Acute Coronary Syndromes

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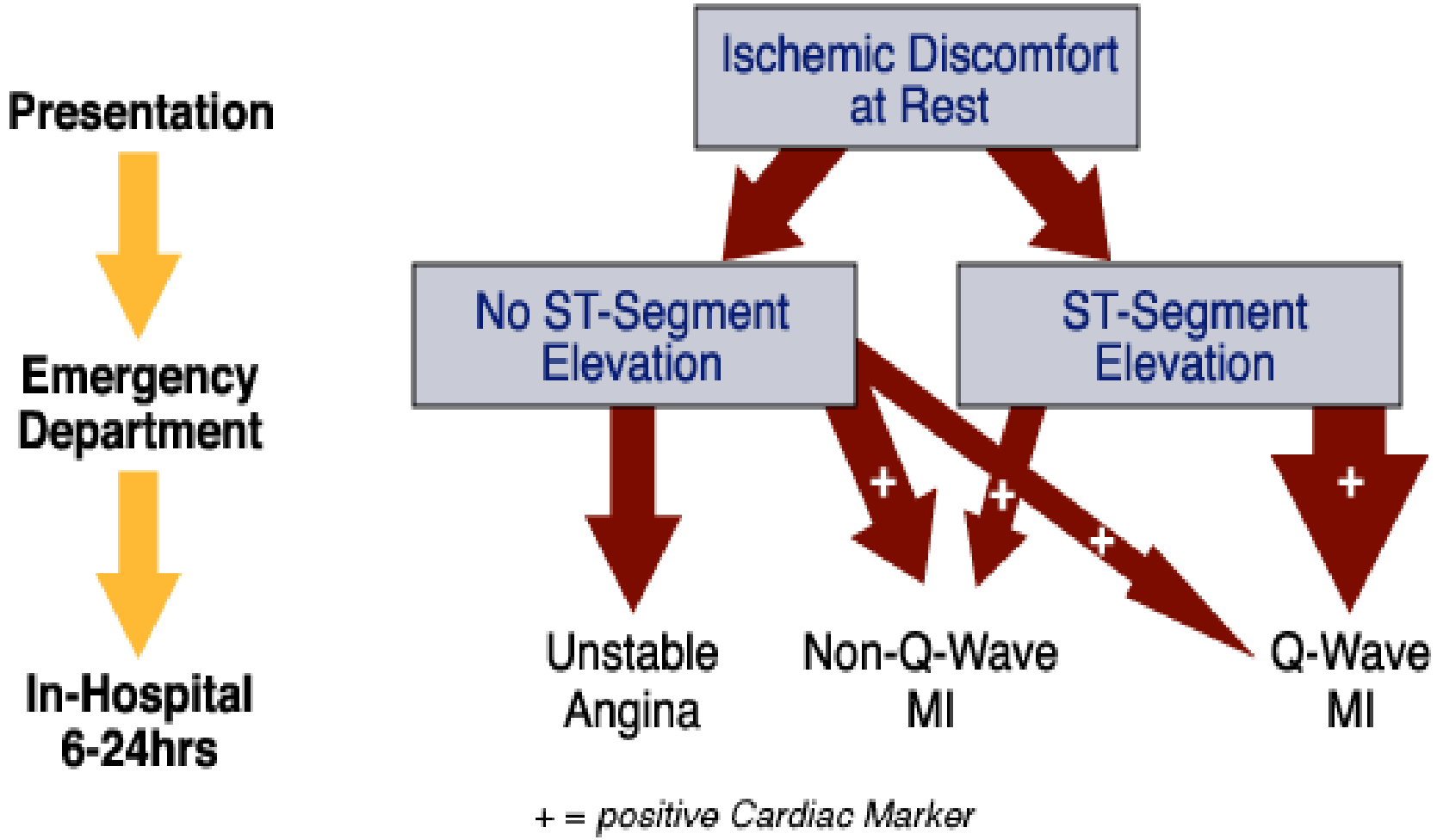
Spectrum of Acute Coronary Syndromes



+ = *positive Cardiac Marker*

Pathophysiology

- AMI is the result of inadequate myocardial perfusion of sudden onset.
- Atherosclerosis of the coronary arteries is by far the most frequent underlying cause of ischemic heart disease.
- Life-threatening ACSs, including AMI, are precipitated by plaque disruption with superimposed thrombosis, with or without concomitant vasoconstriction

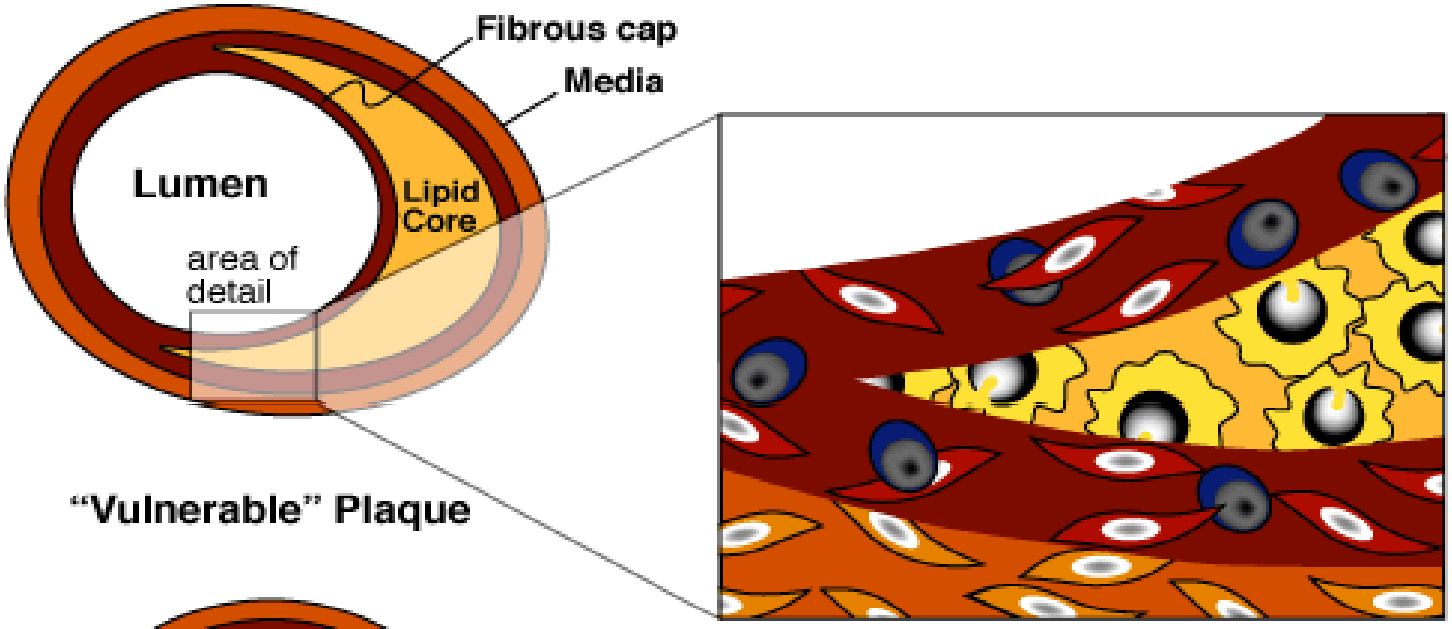
Plaque Prone to Rupture Defined as Plaque Vulnerability

- The risk of plaque rupture depends more on plaque type than on plaque size or degree of stenosis caused by the plaque; lipid-rich and soft plaques are more vulnerable and prone to rupture than collagen-rich and hard plaques.
- Lipid accumulation, cap thinning, macrophage infiltration, and local loss of SMCs destabilize plaques, making them vulnerable to rupture.
- In contrast, SMC-mediated healing and repair processes stabilize plaques, protecting them against rupture
- *Many vulnerable plaques are invisible angiographically* due to compensatory vascular remodeling.

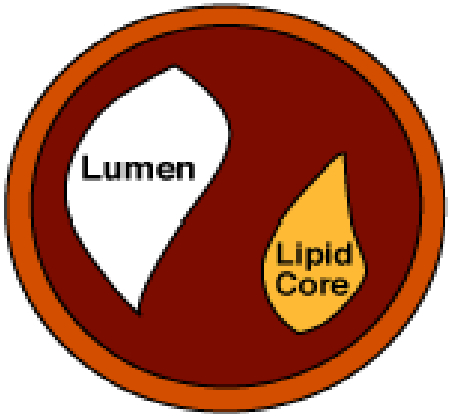
Pre-Existing Stenosis

- As many as three-fourths of all infarct-related thrombi appear to evolve over plaques causing only mild-to-moderate stenosis prior to infarction
- Thus, the great majority of heart attacks originate from atherosclerotic lesions that, prior to the acute events, were hemodynamically insignificant and probably asymptomatic
- The vulnerable and most dangerous lesions tend to preserve the lumen (outward remodeling), and may thus be invisible angiographically, whereas the generally smaller but fibrotic and stable lesions tend to narrow the lumen (inward remodeling), and are thus those seen angiographically

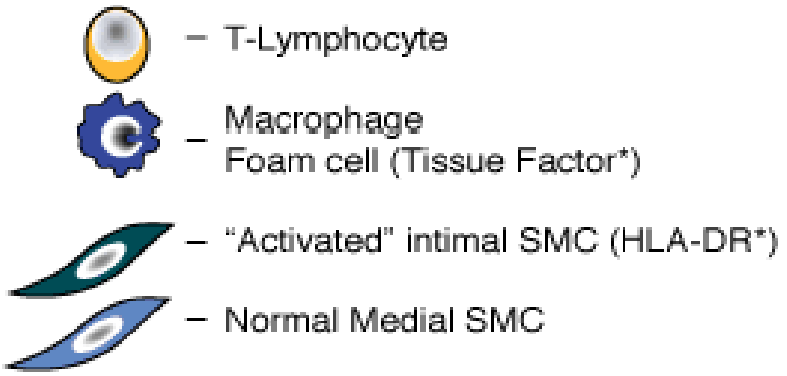
Comparison of Vulnerable and Stable Plaques



