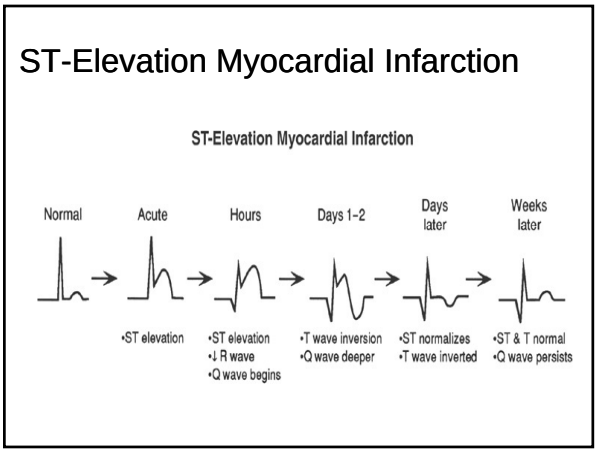
Class I recommendations

Any of these high-risk indicators:
(Level of Evidence: A)

- Recurrent angina at rest/low-level activity despite Rx
- Elevated TnT or TnI
- New ST-segment depression
- Recurrent angina/ischemia with CHF symptoms, rales, mitral regurgitation
- Positive stress test
- EF <0.40
- ↓ BP
- Sustained VT
- PCI <6 mos, prior CABG







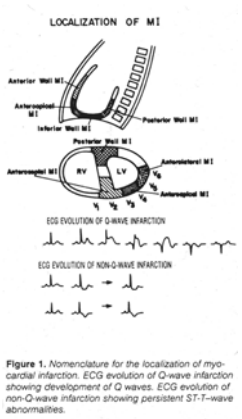
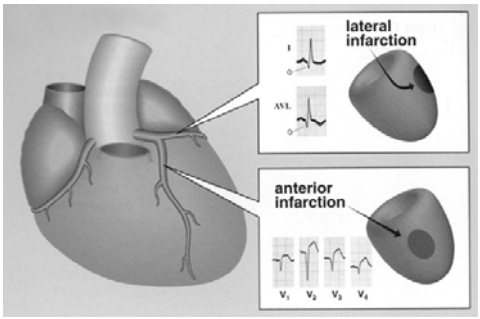


Figure 1. *Nomenclature for the localization of myocardial infarction. ECG evolution of Q-wave infarction showing development of Q waves. ECG evolution of non-Q-wave infarction showing persistent ST-T–wave abnormalities.*

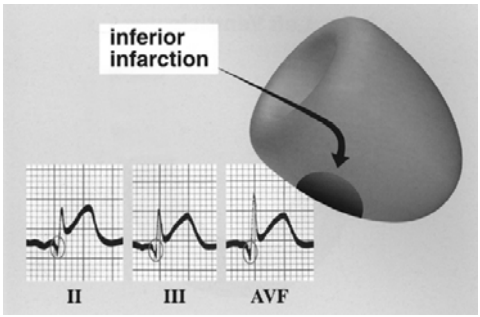
Table 2. Localizing Myocardial Infarction

LOCATION	ECG LEADS INVOLVED	PROBABLE ARTERY INVOLVED
Anteroseptal	V_1-V_2	Proximal left anterior descending (LAD), septal perforators
Anteroapical	V_2-V_4	LAD or its branches
Anterolateral	V_4-V_6, I, aVL	Mid-LAD or circumflex
Extensive anterior	V_1-V_6	Proximal LAD
High lateral	I, aVL	Circumflex
Inferior	II, III, aVF	Right coronary; less often circumflex or distal LAD
Posterior	Mirror image in V_1 and V_2 (ST depression, peaked T, tall R, loss of S waves)	Posterior descending
Right ventricular	V_4 and reversed chest leads rV_3-rV_4	Right coronary

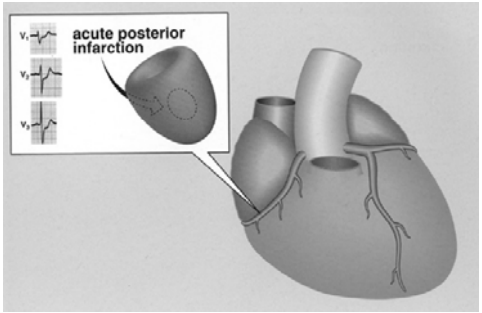
Lateral: Circumflex / Anterior: LAD

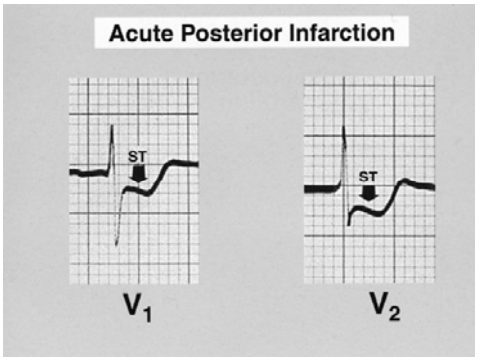


Vessel: RCA, Circumflex



Vessel: RCA





PROGNOSIS IN ACUTE MYOCARDIAL INFARCTION

1. LVEF - mechanical (pump failure)
2. Arrhythmias - electrical

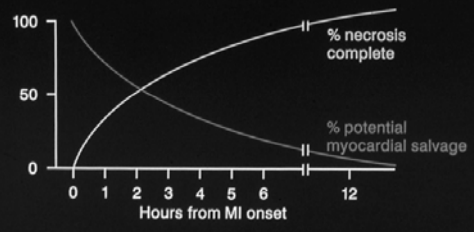
PRINCIPAL OBJECTIVES IN MANAGEMENT OF ACUTE MYOCARDIAL INFARCTION

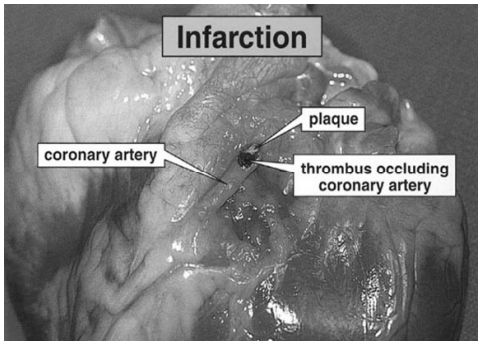
1. Salvage myocardium - minimize the mass of infarcted tissue.
2. Prevent death from arrhythmias.

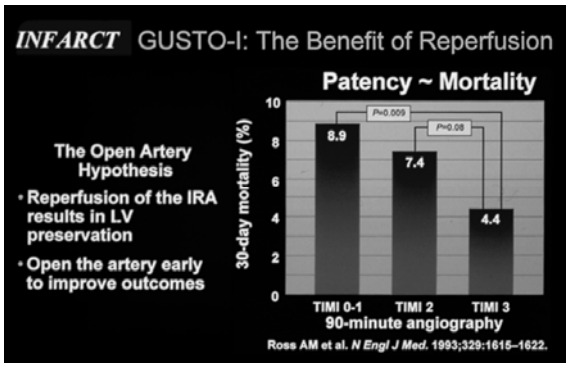
REPERFUSION IN ACUTE MYOCARDIAL INFARCTION

Early reperfusion (pharmacologic with thrombolytic treatment or mechanical with PTCA) of ischemic myocardium can salvage tissue before it becomes irreversibly injured.