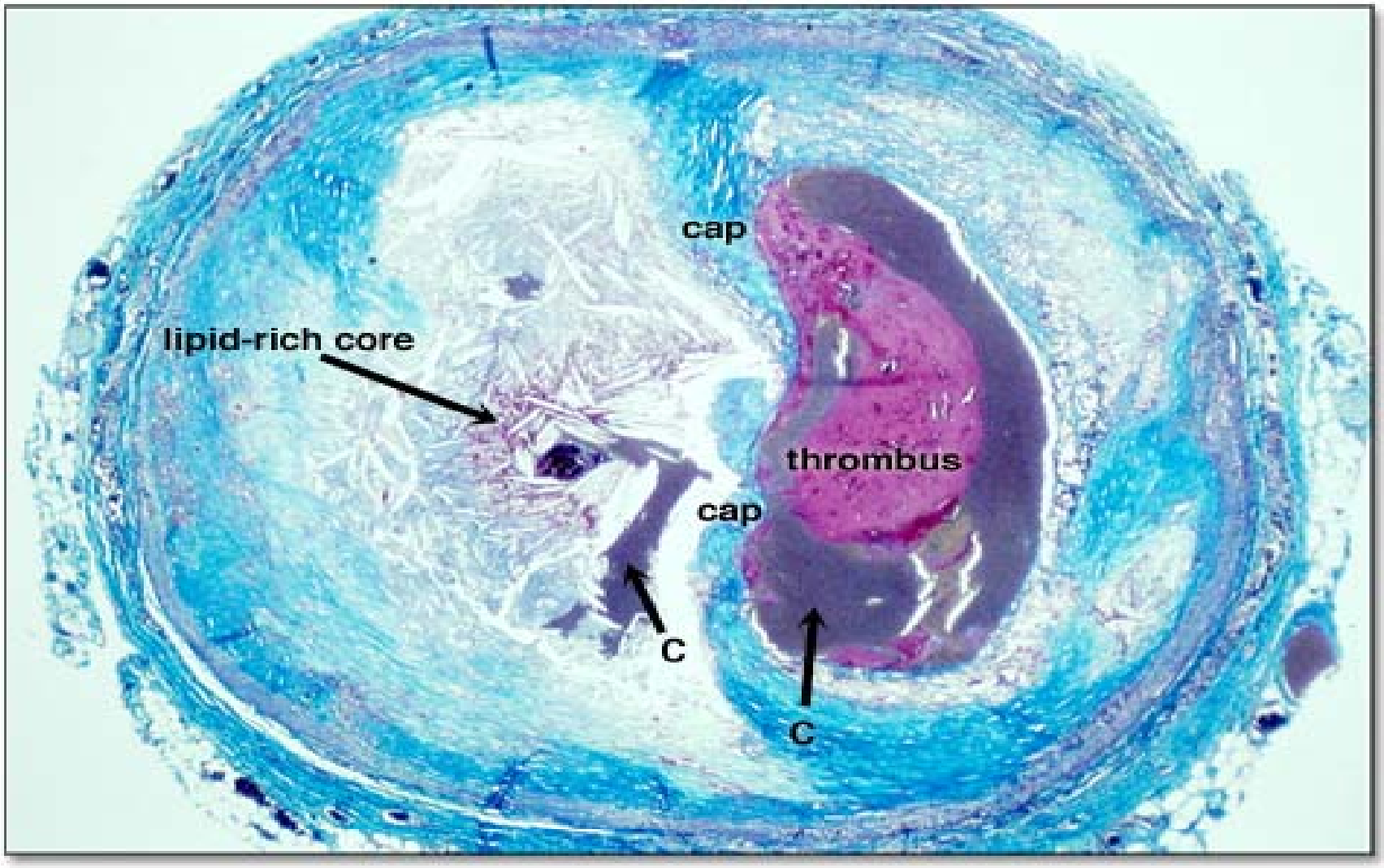
"Vulnerable" Plaque



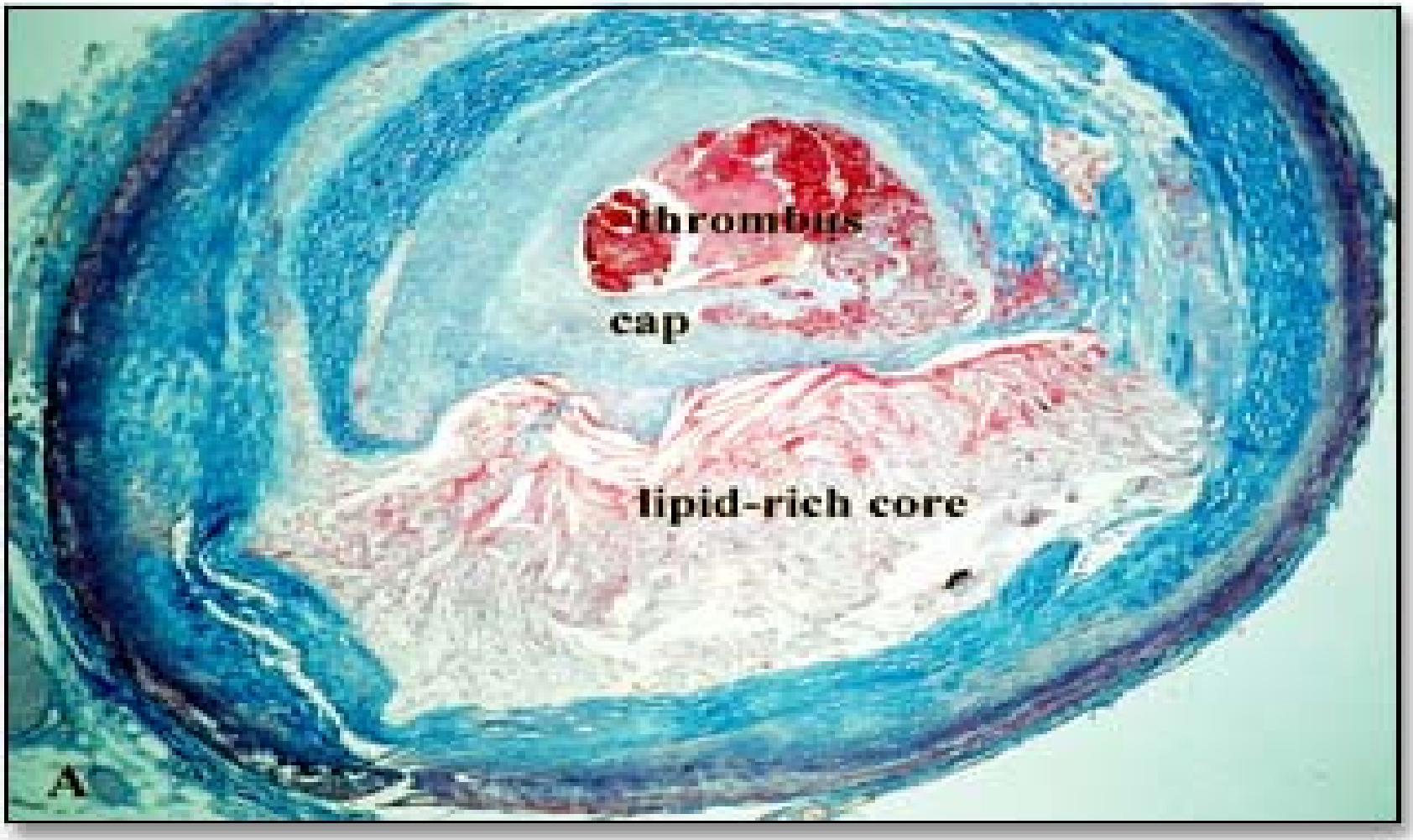
"Stable" Plaque



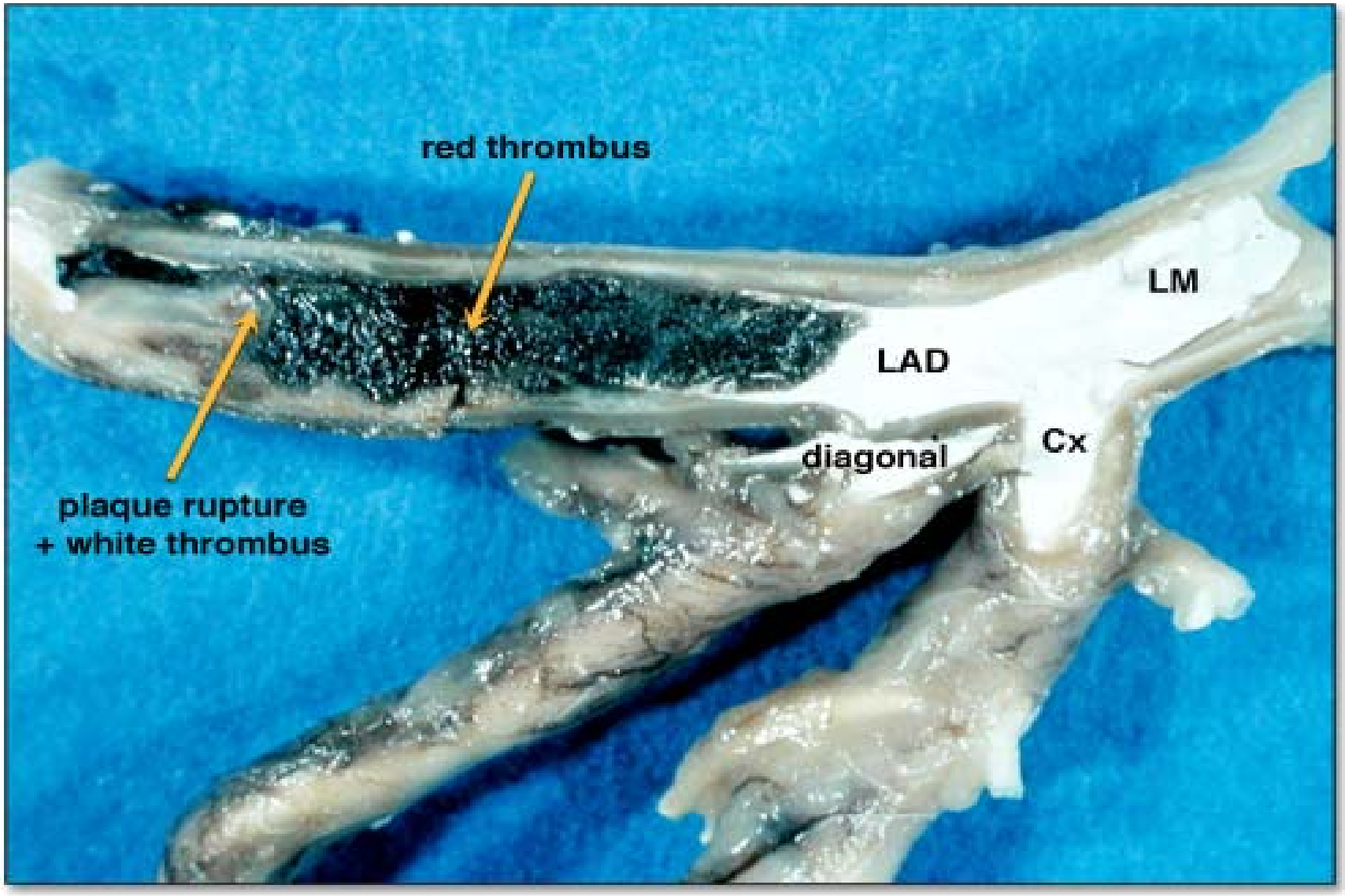
High Shear Promotes Arterial Thrombosis



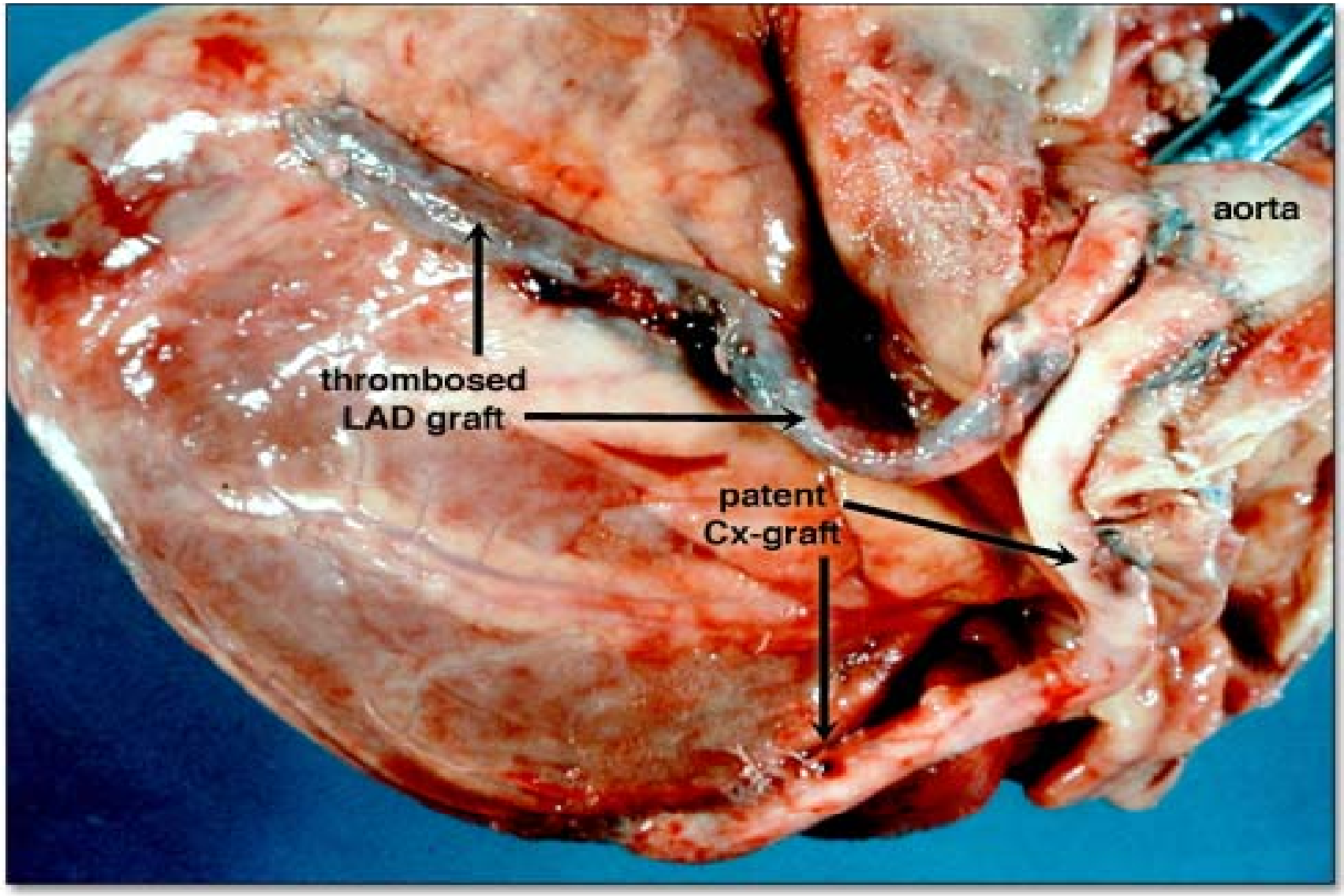
Dynamic Coronary Thrombus



Stagnation Thrombosis

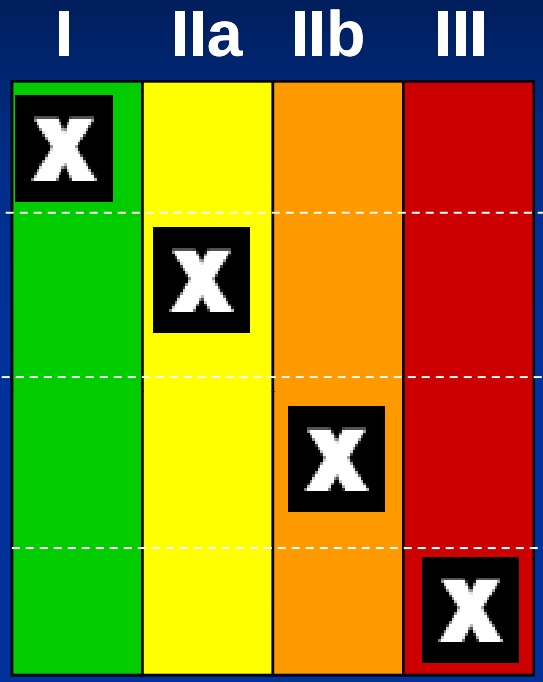


Thrombosed Vein Graft



ACC/AHA NSTE ACS Guidelines

Classes of Recommendations



Generally agreed to be useful/effective

Weight of evidence/opinion in favor of efficacy/usefulness

Efficacy/usefulness less well established by evidence/opinion

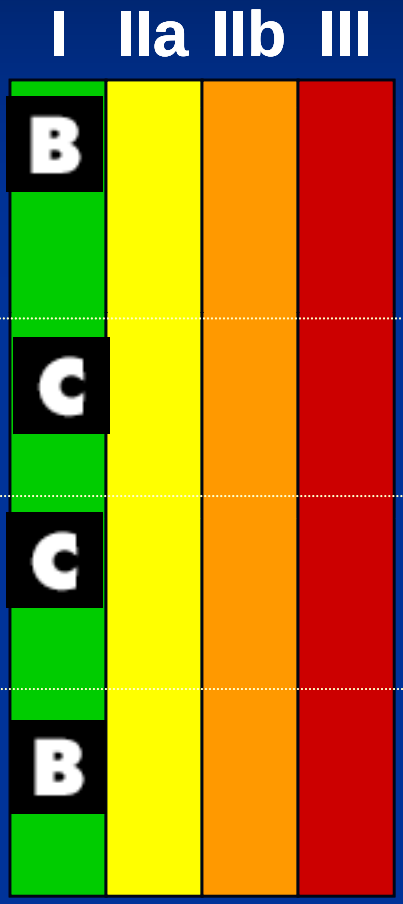
Generally agreed not to be useful/effective and may be harmful

Weight of Evidence Grades

- A = Data from many large, randomized trials
- B = Data from fewer, smaller randomized trials, careful analyses of nonrandomized studies, observational registries
- C = Expert consensus

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

Initial Evaluation



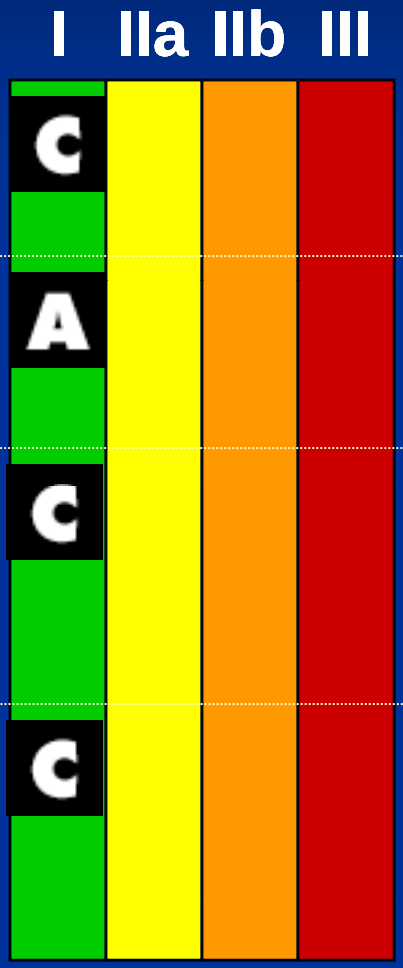
Early risk stratification by symptoms, physical findings, ECG, cardiac markers

12-lead ECG within 10 min for ongoing pain, or ASAP if pain has resolved at presentation

Cardiac markers, Troponins and CK-MB, for initial assessment

Monitoring, repeat ECG and cardiac markers in 6-12 hours, if initial results normal

Immediate Management



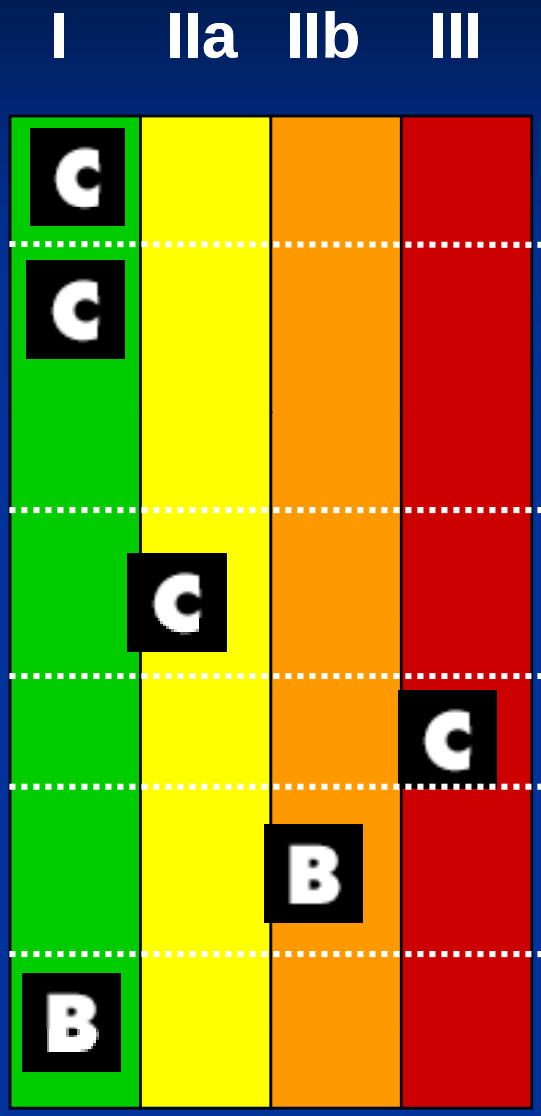
Classify as non-cardiac, chronic stable angina, possible ACS, or definite ACS

Evaluate for immediate reperfusion therapy if definite ACS and ST-segment ↑ present

Pharmacological or exercise stress test, if possible ACS and serial biomarkers and ECGs are normal

Admit pts with definite ACS, ongoing pain, ↑ biomarkers, new ST Δ or deep T-wave inversion, abnormal hemodynamics, or (+) stress test

Early Risk Stratification of NSTE ACS



12-lead ECG (within 10 minutes)

Troponin of CK-MB assay

- Immediate
- Repeated within 6-12 hrs, if negative

Myoglobin of CK-MB subforms assay within 6 hours of onset of symptoms

Total CK without MB

C-reactive protein (CRP) and other markers of inflammation

Search for non-coronary causes of symptoms

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

Estimating the Likelihood That the Suspected ACS Event Is Secondary to CAD

Feature	High Likelihood	Intermediate Likelihood	Low Likelihood
	Any Below:	No High Likelihood Features but Any Below:	No High- or Intermediate-Likelihood Features but May Have:
History	Typical Angina	Probable Angina Known hx of CAD, Including MI	Atypical Symptoms Age >70 Years Male DMr
Examination	CHF	PVD, CVA	Pain on Palpation
ECG	New ECG Δs	Old ECG Abnormalities	Normal ECG Abnormalities
Cardiac Markers	⊕	Normal	Normal

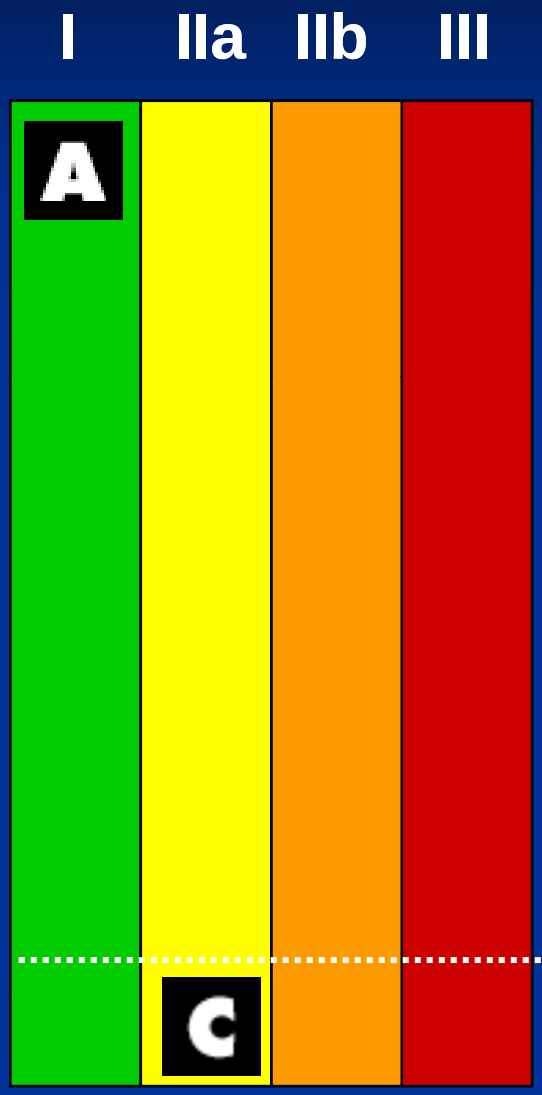
TIMI Risk Score for UA/NSTEMI

Historical	Points
Age ≥ 65	1
≥ 3 CAD risk factors (FHχ, HTN, ↑dhol, DM, active, smoker)	1
Known CAD (Stenosis ≥ 50%)	1
ASA use in past 7 days	1
Presentation	
Recent (≤ 24h) severe angina	1
↑ cardiac markers	1
ST deviation ≥ 0.5 mm	1
Risk Score = Total Points (0 - 7)	