TIME COURSE OF MYOCARDIAL INFARCTION



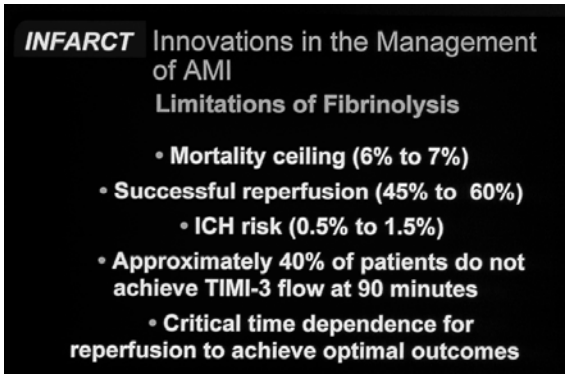




Goal

"Door-to-lytic"
30 minutes

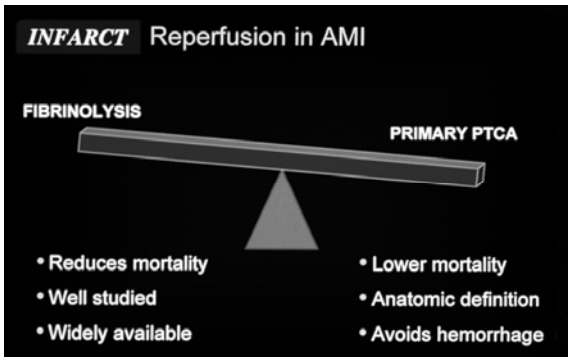
"Door-to-balloon"
90 minutes



Pharmacological Reperfusion for STEMI
Fibrinolysis Background/Limitations

- Initial occluded artery remains (TFG 0/1), in ~20% of patients → 2-fold ↑ in mortality[1,2]
- Reocclusion occurs in 5-10% of patients → 3-fold ↑ in mortality [3,4]
- Reinfarction occurs in ~5% of patients → 3-fold ↑ in mortality [5]

1.TIMI 1, *Am J Cardiol* 1998;62:179 2. GUSTO I Angio, *NEJM* 1993;329:1615) 3.Ohman et al., *Circulation* 1990;82:781 4. HART II, *Circ* 2001;104:648; PENTALYSE, *EHJ* 2001;22:1716 5. TIMI 2, *JACC* 1995;26:900; TIMI 4 & 5, *Am J Cardiol* 1997;80:696



Percutaneous Coronary Intervention (PCI)

The advantages of Primary PCI

- High 85-95% infarct vessel patency rate
- Low rates of recurrent ischemia, reinfarction, death, and stroke
- Avoidance of ICH
- Shortened LOS
- Ability to treat lytic-ineligible patients

Transfer for PCI in STEMI: NRMI (1999–2002), 4278 Patients

Door-to-Balloon Time	% of Patients
<90 min	4.2
<2 h	16.2
2–4 h	55.4
>4 h	28.4

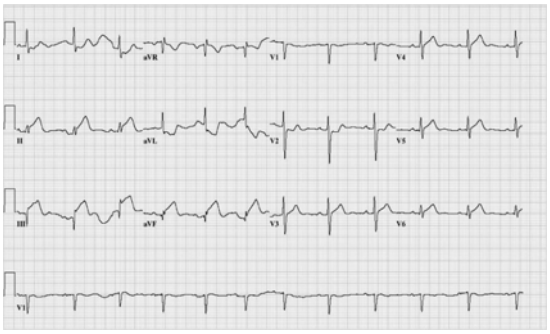
Nallamothu BK, et al. *Circulation.* 2005;111:761-767.

D2B: Strategies Associated With a Significant Reduction in Door-to-Balloon Time ("Code 90")

Strategy	Mean reduction in door-to-balloon time (min)*
Having emergency medicine physicians activate the cath lab	8.2
Having a single call to a central page operator activate the cath lab	13.8
Having the ED activate the cath lab while patient is still en route	15.4
Expecting staff to arrive at the cath lab within 20 minutes after page	19.3
Having an attending cardiologist always on site	14.6
Having staff in the ED and cath lab use and receive real-time feedback	8.6

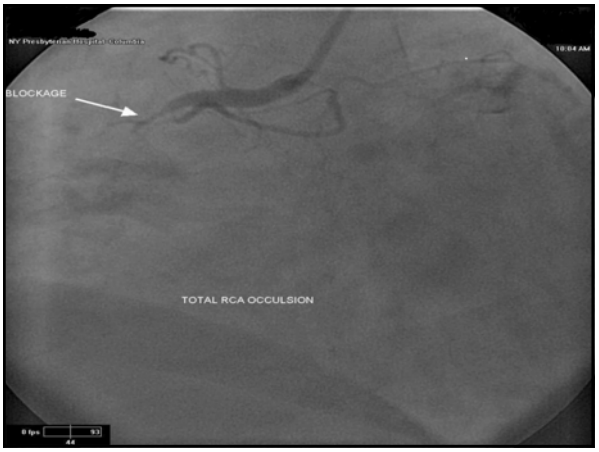
*$P<.05$ for all.

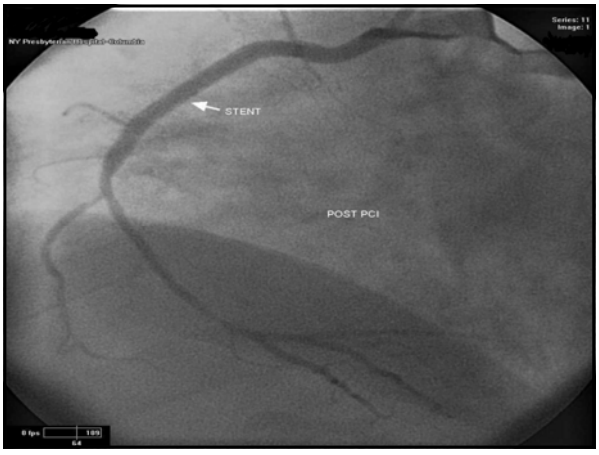
Bradley EH, et al. *N Engl J Med.* 2006;355:2308-2320.



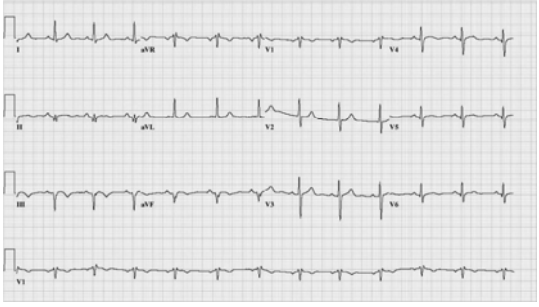
Artery, Lead, Location !

Coronary Artery Involved	Leads	Type of Infarct	Possible Consequences
RCA	II, III, AVF	Inferior	AV block, bradycardia, hypotension, N/V, hiccoughs
RCA	V3R, V4R	RV Infarct	Increased JVD, CVP, large a waves, clear lungs, AV blocks



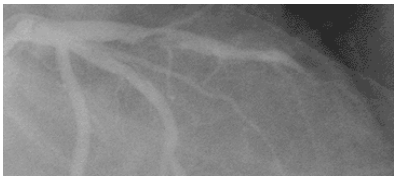


What happened to the ST elevations ?

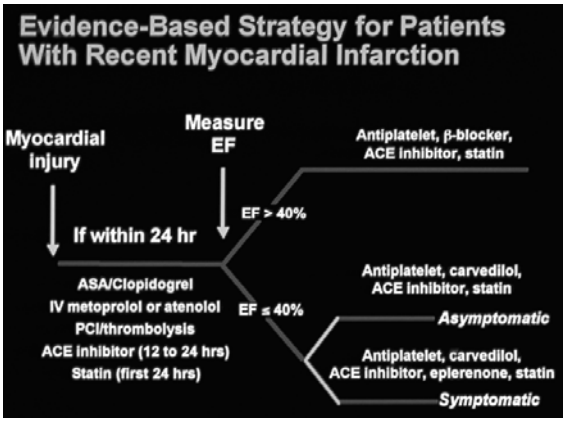


MI BEEPER TO TABLE	TRIAGE TO TABLE	TRIAGE TO WIRE	TRIAGE TO BALLOON
30 min.	48 min.	73 min.	77 min.