Risk of Cardiac Events (%) by 14 Days in TIMI 11B*		
Risk Score	**Death or MI**	**Death, MI or Urgent Revasc**
0/1	3	5
2	3	8
3	5	13
4	7	20
5	12	26
6/7	19	41

*Entry criteria: UA or NSTEMII defined as ischemic pain at rest within past 24 hours, with evidence of CAD (ST segment deviation or + marker)

Patients Requiring an Early Invasive Management Strategy

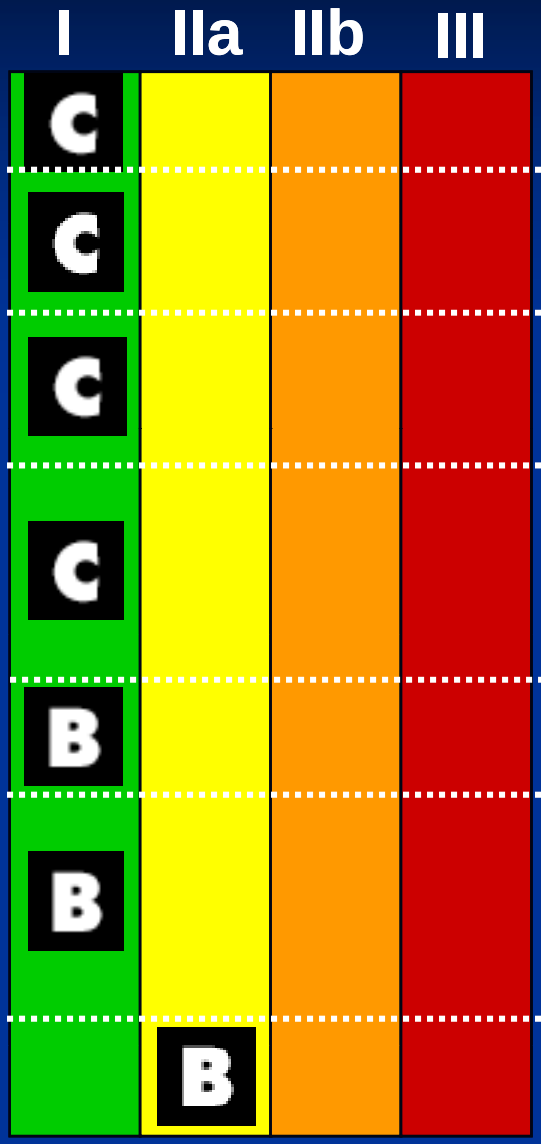


- New or presumably new ST-segment depression
- Elevated cardiac troponin
- Recurrent angina or ischemia at rest despite anti-ischemia therapy
- Recurrent angina or ischemia at rest with CHF, S_3 gallop, pulmonary edema, worsening rales, new/worsening mitral regurgitation (MR)
- High-risk findings from non-invasive stress testing
- ↓ LV function
- Hemodynamic instability; angina at rest with BP ↓
- Sustained ventricular tachycardia
- Prior PCI (< 6 months) or prior CABG

- Patient with repeat ACS

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

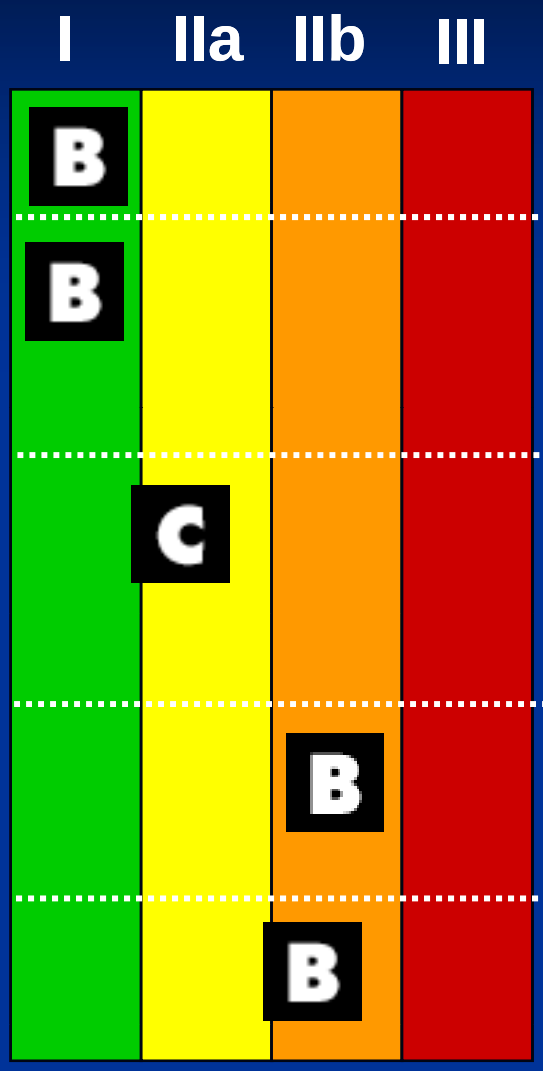
Initial Anti-ischemic Therapies



- Bed rest with continuous ECG monitoring
- Nitroglycerin, sublingual or spray, followed by intravenous
- Supplemental O_2, if cyanosis, respiratory distress, or Sa $O_2 \leq 90\%$
- IV morphine sulfate, if symptoms persist despite NTG, or when acute pulmonary congestion and/or severe agitation is present
- ACE inhibitor in patients with diabetes
- ACE inhibitor if ↑ BP persists with NTG + β blocker in patients with LV systolic dysfunction or CHF
- ACE inhibitor for all post-ACS patients

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

Initial Anti-ischemic Therapies



β blocker (IV →oral), if not contraindicated

Non-dihydropyridine Ca^{2+} antagonist in patients with continuing/frequent ischemia (if β blocker contraindicated and if no severe LV dysfunction)

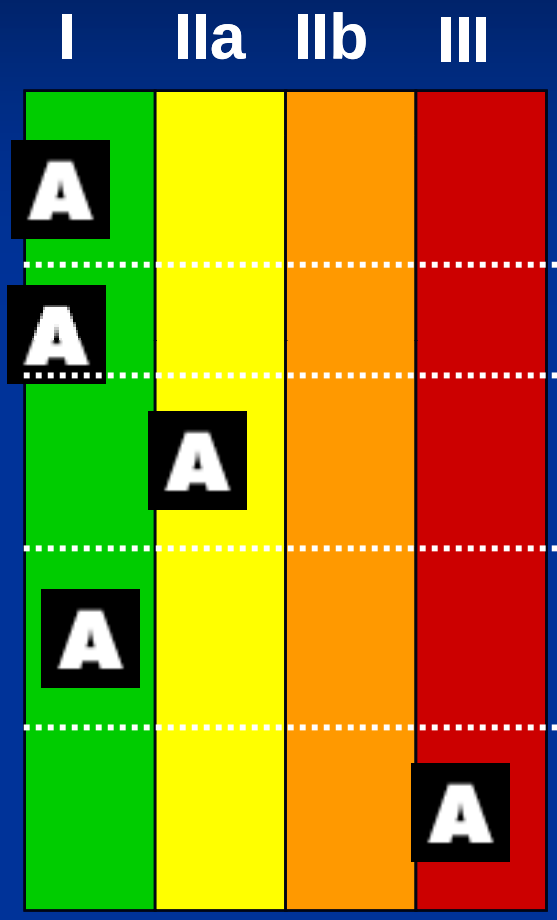
Long-acting Ca^{2+} antagonist in addition to NTG + β blocker for recurrent ischemia (if no contraindications)

Extended-release non-dihydropyridine Ca^{2+} antagonist, instead of β blocker

Immediate-release dihydropyridine Ca^{2+} antagonist with β blocker

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

Early Invasive Approach: Initial Anti-thrombotic Therapies



Aspirin; clopidogrel, if aspirin is contraindicated

LMWH or UFH

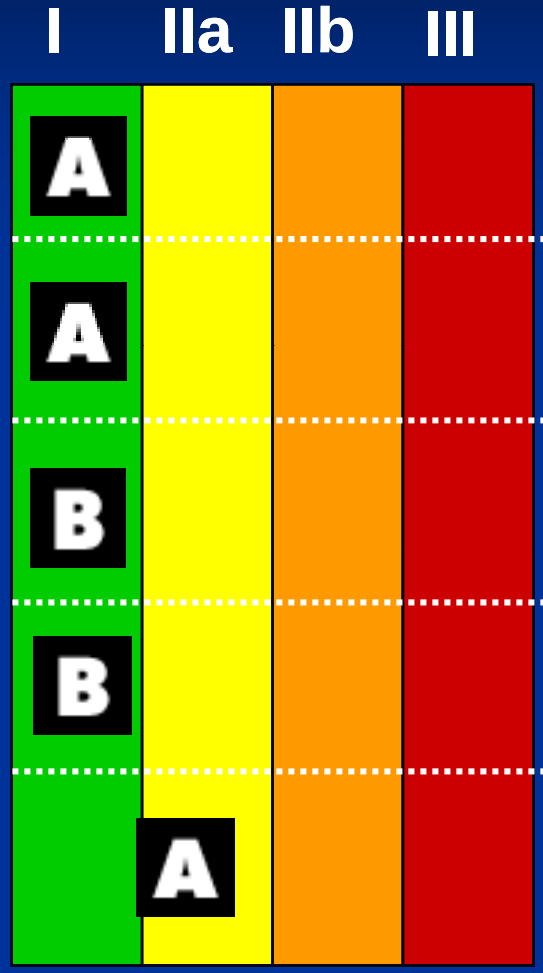
Enoxaparin is preferable to UFH unless CABG is planned within 24 hours

GP IIb-IIIa inhibitor if catheterization or PCI planned

Abciximab is not recommended if early PCI is not planned

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

At Time of Revascularization: Anti-thrombotic Therapies



GP IIb-IIIa inhibitor prior to PCI, if not initiated earlier

Clopidogrel prior to PCI & continued for up to 1 month

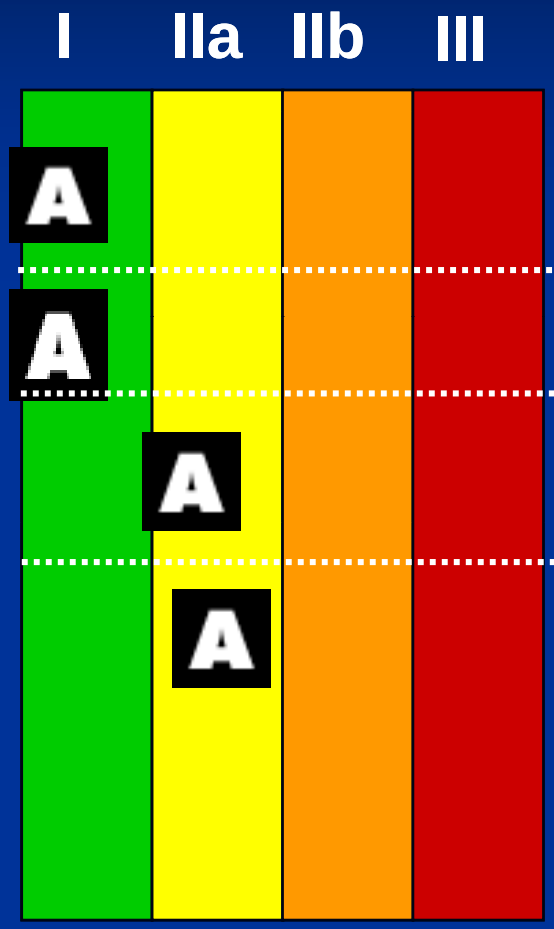
Clopidogrel prior to PCI & continued for up to 9 months

Withhold clopidogrel if CABG is planned within 5-7 days

Withhold enoxaparin if CABG is planned within 24 hours

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

Early Conservative Approach: Initial Anti-thrombotic Therapies



Aspirin; clopidogrel, if aspirin is contraindicated

LMWH or UFH

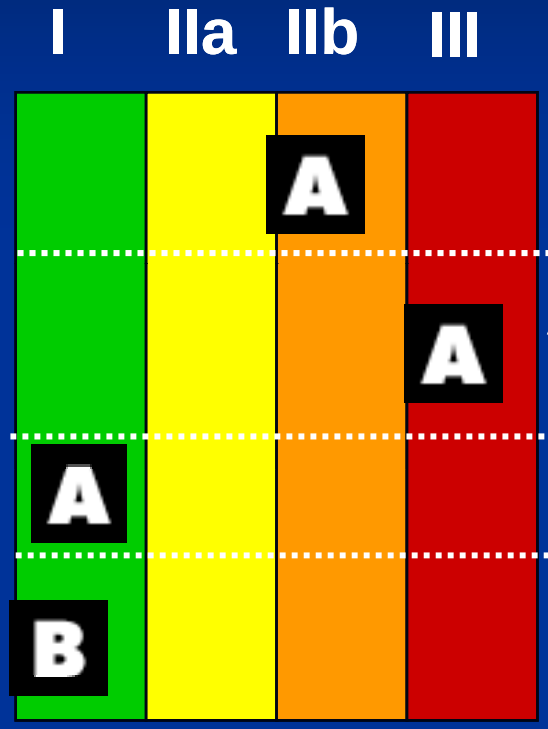
Enoxaparin is preferable to UFH unless CABG is planned within 24 hours

Eptifibatide or tirofiban in patients with:

- continuing ischemia
- elevated TnI or TnT
- other high-risk features

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

Early Conservative Approach: Initial Anti-thrombotic Therapies



Eptifibatide or tirofiban in patients without continuing ischemia or high-risk features

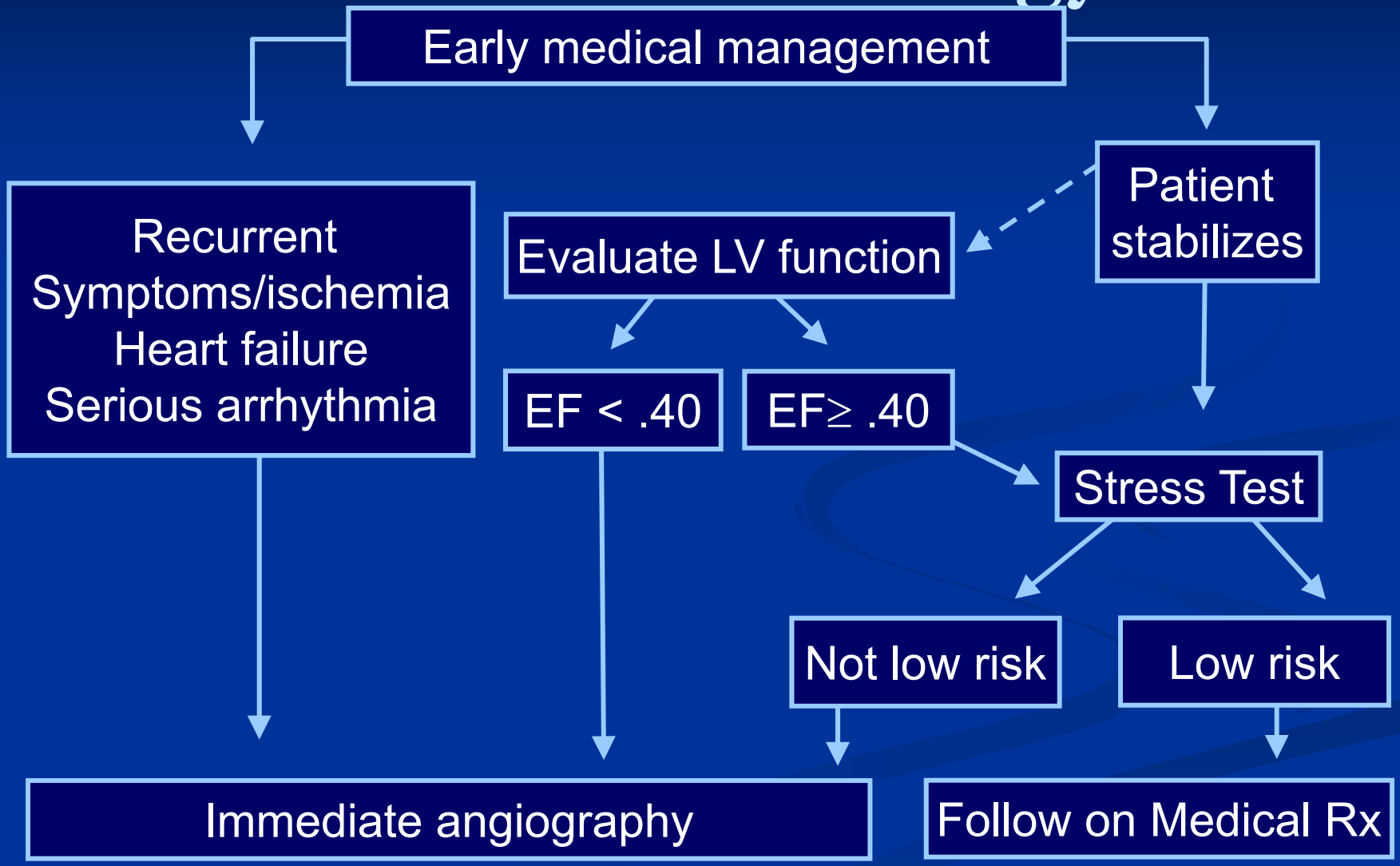
Abciximab administration is not recommended if PCI is not planned