Microembolism



Evidence-Based Strategy for Patients With Recent Myocardial Infarction



2007 ACC/AHA STEMI Focused Update

Secondary Prevention: Additional Class I Recommendations

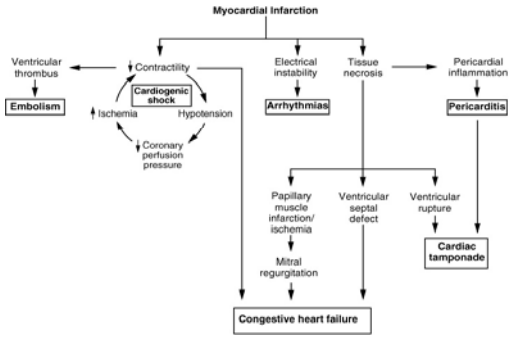
- Complete smoking cessation/no exposure to environmental tobacco smoke
- Statin goal: LDL-C <100 mg/dL
  - Consider LDL-C <70 mg/dL
- Daily physical activity 30 min/d, min 5 d/wk
- Annual influenza immunization
- Avoid NSAIDs whenever possible

Antman EM, et al. *J Am Coll Cardiol.* 2008;51:210-247.

POSTINFARCTION RISK STRATIFICATION

1. Submaximal ETT prior to discharge to detect residual ischemia and ventricular ectopy
2. Maximal (symptom-limited) ETT 4-6 weeks post infarction
3. Assessment of LVEF:
   2-D Echo
   Radionuclide Ventriculography
4. High risk findings:
   angina at low workload
   large reversible defect on perfusion imaging
   depressed LVEF with ischemia
   ETT-induced symptomatic ventricular arrhythmias
5. Proceed with cardiac catheterization and/or invasive electrophysiologic study as needed

Complications of MI



VENTRICULAR FIBRILLATION AND ACUTE MYOCARDIAL INFARCTION

1. Most common form of arrhythmic death in acute MI.
2. Vast majority of deaths due to v fib occur within the first 24 hrs. of the advent of symptoms; of these deaths, over half occur in the first hour.
3. Most out-of-hospital deaths from MI are due to v fib.
4. May occur without warning symptoms or warning arrhythmias.
5. In-hospital mortality from acute MI has decreased from 30% to 10-15%; death from in-hospital ventricular arrhythmia is now unusual.

VENTRICULAR FIBRILLATION POSTINFARCTION

1. Primary v fib (owing to acute ischemia; not associated with CHF, shock, BBB, or LV aneurysm) has a good long-term survival (>90% at one year).
2. Secondary v fib (owing to severe pump failure) occurring late in the hospital course has an extremely poor prognosis (85% mortality at one year); consider EPS.

ATRIOVENTRICULAR AND INTRAVENTRICULAR CONDUCTION DISTURBANCES POSTINFARCTION

1. Anterior MI and CHB: 60-75% mortality
   Inferior MI and CHB: 25-40% mortality
2. Anterior MI and heart block - ischemic malfunction of all 3 fascicles of conduction system - extensive myocardial necrosis
3. Inferior MI and heart block - AV nodal ischemia - small amount of myocardium
4. Anterior MI: Mobitz type II block
   Inferior MI: First-degree AV block
   Mobitz type I block

MANAGEMENT OF CHF POSTINFARCTION

1. Cautious use of Lasix
2. Nitrates - reduce preload; help LV remodeling
3. ACE inhibitors to attenuate LV dilatation - SAVE study
4. Avoid digoxin
5. Optimize PCWP to 18-20 mmHg

CARDIOGENIC SHOCK

1. Infarction of >40% of LV; days 1-6
2. Marked hypotension (<80 mmHg), marked reduction in cardiac index (<1.8 L/min/m^2) with PCWP >18 mmHg
3. Mortality of 80%
4. Risk Factors for Cardiogenic Shock:
   advanced age
   depressed LVEF
   large MI
   previous MI
   diabetes mellitus
5. Treatment: Hemodynamic Monitoring
   Vasopressors
   IABP
   thrombolytic therapy/PTCA

IABP

1. Inflates during early diastole, enhancing coronary blood flow and peripheral perfusion.
2. Deflates in early systole, reducing afterload
3. Indications: intractable ischemia
   cardiogenic shock
   VSD
   MR
4. Contraindications: aortic regurgitation
   aortic dissection
   severe peripheral vascular disease
5. Morbidity of 10%

POSTINFARCTION MITRAL REGURGITATION

1. MR murmur in up to 50% of post MI patients; hemodynamically significant MR in only a minority
2. Papillary muscle dysfunction secondary to ischemia or infarction > MR due to change in LV size or shape from aneurysm or impaired contractility
3. Involvement of Posteromedial muscle (circumflex artery) > Anterolateral muscle (circumflex and LAD arteries)
4. 2-D Echo
5. Papillary muscle dysfunction is frequently compatible with long-term survival
6. Mild MR: no therapy
   Moderate-Severe MR: surgery
   ACE inhibitors

PAPILLARY MUSCLE RUPTURE

1. Occurs in 1% of MIs and accounts for 1-5% of MI deaths
2. Days 2-7 post MI: sudden onset of pulmonary edema with murmur in patients with inferior and/or lateral MI
3. Posteromedial papillary muscle is 6-12 x's more likely to rupture than anterolateral papillary muscle
4. Diagnosis: 2-D Echo and Swan-Ganz catheter
5. Treatment: IABP with vasodilator and inotropic therapy for stabilization → surgery

VSD POSTINFARCTION

1. Occurs in 1-3% of MIs and accounts for 5% of MI deaths
2. Equal frequency between anterior and inferior MIs
3. Majority occur during first week post MI
4. New murmur, CHF, hypotension
5. Diagnosis: Doppler-Echo and Swan-Ganz catheter
6. Treatment: IABP, inotropes, vasopressors → rapid surgery (high risk)
7. Surgical results are worse if VSD complicates an inferior MI and if there is concomitant RV dysfunction

MYOCARDIAL RUPTURE

1. 24% of fatal MIs
2. Free wall of LV ruptures
3. Characteristics: first week post MI
   first MI
   age > 70
   history of hypertension; no LVH
   no history of angina
   large Q wave infarct
   women
4. Prevented by intravenous beta-blockade
5. EMD - almost universally fatal

LV ANEURYSM

1. Dyskinesis - local expansile paradoxical wall motion
2. Scar tissue - not associated with cardiac rupture
3. Complications occur weeks-months after MI: CHF, arterial embolism, ventricular arrhythmia.
4. Apical aneurysms - double, diffuse, or displaced apical impulse
5. EKG finding of ST segment elevation at rest in precordial leads in 25% of patients with apical or anterior aneuryms
6. 2-D Echo: detect mural thrombus
7. Pseudoaneurysm - limited myocardial rupture - needs surgical repair.

RIGHT VENTRICULAR INFARCTION

1. 1/3 of patients with inferoposterior MI have some degree of RV necrosis.
2. Severe RV failure (JVD, Kussmaul's sign, hepatomegaly) with or without hypotension; low cardiac output if severe. Lungs are clear.
3. ST-segment elevations of right-sided precordial leads, particularly lead V4R.
4. Diagnosis: 2-D Echo; Swan-Ganz catheterization reveals equalization of diastolic pressures.
5. Treatment: volume expansion - Swan-Ganz catheter
   avoid nitrates, vasodilators, diuretics
   use IABP, dopamine, dobutamine as needed
   thrombolytic therapy/PTCA
   dual chamber A-V sequential pacing if CHB
6. Mortality: 20%

PERICARDITIS POSTINFARCTION

1. Pericardial friction rub with pericarditic pain
2. Manage with high dose aspirin (650 mg p.o. q.i.d.)
3. Avoid NSAIDs and steroids
4. Must be careful in using heparin or Coumadin because of danger of tamponade

THROMBOEMBOLISM POSTINFARCTION

1. Clinically apparent in 10% of MI cases
2. Embolic lesions in 45% at autopsy
3. Contributes to death in 25% of MI patients
4. LV mural thrombi on 2-D Echo; 20% spontaneous regression; treat with Coumadin for 3-6 months, particularly if large anterior wall MI with CHF, akinesis, or dyskinesis
5. SQ heparin to prevent pulmonary emboli arising from leg veins