Clopidogrel for at least 1 month

Clopidogrel for up to 9 months

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

Ongoing Evaluation in an Early Conservative Strategy



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

2002 ACC/AHA Guidelines for the Management of High-risk NSTE ACS

At presentation

ST-segment depression &/or elevated cardiac troponin

Need to immediately arrest thrombus progression

Need to eliminate occlusive ruptured plaque

Start immediate

- Aspirin
- Heparin or low-molecular-weight heparin
- GP IIb-IIIa inhibitor

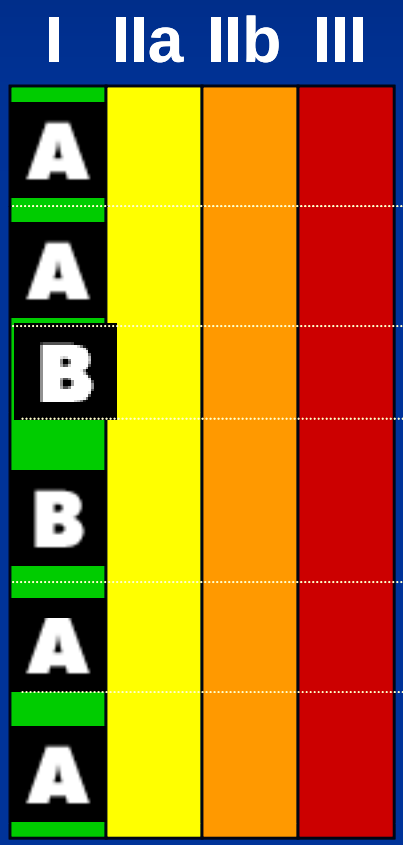
Send for catheterization & revascularization within 24-48 hours

Cautionary information

- No clopidogrel within 5-7 days prior to CABG surgery
- No enoxaparin within 24 hours prior to CABG surgery
- No abciximab, if PCI is not planned

Adapted from Braunwald E, et al. 2002. http://www.acc.org/clinical/guidelines/unstable/unstable.pdf.

Discharge/Post-Discharge Medications



ASA, if not contraindicated

Clopidogrel, when ASA contraindicated

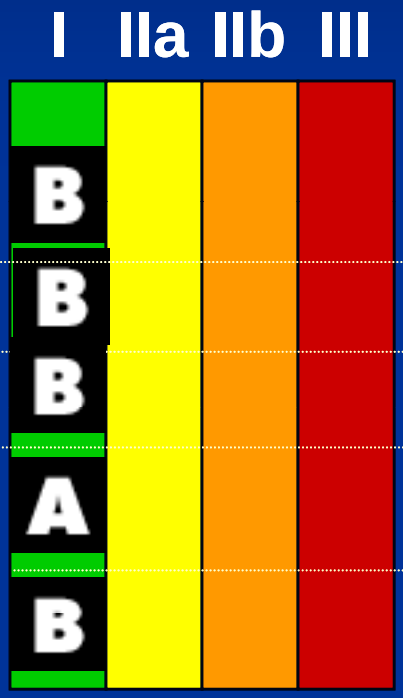
Aspirin + Clopidogrel, for up to 9 months

β-blocker, if not contraindicated

Lipid ↓ agents + diet, if LDL >130 mg/dL

ACE Inhibitor: CHF, EF < 40%, DM, or HTN

Risk Factor Modification



Smoking Cessation Counseling

Dietary Counseling and Modification

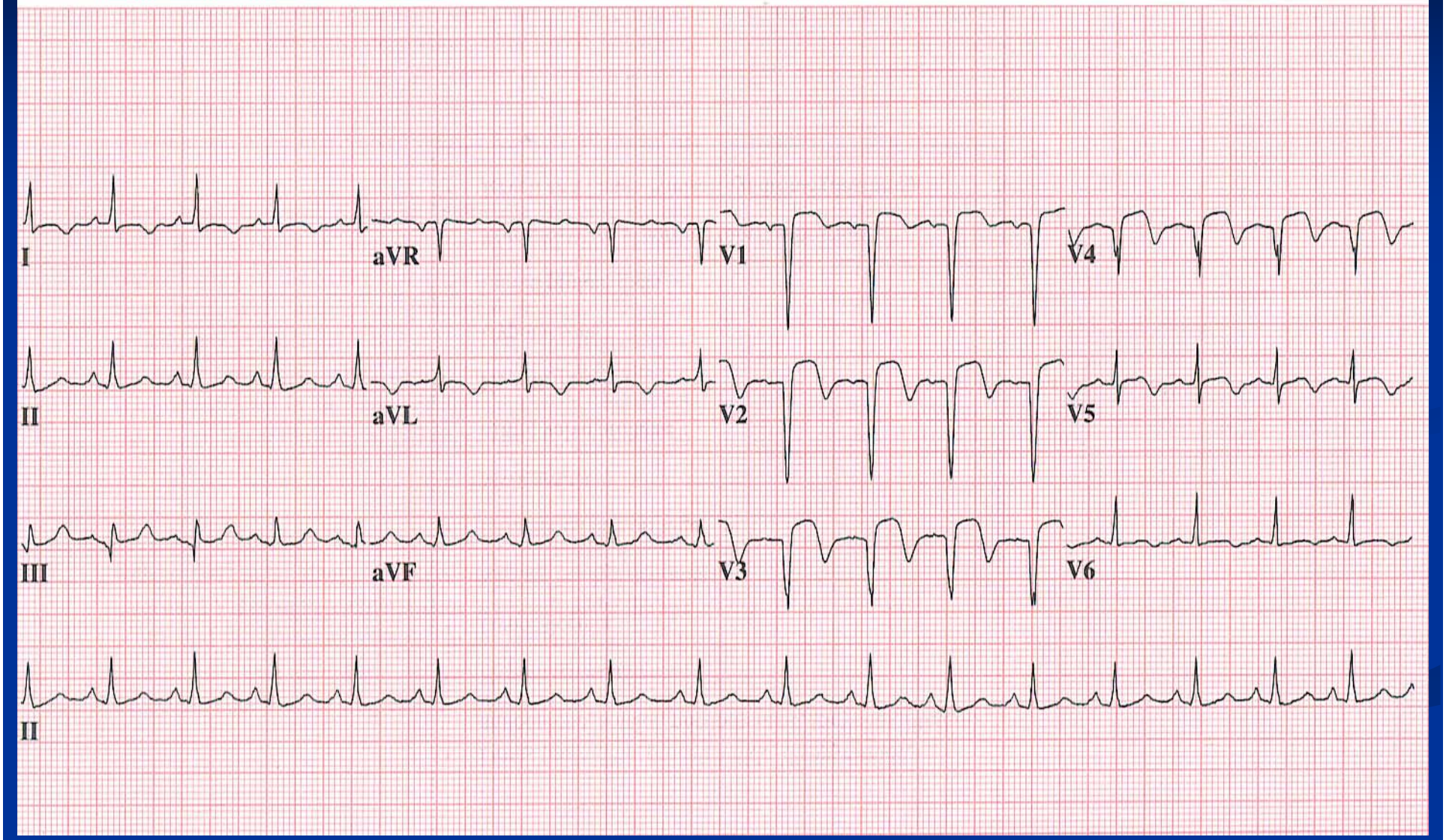
Cardiac Rehabilitation Referral

HTN Control (BP < 130/85 mm Hg)

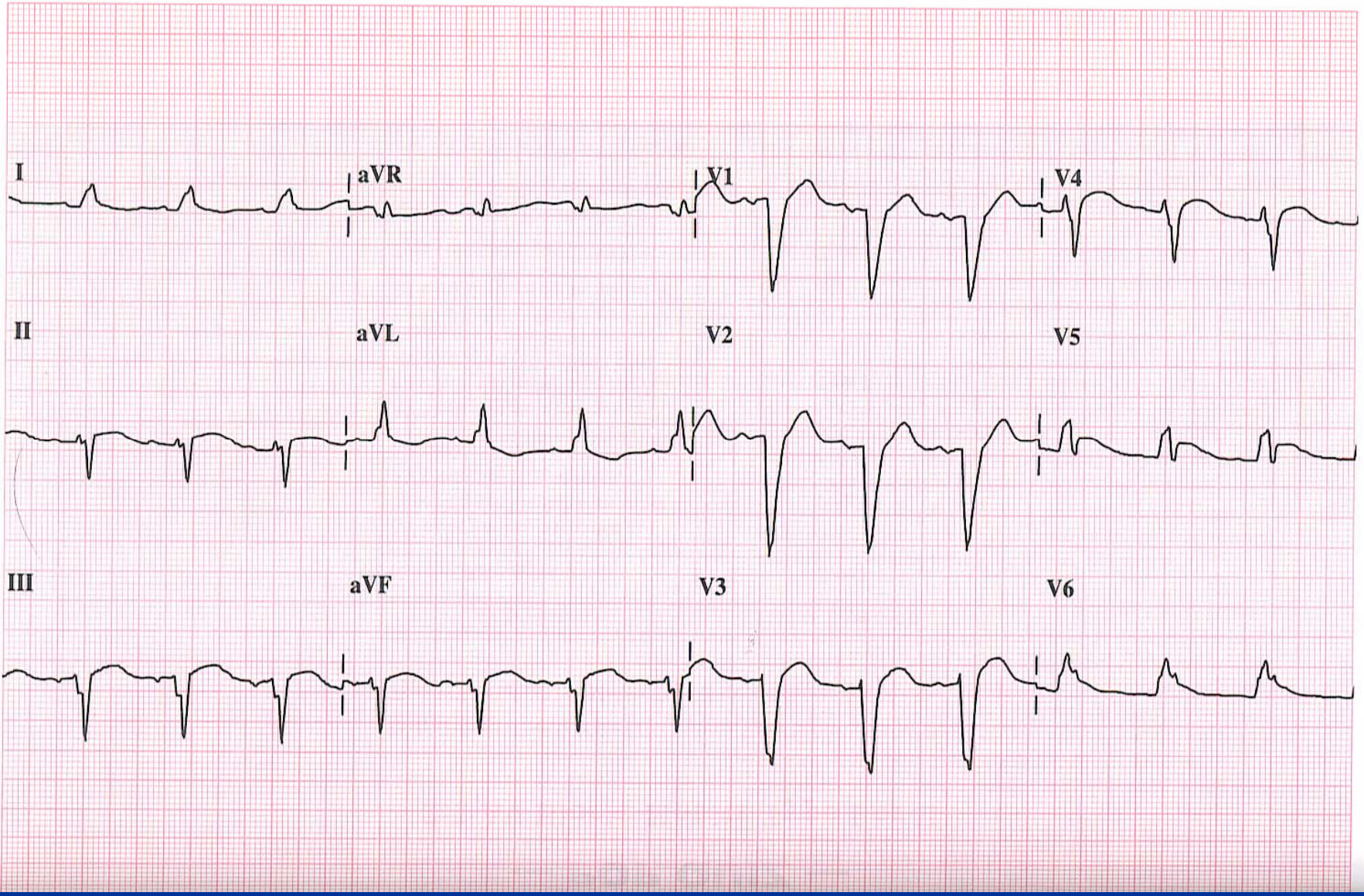
Tight Glycemic Control in Diabetics

STEMI

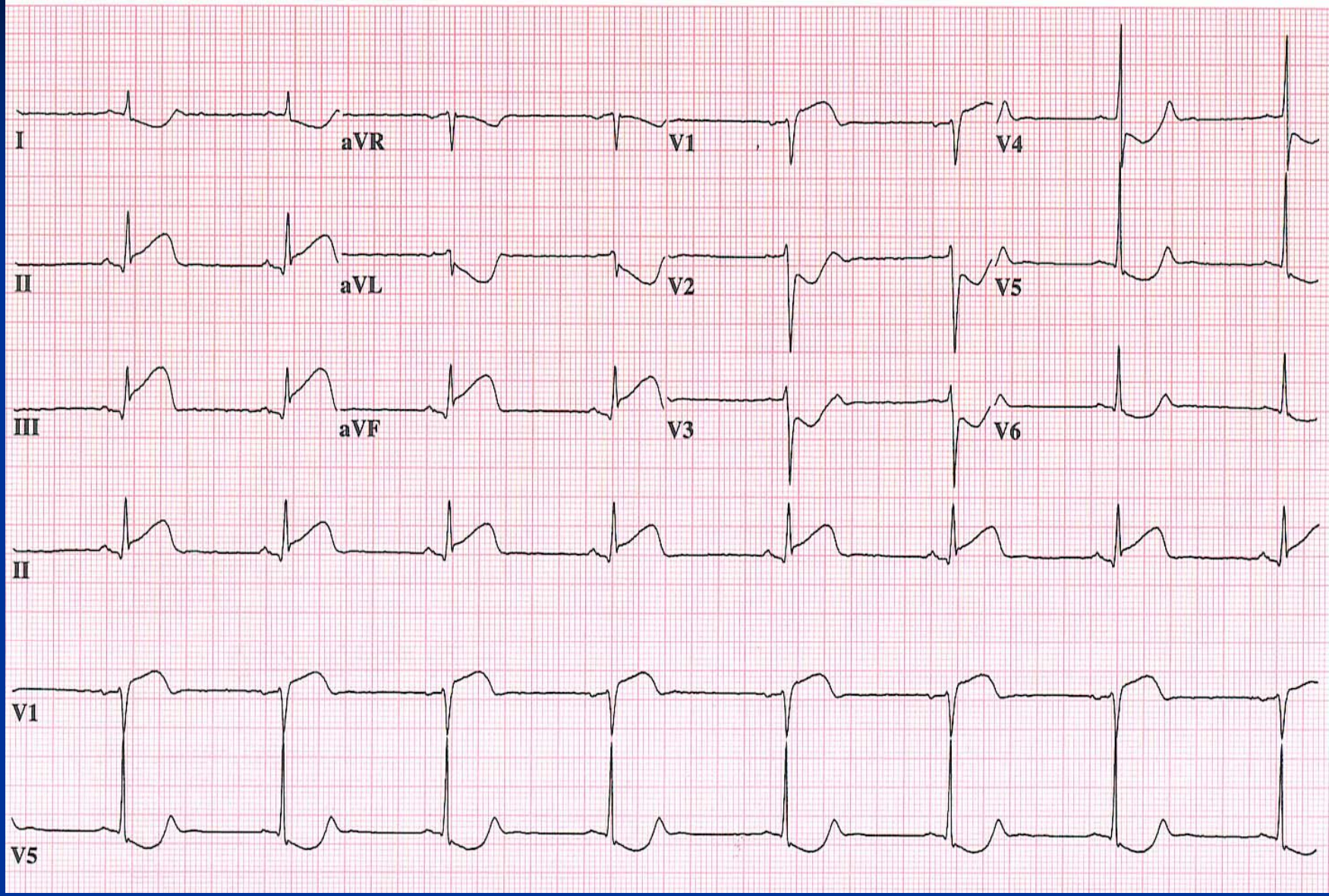
ECG 13. 52-year-old female with chest pain:



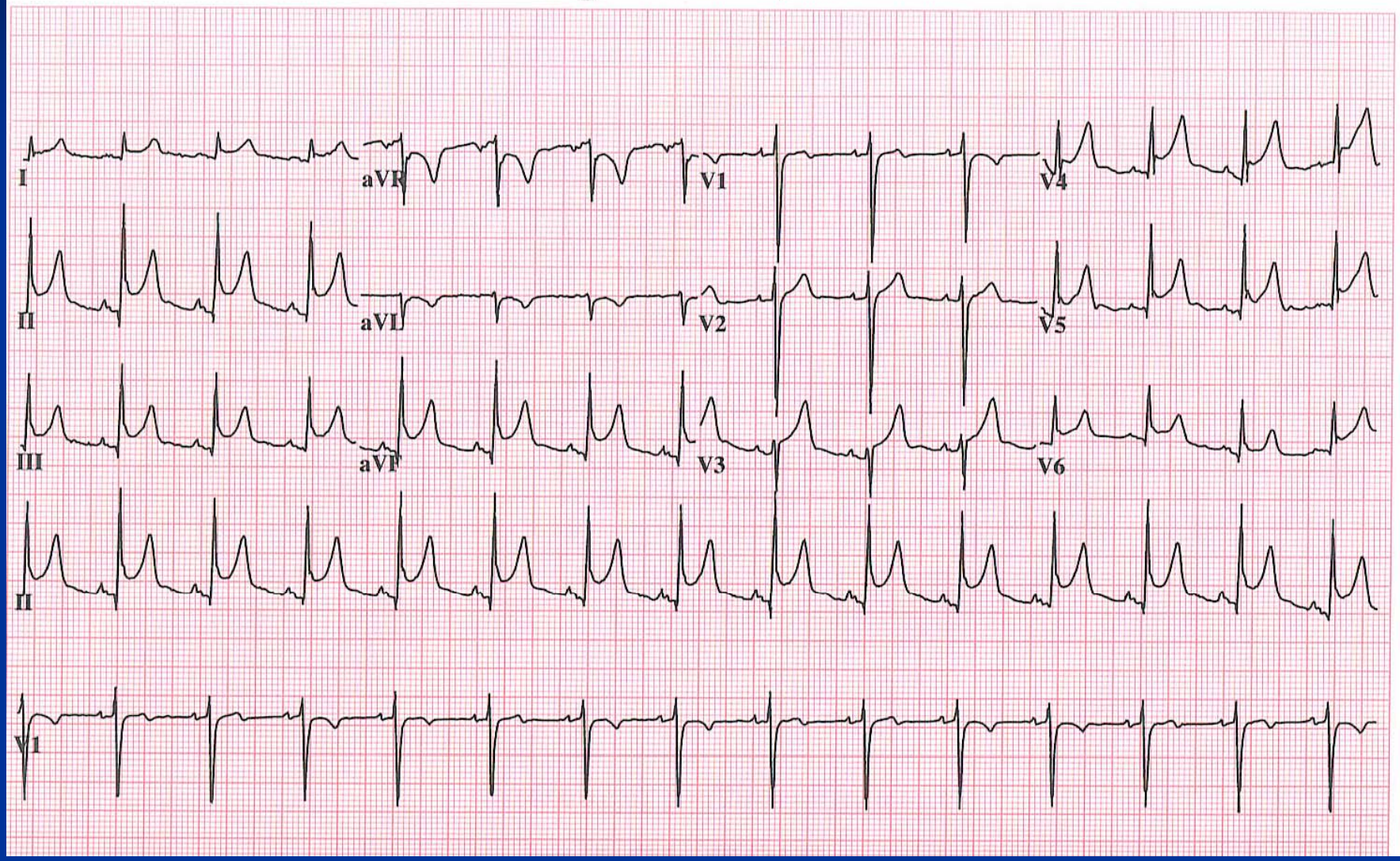
ECG 57. 76-year-old female with severe substernal chest pressure:



ECG 43. 46-year-old male with chest discomfort:



ECG 39. 59-year-old male with chest pain and cough of several days duration:



Cases Presentation

- A 79-year-old widow in excellent health, living independently, developed severe dyspnea and profound diaphoresis while eating breakfast 2 hours prior to being evaluated in the emergency department. During the days prior to this episode, she has been fully active, driving a car, and denies all symptoms. She has no important chronic illnesses, although she takes hydrochlorothiazide 25 mg daily for systolic hypertension. She is not a smoker.

  On evaluation, she is moderately uncomfortable, sweating, moderately dyspneic with minimal wheezing. Temperature is 99.2. Her blood pressure is 180/105; heart rate 105 (regular). Her mucous membranes are of good color. She has no jugular venous hypertension. Peripheral vessels are moderately tortuous and sclerotic to palpation. There is moderate wheezing but no rales on pulmonary exam. Heart sounds are somewhat distant with a Grade I to II systolic ejection murmur at the base and a possible fourth heart sound (auscultation is difficult.) The remainder of her general examination is normal including good leg pulses and no edema or tenderness in the legs.

- Electrocardiogram shows left bundle branch block and chest x-ray shows no cardiomegaly and minimal cephalization in reference to pulmonary vasculature. She was started on intravenous nitroglycerin and given an injection of furosemide 20 mg intravenously. In a hospital where all options are available, which of the following is the preferred management strategy?
  - A. Observe for 1-2 hours on the current regimen, increasing drug dose depending on patient's response.
  - B. Institute appropriate aggressive therapy for bronchospasm and suspected bronchial pneumonia.
  - C. Perform an emergency cardiac catheterization.
  - D. Institute thrombolytic therapy.
  - E. Spiral CT to determine if pulmonary embolism is present

- For which of the following clinical scenarios is thrombolytic therapy appropriate?
  - A. A 37-year-old woman with active menses, chest pain for three hours, inferior ST-segment elevation, and no history of stroke or bleeding disorder.
  - B. A 66-year-old diabetic man with crushing chest pain for 90 minutes, diaphoresis, rales, and precordial ST-segment depression.
  - C. A 72-year-old woman with dyspnea on exertion increasing progressively over 10 days, vague epigastric discomfort the day previously, and LBBB on ECG.
  - D. A 68-year-old man with out-of-hospital arrest, resuscitated in the field, nonspecific ST, and T wave changes on ECG.

- A 56-year-old man with a history of smoking and hypertension comes to the emergency department with a four-hour history of chest burning and tightness. He is treated with hydrochlorothiazide and diltiazem. He has no allergies. His stated weight is 85 kg. On exam, he is diaphoretic and anxious, but has no rales or edema. He has a fourth heart sound. His ECG shows 0.2-0.3 mV of ST-segment elevation in leads V1 through V4.

- The most appropriate course of action would be:
  - A. Administer oxygen, IV nitroglycerin, aspirin, IV heparin, and admit to a CCU for serial CK and MB as well as troponin blood tests.
  - B. Administer oxygen, IV nitroglycerin, aspirin, IV heparin, and arrange transfer to a hospital 3-4 hours away, the nearest facility with a cardiac catheterization laboratory.
  - C. Administer oxygen, morphine, aspirin, IV heparin, tenecteplase 45 mg IV bolus, and admit to CCU for observation.
  - D. Administer oxygen, IV nitroglycerin, aspirin, IV heparin, alteplase 100 mg over three hours, and arrange for urgent catheterization and possible angioplasty

- A 56-year-old patient with diabetes and hypertension is treated with thrombolysis for an anterior ST-segment elevation MI. She is seen two hours after initiation of treatment in the CCU. Which of the following scenarios suggests that coronary reperfusion has taken place?
  - A. Evidence of CHF on initial exam (dyspnea, basilar rales) are now resolved after thrombolysis and medical treatment.
  - B. Chest pain and shortness of breath are now resolved.
  - C. Chest pain but not shortness of breath are resolved, but ST-segment elevation of 0.4 mV in the precordial leads is now 0.1 mV.
  - D. The patient experienced a 22-beat run of nonsustained VT at a rate of 140, which was asymptomatic

- A 72-year-old woman presents with two hours acute onset chest pain and dyspnea. On arrival to the emergency department, her ECG reveals ST-segment elevation of 3 mm in leads V2, V3, and V4 with ST depression in avL. Which is the most appropriate reperfusion regimen?
  - A. Aspirin, accelerated alteplase (100 mg), and IV UFH (70 U/kg bolus, followed by 17 U/kg/h infusion.
  - B. Aspirin, reteplase (10 U + 10 U IV), and subcutaneous enoxaparin 1 mg/kg/bid.
  - C. Aspirin, clopidogrel (300 mg PO load), tenecteplase (0.5 mg/kg IV), and subcutaneous enoxaparin (1 mg/kg bid).
  - D. Asprin, accelerated alteplase (100 mg), and IV UFH (60 U/kg bolus followed by 12 U/kg/h infusion.
  - E. Asprin, streptokinase 1.5 million U IV, and intravenous unfractionated heparin (70 U/kg bolus followed by 15 U/kg/hour infusion.

ED Evaluation of Patients With STEMI

Differential Diagnosis of STEMI: *Life-Threatening*

- Aortic dissection
- Pulmonary embolus
- Perforating ulcer
- Tension pneumothorax
- Boerhaave syndrome (esophageal rupture with mediastinitis)

ED Evaluation of Patients With STEMI

Differential Diagnosis of STEMI: *Other Cardiovascular and Nonischemic*

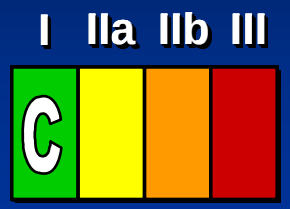
- Pericarditis
- Atypical angina
- Early repolarization
- Wolff-Parkinson-White syndrome
- Deeply inverted T-waves suggestive of a central nervous system lesion or apical hypertrophic cardiomyopathy
- LV hypertrophy with strain
- Brugada syndrome
- Myocarditis
- Hyperkalemia
- Bundle-branch blocks
- Vasospastic angina
- Hypertrophic cardiomyopathy

ED Evaluation of Patients With STEMI

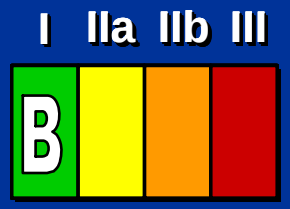
Differential Diagnosis of STEMI: *Other Noncardiac*

- Gastroesophageal reflux (GERD) and spasm
- Chest-wall pain
- Pleurisy
- Peptic ulcer disease
- Panic attack
- Cervical disc or neuropathic pain
- Biliary or pancreatic pain
- Somatization and psychogenic pain disorder

Electrocardiogram

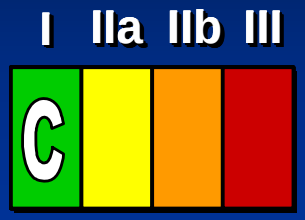


Show 12-lead ECG results to emergency physician within 10 minutes of ED arrival in all patients with chest discomfort (or anginal equivalent) or other symptoms of STEMI.



In patients with inferior STEMI, ECG leads should also be obtained to screen for right ventricular infarction.

Laboratory Examinations



Laboratory examinations should be performed as part of the management of STEMI patients, but should not delay the implementation of reperfusion therapy.

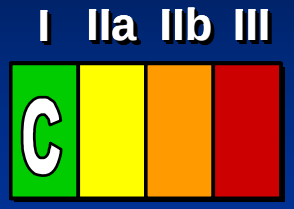
- Serum biomarkers for cardiac damage
- Complete blood count (CBC) with platelets
- International normalized ratio (INR)
- Activated partial thromboplastin time (aPTT)
- Electrolytes and magnesium
- Blood urea nitrogen (BUN)
- Creatinine
- Glucose
- Complete lipid profile

Biomarkers of Cardiac Damage

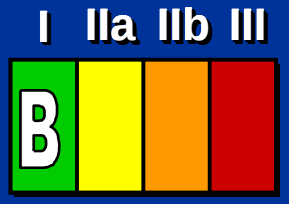
Cardiac-specific troponins should be used as the optimum biomarkers for the evaluation of patients with STEMI who have coexistent skeletal muscle injury.

For patients with ST elevation on the 12-lead ECG and symptoms of STEMI, reperfusion therapy should be initiated as soon as possible and is not contingent on a biomarker assay.

Imaging



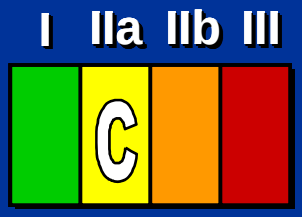
Patients with STEMI should have a portable chest X-ray, but this should not delay implementation of reperfusion therapy (unless a potential contraindication is suspected, such as aortic dissection).



Imaging studies such as a high quality portable chest X-ray, transthoracic and/or transesophageal echocardiography, and a contrast chest CT scan or an MRI scan should be used for differentiating STEMI from aortic dissection in patients for whom this distinction is initially unclear.

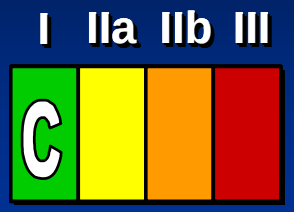
Oxygen

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

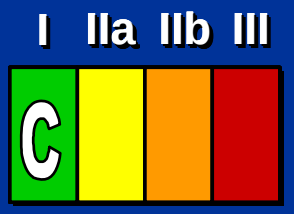


It is reasonable to administer supplemental oxygen to all patients with uncomplicated STEMI during the first 6 hours.

Nitroglycerin

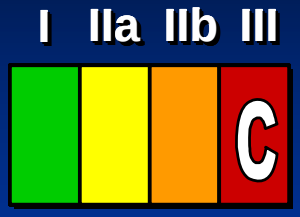


Patients with ongoing ischemic discomfort should receive sublingual NTG (0.4 mg) every 5 minutes for a total of 3 doses, after which an assessment should be made about the need for intravenous NTG.



Intravenous NTG is indicated for relief of ongoing ischemic discomfort that responds to nitrate therapy, control of hypertension, or management of pulmonary congestion.

Nitroglycerin

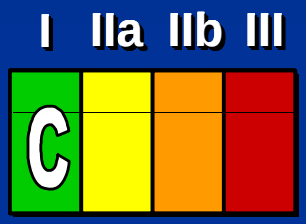


Nitrates should not be administered to patients with:

- systolic pressure < 90 mm Hg or ≥ to 30 mm Hg below baseline
- severe bradycardia (< 50 bpm)
- tachycardia (> 100 bpm) or
- suspected RV infarction.

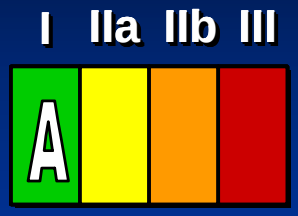
Nitrates should not be administered to patients who have received a phosphodiesterase inhibitor for erectile dysfunction within the last 24 hours (48 hours for tadalafil).

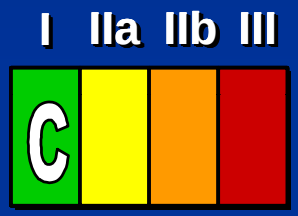
Analgesia



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

Aspirin



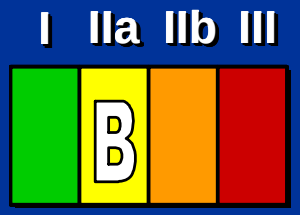


Aspirin should be chewed by patients who have not taken aspirin before presentation with STEMI. The initial dose should be 162 mg (*Level of Evidence: A*) to 325 mg (*Level of Evidence: C*)

Although some trials have used enteric-coated aspirin for initial dosing, more rapid buccal absorption occurs with non–enteric-coated formulations.

Beta-Blockers

Oral beta-blocker therapy should be administered promptly to those patients without a contraindication, irrespective of concomitant fibrinolytic therapy or performance of primary PCI.

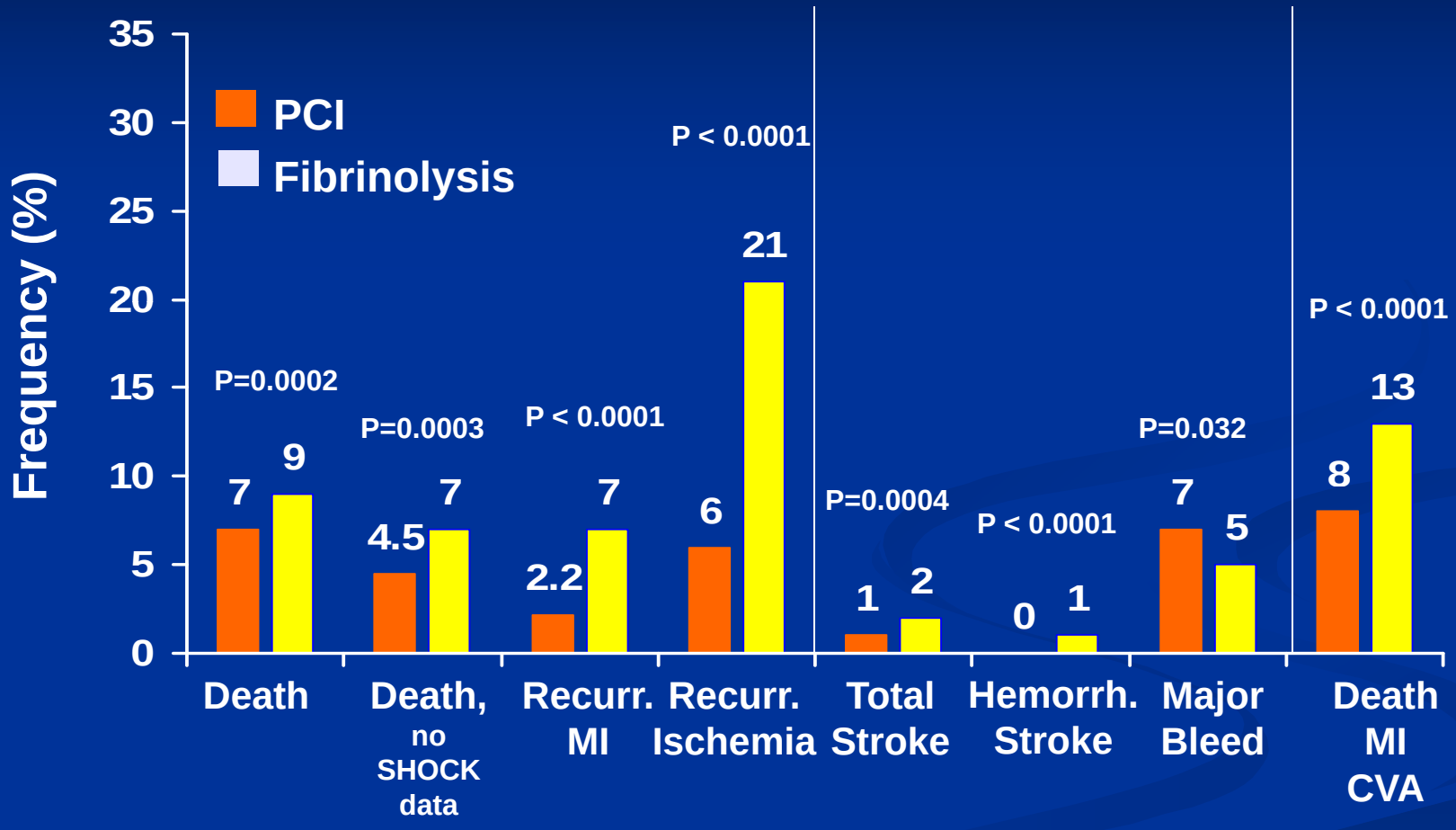


It is reasonable to administer intravenous beta-blockers promptly to STEMI patients without contraindications, especially if a tachyarrhythmia or hypertension is present.

Reperfusion

The medical system goal is to facilitate rapid recognition and treatment of patients with STEMI such that **door-to-needle** (or medical contact–to-needle) time for initiation of **fibrinolytic therapy** can be achieved **within 30 minutes** or that **door-to-balloon** (or medical contact–to-balloon) time for **PCI** can be kept **within 90 minutes**.

PCI vs Fibrinolysis for STEMI: *Short Term Clinical Outcomes*



N = 7739

Keeley et al. The Lancet 2003;361:13.

Contraindications and Cautions for Fibrinolysis in STEMI

Absolute Contraindications

- 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

NOTE: Age restriction for fibrinolysis has been removed compared with prior guidelines.

Contraindications and Cautions for Fibrinolysis in STEMI

Absolute Contraindications

- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head or facial trauma within 3 months

Contraindications and Cautions for Fibrinolysis in STEMI

Relative Contraindications

- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP > 180 mm Hg or DBP > 110 mm Hg)
- History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (> 10 minutes) CPR or major surgery (< 3 weeks)

Contraindications and Cautions for Fibrinolysis in STEMI

Relative Contraindications

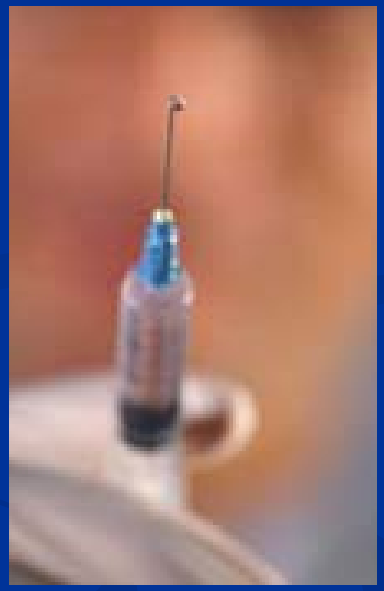
- Recent (< 2 to 4 weeks) internal bleeding
- Noncompressible vascular punctures
- For streptokinase/anistreplase: prior exposure (> 5 days ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

Reperfusion Options for STEMI Patients
Step 2: Select Reperfusion Treatment.

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

Fibrinolysis generally preferred

- *Early presentation (≤ 3 hours from symptom onset and delay to invasive strategy)*
- *Invasive strategy not an option*
  - Cath lab occupied or not available
  - Vascular access difficulties
  - No access to skilled PCI lab
- *Delay to invasive strategy*
  - Prolonged transport
  - Door-to-balloon more than 90 minutes
  - > 1 hour vs fibrinolysis (fibrin-specific agent) now



Reperfusion Options for STEMI Patients
Step 2: Select Reperfusion Treatment.

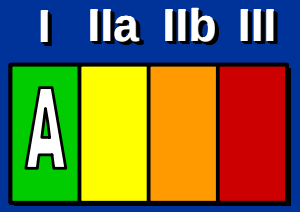
If presentation is < 3 hours and there is no delay to an invasive strategy, there is no preference for either strategy.

Invasive strategy generally preferred

- *Skilled PCI lab available with surgical backup*
  - Door-to-balloon < 90 minutes
- *High Risk from STEMI*
  - Cardiogenic shock, Killip class ≥ 3
- *Contraindications to fibrinolysis,* including increased risk of bleeding and ICH
- *Late presentation*
  - > 3 hours from symptom onset
- *Diagnosis of STEMI is in doubt*

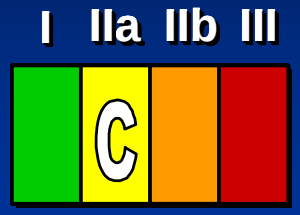
Fibrinolysis

In the absence of contraindications, fibrinolytic therapy should be administered to STEMI patients with symptom onset within the prior 12 hours.

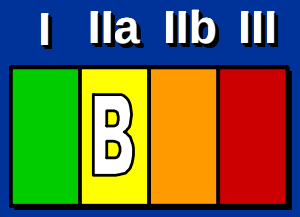


In the absence of contraindications, fibrinolytic therapy should be administered to STEMI patients with symptom onset within the prior 12 hours and new or presumably new left bundle branch block (LBBB).

Fibrinolysis

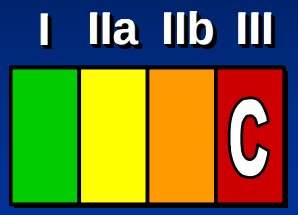


In the absence of contraindications, it is reasonable to administer fibrinolytic therapy to STEMI patients with symptom onset within the prior 12 hours and 12-lead ECG findings consistent with a true posterior MI.



In the absence of contraindications, it is reasonable to administer fibrinolytic therapy to patients with symptoms of STEMI beginning in the prior 12 to 24 hours who have continuing ischemic symptoms and ST elevation > 0.1 mV in ≥ 2 contiguous precordial leads or ≥ 2 adjacent limb leads.

Fibrinolysis



Fibrinolytic therapy should not be administered to asymptomatic patients whose initial symptoms of STEMI began more than 24 hours earlier.

Fibrinolytic therapy should not be administered to patients whose 12-lead ECG shows only ST-segment depression, except if a true posterior MI is suspected.

Criteria for Emergency Catheterization

- Symptoms or Signs of AMI <12 Hours Duration (1mm ST Elevation in 2 or More Contiguous Leads, New LBBB)
- Acute MI 12-24 Hours Duration With Continued Chest Pain
- Cardiogenic Shock Within 24 Hours (Patient Less than Age 75)
- Thrombolytic Failure Within 12 Hours of Chest Pain Onset
- Suspected Reocclusion After Thrombolytic Therapy
- ECG Evidence of True Posterior MI, Echocardiographic Wall Motion Abnormality, Positive Serum Markers, Refractory Angina, or Hemodynamic Instability/CHF

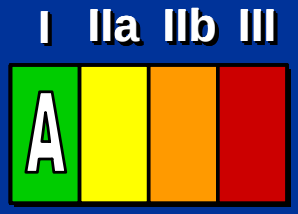
Angiographic Exclusions for the Performance of PTCA

- Unprotected Left Main Stenosis > 60%
- Infarct Related Artery Stenosis < 70% With TIMI 3 Flow
- Infarct Related Artery Supplies a Small Amount of Myocardium, Risks of PTCA Outweigh Benefits
- Inability to Identify Infarct Related Artery
- Asymptomatic Patient With Multivessel Disease, TIMI 3 Flow, and Bypass Surgery Indicated

Delayed PCI Following Thrombolysis

- Delayed PCI refers to angioplasty performed electively (1-7 days following thrombolysis) in asymptomatic patients
- Present data do not support the routine performance of delayed PCI in an asymptomatic patient following clinically successful thrombolysis
- Elective angiography and revascularization should be more targeted to post-STEMI patients with recurring symptoms, positive noninvasive stress tests, or other high-risk indicators (LVEF $\leq$0.40)

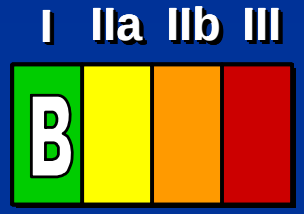
Primary PCI for STEMI: *General Considerations*



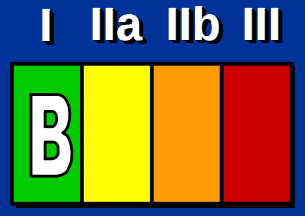
- Patient with STEMI (including posterior MI) or MI with new or presumably new LBBB
- PCI of infarct artery within 12 hours of symptom onset
- Balloon inflation within 90 minutes of presentation
- Skilled personnel available (individual performs > 75 procedures per year)
- Appropriate lab environment (lab performs > 200 PCIs/year of which at least 36 are primary PCI for STEMI)
- Cardiac surgical backup available

Primary PCI for STEMI:
Specific Considerations

Medical contact–to-balloon or door-to-balloon should be within 90 minutes.

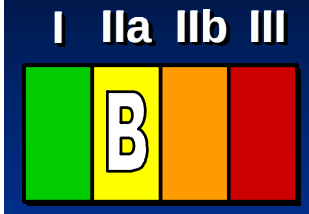


PCI preferred if > 3 hours from symptom onset.



Primary PCI should be performed in patients with severe congestive heart failure (CHF) and/or pulmonary edema (Killip class 3) and onset of symptoms within 12 hours.

Assessment of Reperfusion

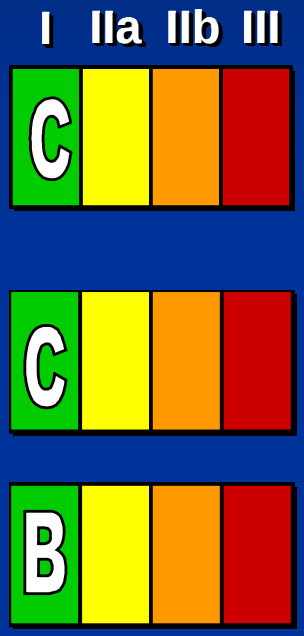


It is reasonable to monitor the pattern of ST elevation, cardiac rhythm and clinical symptoms over the 60 to 180 minutes after initiation of fibrinolytic therapy.

Noninvasive findings suggestive of reperfusion include:

- Relief of symptoms
- Maintenance and restoration of hemodynamic and/or electrical instability
- Reduction of ≥ 50% of the initial ST-segment elevation pattern on follow-up ECG 60 to 90 minutes after initiation of therapy.

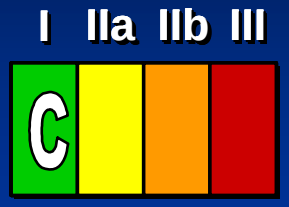
Ancillary Therapy to Reperfusion



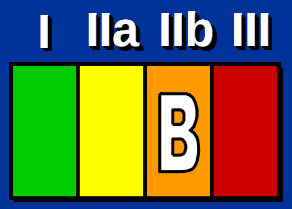
Unfractionated heparin (UFH) should be given intravenously in:

- Patients undergoing PCI or surgical revascularization
- After alteplase, reteplase, tenecteplase
- After streptokinase, anistreplase, urokinase in patients at high risk for systemic emboli.

Ancillary Therapy to Reperfusion



Platelet counts should be monitored daily in patients taking UFH.



Low molecular-weight heparin (LMWH) might be considered an acceptable alternative to UFH in patients less than 75 years who are receiving fibrinolytic therapy in the absence of significant renal dysfunction.

Enoxaparin used with tenecteplase is the most comprehensively studied.

Aspirin

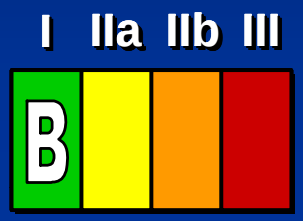
A daily dose of aspirin (initial dose of 162 to 325 mg orally; maintenance dose of 75 to 162 mg) should be given indefinitely after STEMI to all patients without a true aspirin allergy.

Thienopyridines

In patients for whom PCI is planned, clopidogrel should be started and continued:

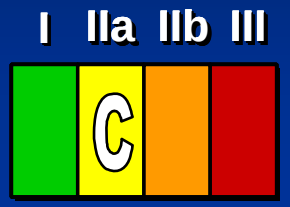
- ≥ 1 month after bare-metal stent
- ≥ 3 months after sirolimus-eluting stent
- ≥ 6 months after paclitaxel-eluting stent
- Up to 12 months in absence of high risk for bleeding.

Thienopyridines



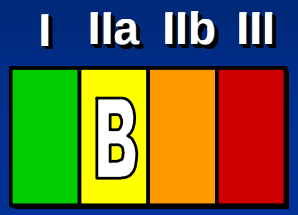
In patients taking clopidogrel in whom CABG is planned, the drug should be withheld for at least 5 days, and preferably for 7 days, unless the urgency for revascularization outweighs the risk of excessive bleeding.

Thienopyridines

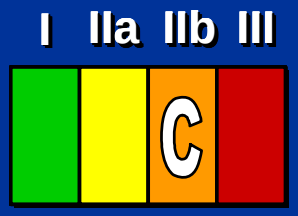


Clopidogrel is probably indicated in patients receiving fibrinolytic therapy who are unable to take aspirin because of hypersensitivity or gastrointestinal intolerance.

Glycoprotein IIb/IIIa Inhibitors



It is reasonable to start treatment with abciximab as early as possible before primary PCI (with or without stenting) in patients with STEMI.

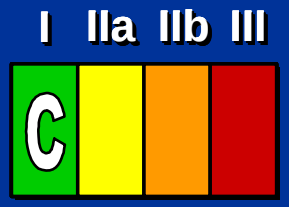


Treatment with tirofiban or eptifibatide may be considered before primary PCI (with or without stenting) in patients with STEMI.

ACE/ARB: Within 24 Hours

An ACE inhibitor should be administered orally within the first 24 hours of STEMI to the following patients without hypotension or known class of contraindications:

- Anterior infarction
- Pulmonary congestion
- LVEF < 0.40



An ARB should be given to ACE-intolerant patients with either clinical or radiological signs of HF or LVEF < 0.40.

Adjunctive Therapies for AMI

Agent	Mechanism	Clinical Effect
Aspirin	Antiplatelet	Improve survival Decrease reinfarction, CVA
Thienopyridines (clopidogrel, ticlopidine)	Antiplatelet	Recommended in aspirin-allergic patients Decrease death, MI, CVA in Non-ST ↑ ACS
Glycoprotein IIb/IIIa Inhibitors	Antiplatelet	Decrease MI, ischemic complications following primary PCI Decrease death or MI in high-risk Non-ST ↑ ACS
Unfractionated Heparin	Antithrombin	Decrease death and MI in prefibrinolytic era
Low Molecular Weight Heparins	Antithrombin	Reduce cardiac events in Non-ST ↑ ACS versus unfractionated heparin
Direct Thrombin Inhibitors	Antithrombin	Recommended in heparin-induced thrombocytopenia
Beta-Blockers	Decrease myocardial oxygen demand (↓ HR, ↓ BP)	Improve survival Reduce infarct size, ventricular arrhythmias, recurrent ischemia

Adjunctive Therapies for AMI

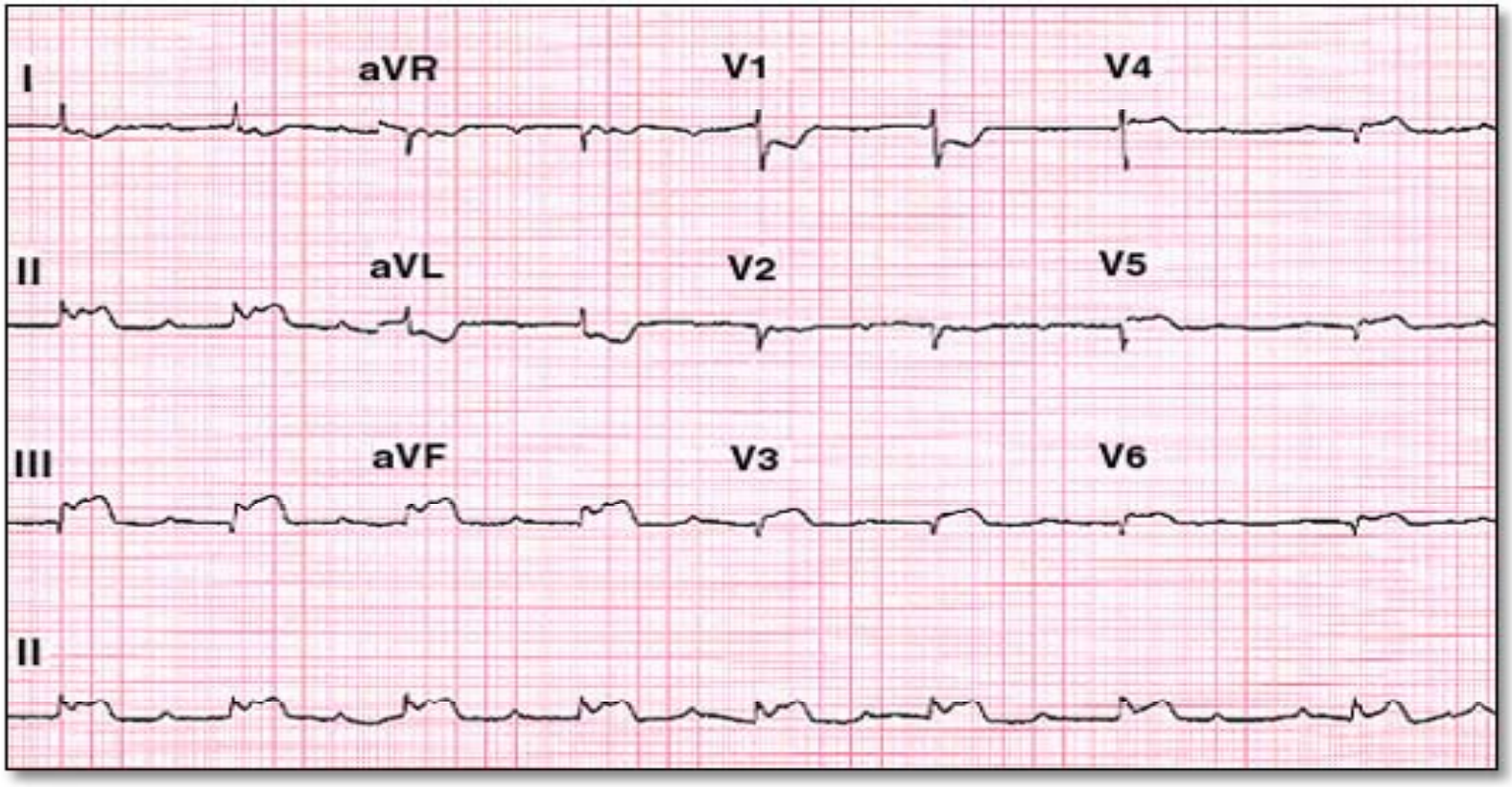
Agent	Mechanism	Clinical Effect
ACE Inhibitors	Vasodilator (↓ BP) Prevent LV remodeling	Improve survival Decrease heart failure, LV dysfunction
IV Nitroglycerine	Venous, arterial, coronary vasodilator (↓ BP, ↓ preload)	No effect on survival Decrease recurrent ischemia
HMG CoA Reductase Inhibitors	Lipid lowering Anti-inflammatory	Decrease future CV death & MI May decrease early ischemic events
Magnesium	Myocardial protective Anti-arrhythmic	Therapy for torsades depointes May improve reperfusion outcomes
Calcium Channel Blocker	Decrease myocardial oxygen demand (↓ HR, ↓ BP)	No survival benefit Possible use in beta-blocker intolerant patients without CHF or LV dysfunction
Warfarin	Oral anticoagulant	Reduced embolic risk with atrial fibrillation, LV thrombus or dysfunction
Glucose-Insulin-Potassium Infusion	Metabolic modulator	May improve intracellular myocardial energy stores and outcome

Clinical Cases

- A 75-year-old male presented with four hours of chest pain and ST-segment elevation in ECG leads II, III, aVF, and V5-6. He received tenecteplase in the ER, with resolution of the chest pain and ST-segment changes. Upon admission to the critical ICU, the patient was hemodynamically stable. TTE on hospital day two revealed normal LV systolic size and function, with no significant valvular abnormalities.

- On hospital day three, he developed acute hypotension, tachycardia, and hypoxemia, and was noted on exam to have pulmonary rales, and a new holosystolic murmur along the left parasternal border. What is your diagnosis?
  - A. Ventricular septal rupture.
  - B. Acute mitral regurgitation due to papillary muscle rupture.
  - C. Free wall rupture and tamponade.
  - D. RV infarct.

- A 60-year-old female presented to the ER with one-day history of intense left-sided chest pressure, 10 out of 10, associated with nausea, vomiting, and diaphoresis. Vital signs were: BP 141/91, pulse 80. Physical exam revealed bilateral carotid bruits, no elevated jugular venous pressure. Heart exam showed S1 S2 with S4, no murmur, and clear lungs.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

- Initial troponin I was 1.04 (peaked at 35 ng/dl). 2D echo showed 35% EF with postero-inferior hypokinesis and no major valvular heart disease. The patient had more chest pain on day 4 and was referred for heart catheterization. Cardiac cath revealed 90% lesion in the mid circumflex artery and nonobstructive disease in the LAD artery and the right coronary artery. Awaiting angioplasty of the circumflex artery, the patient suddenly became pulseless and unresponsive. ECG showed sinus tachycardia. CPR was initiated for pulseless electrical activity. What is the cause of the pulseless electrical activity arrest?
  - A. Ventricular septal rupture.
  - B. Acute mitral regurgitation due to papillary muscle rupture.
  - C. Free wall rupture and tamponade.
  - D. RV infarct

- PCI for persistent coronary occlusion days post-MI reduces cardiac events for patients with:
  - A. Post-MI rest angina.
  - B. Inducible ischemia at low threshold.
  - C. Asymptomatic patients.
  - D. All of the above.
  - E. Both A and B

- The indications for reperfusion therapy 12-24 hours after MI onset are:
  - A. Extensive ST elevation.
  - B. Hemodynamic instability.
  - C. Ongoing ischemic symptoms.
  - D. All of the above.
  - E. None of the above

AMI complications

1) Cardiogenic shock,
2) RV infarction/ischemia,
3) Ischemic mitral valve regurgitation (MR)
4) Ventricular septal defect (VSD)
5) LV free wall rupture.

Chronic Coronary Artery Disease

NCEP/Framingham Estimate of 10-Year Coronary Heart Disease Risk in Men

Age (y):	20-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79
Points:	-9	-4	0	3	6	8	10	11	12	13

	Points				
Total Cholesterol	Age 20-39y	Age 40-49y	Age 50-59y	Age 60-69y	Age 70-79y
<160 mg/dl	0	0	0	0	0
160-199	4	3	2	1	0
200-239	7	5	3	1	0
240-279	9	6	4	2	1
≥280	11	8	5	3	1

HDL Cholesterol	Points
≥60 mg/dl	-1
50-59	0
40-49	1
<40	2

	Points				
	Age 20-39y	Age 40-49y	Age 50-59y	Age 60-69y	Age 70-79y
Nonsmoker:	0	0	0	0	0
Smoker	8	5	3	1	1

	Points	
Systolic BP	Untx'ed	Tx'ed
<120 mm Hg	0	0
120-129	0	1
130-139	1	2
140-159	1	2
≥160	2	3

Points Total:	<0	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	>17
10-Year Risk (%):	<1	1	1	1	1	1	2	2	3	4	5	6	8	10	12	16	20	25	≥30

Untx'ed = Untreated; Tx'ed = Treated; y = Years

NCEP/Framingham Estimate of 10-Year Coronary Heart Disease Risk in Women

Age (y):	20-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79
Points:	-7	-3	0	3	6	8	10	12	14	16

	Points				
Total Cholesterol	Age 20-39y	Age 40-49y	Age 50-59y	Age 60-69y	Age 70-79y
<160 mg/dl	0	0	0	0	0
160-199	4	3	2	1	1
200-239	8	6	4	2	1
240-279	11	8	5	3	2
≥280	13	10	7	4	2

HDL Cholesterol	Points
≥60 mg/dl	-1
50-59	0
40-49	1
<40	2

	Points				
	Age 20-39y	Age 40-49y	Age 50-59y	Age 60-69y	Age 70-79y
Nonsmoker:	0	0	0	0	0
Smoker	9	7	4	2	1

	Points	
Systolic BP	Untx'ed	Tx'ed
<120 mm Hg	0	0
120-129	1	3
130-139	2	4
140-159	3	5
≥160	4	6

Points Total:	<9	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	>25
10-Year Risk (%):	<1	1	1	1	1	2	2	3	4	5	6	8	11	14	17	22	27	≥30

Untx'ed = Untreated; Tx'ed = Treated; y = Years

- Exercise testing for coronary disease is designed to provoke subendocardial ischemia by graded effort, and exercise ECG seeks to identify provoked ischemia on recordings taken from the body surface. Applications of exercise ECG testing for the evaluation of CV risk include:
  - Identification of coronary artery obstruction in symptomatic and asymptomatic populations with varying pretest likelihood of disease.
  - Assessment of the anatomic and functional severity of coronary disease.
  - Prediction of CV morbidity and mortality.
  - The exercise test is also widely used as a measurement of the response to therapeutic intervention. Findings on the ECG during routine exercise testing may be combined with symptomatic, physiologic, and demographic data to produce additional predictive scores.

ACC/AHA/ACP-ASIM Recommendations for Diagnosis of Obstructive CAD With Exercise ECG Testing Without an Imaging Modality

(Adapted from Reference 2)

Class I: Conditions for which there is evidence or general agreement that a given procedure or treatment is useful and effective.

Patients with an intermediate pretest probability of CAD based on age, gender and symptoms, including those with complete right bundle-branch block or <1 mm of ST depression at rest (exceptions are listed below in classes II and III).

Class IIa: Conditions for which there is conflicting evidence or a divergence of opinion about the usefulness/efficacy of a procedure or treatment, and the weight of evidence/opinion is in favor of usefulness/efficacy.

Patients with suspected vasosplastic angina.

Class IIb: Conditions for which there is conflicting evidence or a divergence of opinion about the usefulness/efficacy of a procedure or treatment, and usefulness/efficacy is less well established by evidence/opinion.

1. Patients with a high pretest probability of CAD by age, gender and symptoms.
2. Patients with a low pretest probability of CAD by age, gender and symptoms.
3. Patients taking digoxin whose ECG has <1 mm of baseline ST-segment depression.
4. Patients with ECG criteria for LV hypertrophy and <1 mm of baseline ST segment depression.

Class III: Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/effective and in some cases may be harmful.

1. Patients with the following baseline ECG abnormalities.
    a. Pre-excitation (Wolff-Parkinson-White) syndrome.
    b. Electronically paced ventricular rhythm.
    c. More than 1 mm of ST depression at rest.
    d. Complete left bundle-branch block.
2. Patients with an established diagnosis of CAD due to prior MI or coronary angiography; however, testing can assess functional capacity and prognosis.

- Factors associated with false positive tests (low specificity) include:
  - LV hypertrophy.
  - Digitalis, other drugs, and electrolyte abnormalities.
  - Resting ST-segment depression >0.1 mV.
  - LBBB.
  - Wolff-Parkinson-White syndrome.

Clinical Cases

- The patient is a 60-year-old obese man with recurrent angina at work who develops 3 mm of ST-segment depression after five minutes on a Bruce protocol stress test, with a moderate area of inducible anterior wall hypokinesis on a treadmill stress echo. He does a moderate amount of heavy lifting at work. His resting pulse is 55 bpm, BP is 120/70 mm Hg, with an LDL of 105 and a triglyceride of 180; and medications include moderate doses of metoprolol, amlodipine, aspirin, and a statin

- The appropriate next step in his management would be:
  - A. Increase his beta-blocker dose higher to reach a resting heart rate of 50 bpm.
  - B. Add a nitrate and an ACE inhibitor to his regimen and then repeat the stress test.
  - C. Recommend a coronary angiogram to determine if he is a revascularization candidate, and continue to optimize his lifestyle and medical regimens.
  - D. Encourage the patient to work harder on weight loss and try to titrate his antianginal medications higher.

- A 67-year-old nonsmoking, normotensive female attorney with an unremarkable physical examination and normal resting ECG is referred for exercise ECG testing because of several episodes of nonradiating central chest pain of 5-10 minutes' duration. She notes no clear relationship of her symptom to physical activity, but she has never exercised regularly and rarely uses stairs. On two occasions, the pain was associated with large meals, once just after unusual use of espresso coffee that resolved 10 minutes after chewing an antacid tablet, and once after an argument with her daughter. After one minute of Stage II of the Bruce protocol, at a heart rate of 92, there is convex upward coving of the ST segments in V1 through V4, and within the next minute, this becomes 2-3 mm (0.2-0.3 mV) of ST elevation 60 msec after the j-point in these leads..

- You stop the test just as the patient notes the onset of mild retrosternal discomfort that is similar to her presenting complaint. The most likely explanation for these ECG findings is:
  - A. Occult prior infarction with aneurysm of the anterior LV wall.
  - B. Spasm of an otherwise unobstructed LAD coronary artery.
  - C. High-grade proximal obstruction of the LAD coronary artery.
  - D. Acute anterior infarction due to dissection of the left anterior coronary artery

- A 52-year-old male accountant is referred for exercise testing because of recent onset angina. There is a history of hypertension for 10 years, now treated with atenolol and quinapril, and mild hyperlipidemia treated with atorvastatin. The resting BP on examination is 142/90, and there is a grade 2/6 early systolic ejection murmur and a mildly sustained LV impulse, but with good carotid upstrokes. The ECG reveals voltage criteria for LV hypertrophy with 1.5 mm downsloping ST depression in the lateral leads that is consistent with typical "strain" pattern. A resting echocardiogram done to clarify the aortic outflow murmur reveals no significant obstruction, normal LVEF, and mild septal and free-wall thickening without dilatation. After three minutes of level walking on the treadmill, the heart rate is 72 (atenolol was held on the day of testing), the BP has risen from 145/90 to 155/90, and there is 0.5 mm of additional ST depression in the inferolateral leads.

- After completion of Stage I of the Bruce protocol, there is mild chest pressure, the heart rate is 78, and the BP is 140/90, and there is now 1.0 mm of additional depression. Treadmill exercise is continued for another two minutes until it is stopped because of increasing substernal chest pressure and dyspnea, at which time the heart rate is 84. The BP is not recorded until two minutes later, at which time it is 160/95. Review of the peak exercise ECG reveals a total of 1.5 mm of additional ST depression, which returns to baseline after nine minutes of recovery.

- What is the most appropriate conclusion about this test?
  - A. This was the correct test to perform, the test was terminated appropriately, and the test findings are most consistent with functionally extensive coronary disease.
  - B. This was the wrong test to perform, the test was terminated appropriately, and the test findings are most consistent with functionally extensive coronary disease.
  - C. This was the wrong test to perform, the test was terminated too late, and the test findings are more consistent with hypertensive heart disease than with coronary disease.
  - D. This was the wrong test to perform, the test was terminated too late, and the test findings are most consistent with functionally extensive coronary disease.

Noninvasive Test Results Predicting High Risk for Adverse Outcomes

- **Exercise ECG Testing**
  - Abnormal Horizontal or Down-Sloping ST-Segment Depression With:
    - Onset at Heart Rate <120 BPM or ≤6.5 METS
    - Magnitude ≥2.0 mm
    - Post-Exercise Duration of ≥6 Minutes
    - Depression in Multiple Leads
  - Abnormal Systolic BP Response
    - With Sustained Decrease of >10 mm Hg or Flat BP Response ≤130 mm Hg, Associated with Abnormal ECG
  - Exercise-Induced ST-Segment Elevation
  - Ventricular Tachycardia
- **Radionuclide Myocardial Perfusion Imaging**
  - Abnormal Myocardial Tracer Distribution in More Than One Coronary Artery Region at Rest or With Stress or an Anterior Defect That Reperfuses
  - Abnormal Myocardial Distribution With Increased Lung Uptake
  - Reversible Cavity Dilatation
- **LV Imaging**
  - Stress Radionuclide Ventriculography
    - Exercise EF ≤50%
    - Rest EF ≤35%
    - Fall in Exercise EF ≥10%
  - Stress Echocardiography
    - Rest EF ≤35%
    - Wall Motion Score Index >1

Class I Indications for Revascularization for Chronic Stable Angina

1. CABG for patients with significant left main coronary disease.
2. CABG for patients with three-vessel disease. The survival benefit is greater in patients with abnormal LV systolic function (EF <50%).
3. CABG for patients with two-vessel disease with significant proximal LAD CAD and either abnormal LV systolic function (EF <50%) or demonstrable ischemia on noninvasive testing.
4. PCI for patients with two- or three-vessel obstructive disease with significant proximal LAD disease, who have anatomy suitable for catheter-based therapy, normal LV systolic function, and who do not have diabetes (Level of evidence B).
5. PCI or CABG for patients with one- or two-vessel CAD without significant proximal LAD disease, but with a large area of viable myocardium and high-risk criteria on noninvasive testing.
6. In patients with prior PCI, CABG, or PCI for recurrent stenosis associated with a large area of viable myocardium or high-risk criteria on noninvasive testing.
7. PCI or CABG for patients who have not been successfully treated by medical therapy and can undergo revascularization with acceptable risk.

Secondary Prevention Definition

- Therapy to reduce recurrent cardiovascular events and decrease cardiovascular mortality in patients with established atherosclerotic vascular disease
- Patients covered include those with established coronary and other atherosclerotic vascular disease, including peripheral arterial disease, atherosclerotic aortic disease and carotid artery disease
- Individuals with sub-clinical atherosclerosis and patients whose only manifestation is diabetes are covered in other guidelines

Components of Secondary Prevention

Cigarette smoking cessation Blood pressure control Lipid management to goal Physical activity Weight management to goal Diabetes management to goal
Antiplatelet agents / anticoagulants
Renin angiotensin aldosterone system blockers
Beta blockers
Influenza vaccination

Cigarette Smoking Recommendations

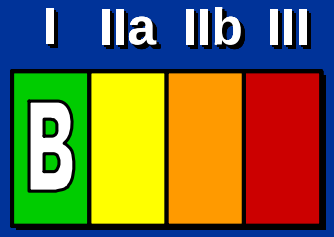
I	IIa	IIb	III
B			

Goal: Complete Cessation and No Exposure to Environmental Tobacco Smoke

- Ask about tobacco use status at every visit.
- Advise every tobacco user to quit.
- Assess the tobacco user's willingness to quit.
- Assist by counseling and developing a plan for quitting.
- Arrange follow-up, referral to special programs, or pharmacotherapy (including nicotine replacement and bupropion.
- Urge avoidance of exposure to environmental tobacco smoke at work and home.

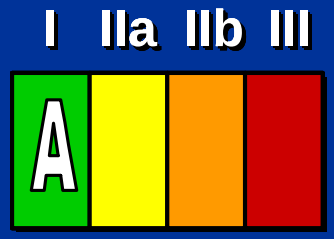
Blood Pressure Control Recommendations

Goal: <140/90 mm Hg or <130/80 if diabetes or chronic kidney disease



Blood pressure 120/80 mm Hg or greater:

- Initiate or maintain lifestyle modification: weight control, increased physical activity, alcohol moderation, sodium reduction, and increased consumption of fresh fruits vegetables and low fat dairy products



Blood pressure 140/90 mm Hg or greater (or 130/80 or greater for chronic kidney disease or diabetes)

- As tolerated, add blood pressure medication, treating initially with beta blockers and/or ACE inhibitors with addition of other drugs such as thiazides as needed to achieve goal blood pressure

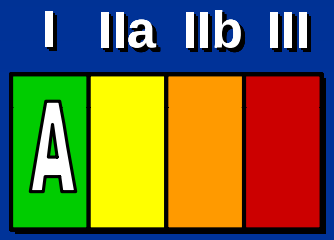
JNC VII Lifestyle Modifications for BP Control

Modification	Recommendation	Approximate SBP Reduction Range
Weight reduction	Maintain normal body weight (BMI=18.5-24.9)	5-20 mmHg/10 kg weight lost
Adopt DASH eating plan	Diet rich in fruits, vegetables, low fat dairy and reduced in fat	8-14 mmHg
Restrict sodium intake	<2.4 grams of sodium per day	2-8 mmHg
Physical activity	Regular aerobic exercise for at least 30 minutes on most days of the week	4-9 mmHg
Moderate alcohol consumption	≤2 drinks/day for men and ≤1 drink/day for women	2-4 mmHg

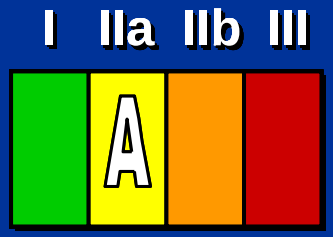
BMI=Body mass index, SBP=Systolic blood pressure

Chobanian AV et al. *JAMA*. 2003;289:2560-2572

Lipid Management Goal



LDL-C should be less than 100 mg/dL

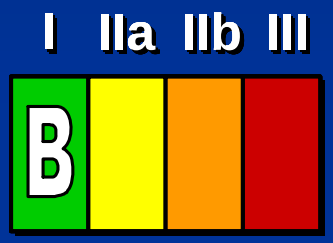


Further reduction to LDL-C to < 70 mg/dL is reasonable

If TG ≥200 mg/dL, non-HDL-C should be < 130 mg/dL*

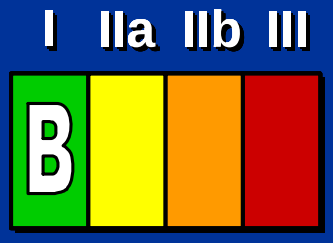
*Non-HDL-C = total cholesterol minus HDL-C

Lipid Management Recommendations



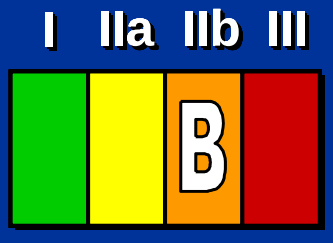
For all patients

Start dietary therapy (<7% of total calories as saturated fat and <200 mg/d cholesterol)



Adding plant stanol/sterols (2 gm/day) and viscous fiber (>10 mg/day) will further lower LDL

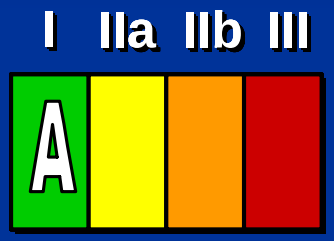
Promote daily physical activity and weight management.



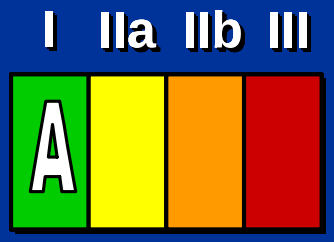
Encourage increased consumption of omega-3 fatty acids in fish or 1 g/day omega-3 fatty acids in capsule form for risk reduction.

Lipid Management Recommendations

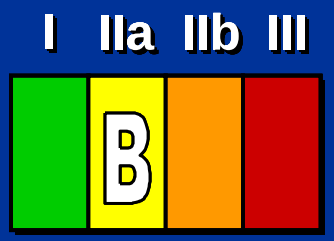
Assess fasting lipid profile in all patients, and within 24 hours of hospitalization for those with an acute event. For patients hospitalized, initiate lipid-lowering medication as recommended below prior to discharge according to the following schedule:



If baseline LDL-C ≥ 100 mg/dL, initiate LDL-lowering drug therapy



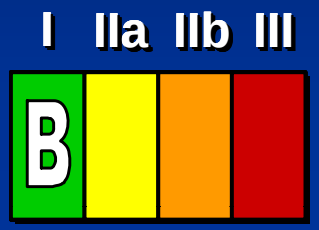
If on-treatment LDL-C ≥ 100 mg/dL, intensify LDL-lowering drug therapy (may require LDL lowering drug combination)



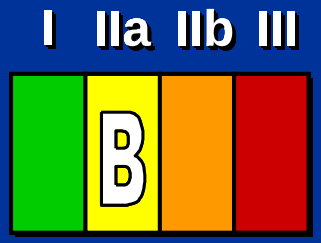
If baseline is LDL-C 70 to 100 mg/dL, it is reasonable to treat to LDL < 70 mg/dL

When LDL lowering medications are used, obtain at least a 30-40% reduction in LDL-C levels.

Lipid Management Recommendations

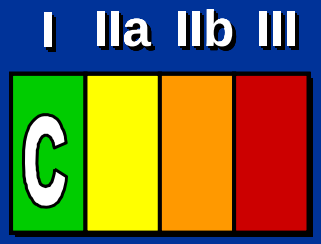


If TG are 200-499 mg/dL, non-HDL-C should be < 130 mg/dL



Further reduction of non-HDL to < 100 mg/dL is reasonable

Therapeutic options to reduce non-HDL-C:
More intense LDL-C lowering therapy I (B) or
Niacin (after LDL-C lowering therapy) IIa (B) or
Fibrate (after LDL-C lowering therapy) IIa (B)



If TG are ≥ 500 mg/dL, therapeutic options to prevent pancreatitis are fibrate or niacin before LDL lowering therapy; and treat LDL-C to goal after TG-lowering therapy. Achieve non-HDL-C < 130 mg/dL, if possible

Physical Activity Recommendations

I IIa IIb III

B

Goal: 30 minutes 7 days/week, minimum 5 days/week

Assess risk with a physical activity history and/or an exercise test, to guide prescription

I IIa IIb III

B

Encourage 30 to 60 minutes of moderate intensity aerobic activity such as brisk walking, on most, preferably all, days of the week, supplemented by an increase in daily lifestyle activities

I IIa IIb III

B

Advise medically supervised programs for high-risk patients (e.g. recent acute coronary syndrome or revascularization, HF)

Weight Management Recommendations

I IIa IIb III

B

I IIa IIb III

B

I IIa IIb III

A

Goal: BMI 18.5 to 24.9 kg/m2
Waist Circumference: Men: < 40 inches
Women: < 35 inches

Assess BMI and/or waist circumference on each visit and consistently encourage weight maintenance/ reduction through an appropriate balance of physical activity, caloric intake, and formal behavioral programs when indicated.

If waist circumference (measured at the iliac crest) ≥35 inches in women and ≥40 inches in men initiate lifestyle changes and consider treatment strategies for metabolic syndrome as indicated.

The initial goal of weight loss therapy should be to reduce body weight by approximately 10 percent from baseline. With success, further weight loss can be attempted if indicated.

*BMI is calculated as the weight in kilograms divided by the body surface area in meters^2.
Overweight state is defined by BMI=25-30 kg/m2. Obesity is defined by a BMI >30 kg/m2.

Diabetes Mellitus Recommendations

Goal: Hb A1c < 7%

I IIa IIb III

B

Lifestyle and pharmacotherapy to achieve near normal HbA1C (<7%).

I IIa IIb III

B

Vigorous modification of other risk factors (e.g., physical activity, weight management, blood pressure control, and cholesterol management as recommended).

I IIa IIb III

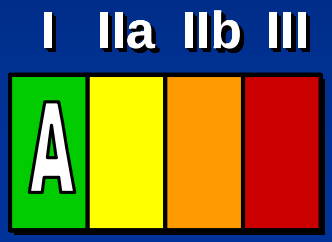
C

Coordinate diabetic care with patient's primary care physician or endocrinologist.)

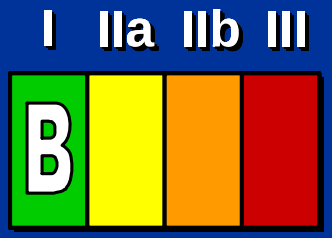
HbA1c = Glycosylated hemoglobin

Antiplatelet Agents / Anticoagulation Recommendations

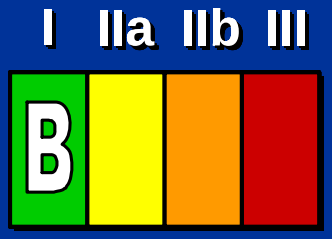
Aspirin Recommendations



Start and continue indefinitely aspirin 75 to 162 mg/d in all patients unless contraindicated

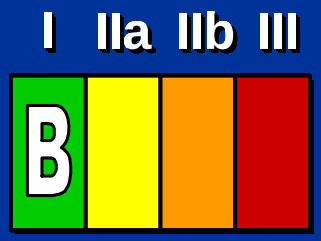


For patients undergoing CABG, aspirin (100 to 325 mg/d) should be started within 48 hours after surgery to reduce saphenous vein graft closure



Post-PCI-stented patients should receive 325 mg per day of aspirin for 1 month for bare metal stent, 3 months for sirolimus-eluting stent and 6 months for paclitaxel-eluting stent

Clopidogrel Recommendations



Start and continue clopidogrel 75 mg/d in combination with aspirin

for post ACS or post PCI with stent placement patients for up to 12 months

for post PCI-stented patients

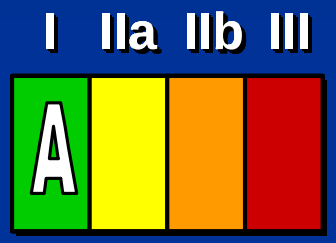
≥1 month for bare metal stent,

≥3 months for sirolimus-eluting stent

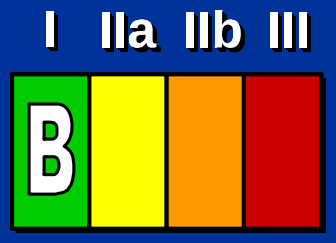
≥6 months for paclitaxel-eluting stent

*Clopidogrel is generally given preference over Ticlopidine because of a superior safety profile

Anticoagulation Recommendations



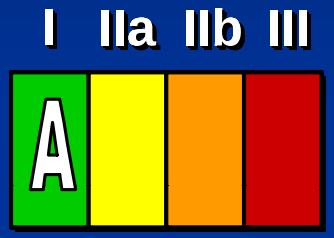
Manage warfarin to international normalized ratio 2.0 to 3.0 for paroxysmal or chronic atrial fibrillation or flutter, and in post-MI patients when clinically indicated (e.g., atrial fibrillation, LV thrombus.)



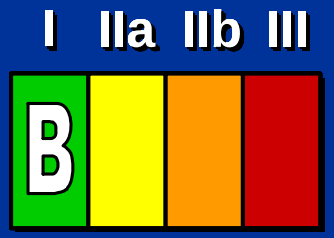
Use of warfarin in conjunction with aspirin and/or clopidogrel is associated with increased risk of bleeding and should be monitored closely

Renin-Angiotensin-Aldosterone System Blockers Recommendations

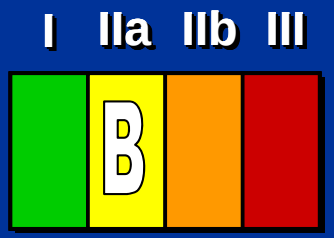
ACE Inhibitor Recommendations



Use in all patients with LVEF ≤ 40%, and those with diabetes or chronic kidney disease indefinitely, unless contraindicated



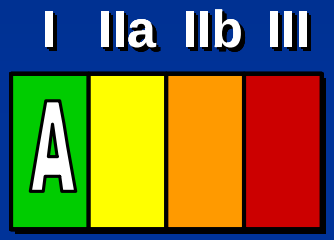
Consider for all other patients



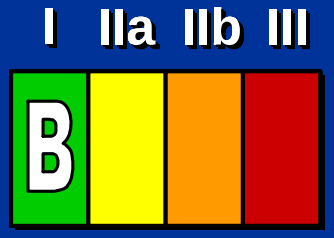
Among lower risk patients with normal LVEF where cardiovascular risk factors are well controlled and where revascularization has been performed, their use may be considered optional

ACE=Angiotensin converting enzyme, LVEF= left ventricular ejection fraction

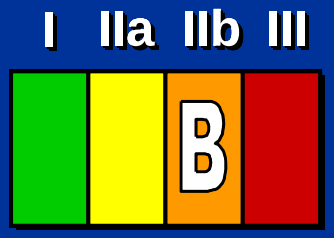
Angiotensin Receptor Blocker Recommendations



Use in patients who are intolerant of ACE inhibitors with HF or post MI with LVEF less than or equal to 40%.



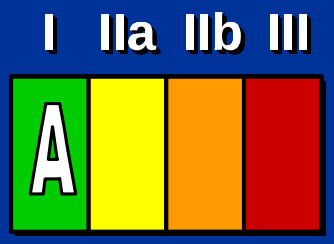
Consider in other patients who are ACE inhibitor intolerant.



Consider use in combination with ACE inhibitors in systolic dysfunction HF.

ACE=Angiotensin converting enzyme inhibitor, LVEF=Left Ventricular Ejection fraction, HF=Heart failure, MI=Myocardial infarction

Aldosterone Antagonist Recommendations



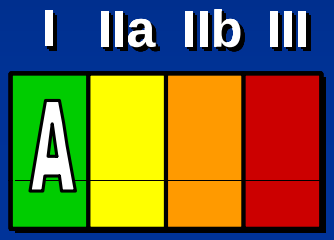
Use in post MI patients, without significant renal dysfunction or hyperkalemia, who are already receiving therapeutic doses of an ACE inhibitor and beta blocker, have an LVEF $\leq$ 40% and either diabetes or heart failure

*Contraindications include abnormal renal function (creatinine >2.5 mg/dL in men or >2.0 mg/dL in women) and hyperkalemia (K+ $\geq$5.0 meq/L)

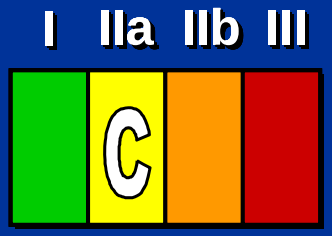
ACE=Angiotensin converting enzyme inhibitor, LVEF=Left Ventricular Ejection fraction, MI=Myocardial infarction

β-blocker Recommendations

β-blocker Recommendations



Start and continue indefinitely in all post MI, ACS, LV dysfunction with or without HF symptoms, unless contraindicated.



Consider chronic therapy for all other patients with coronary or other vascular disease or diabetes unless contraindicated.

*Precautions but still indicated include mild to moderate asthma or chronic obstructive pulmonary disease, insulin dependent diabetes mellitus, severe peripheral arterial disease, and a PR interval >0.24 seconds.

MI=Myocardial infarction, HF=Heart Failure

Secondary Prevention Conclusions

- Evidence confirms that aggressive comprehensive risk factor management improves survival, reduces recurrent events and the need for interventional procedures, and improves the quality of life for these patients.
- Every effort should be made to ensure that patients are treated with evidence-based, guideline recommended, life-prolonging therapies in the absence of contraindications or intolerance.