Evaluation of Acute Coronary Syndromes

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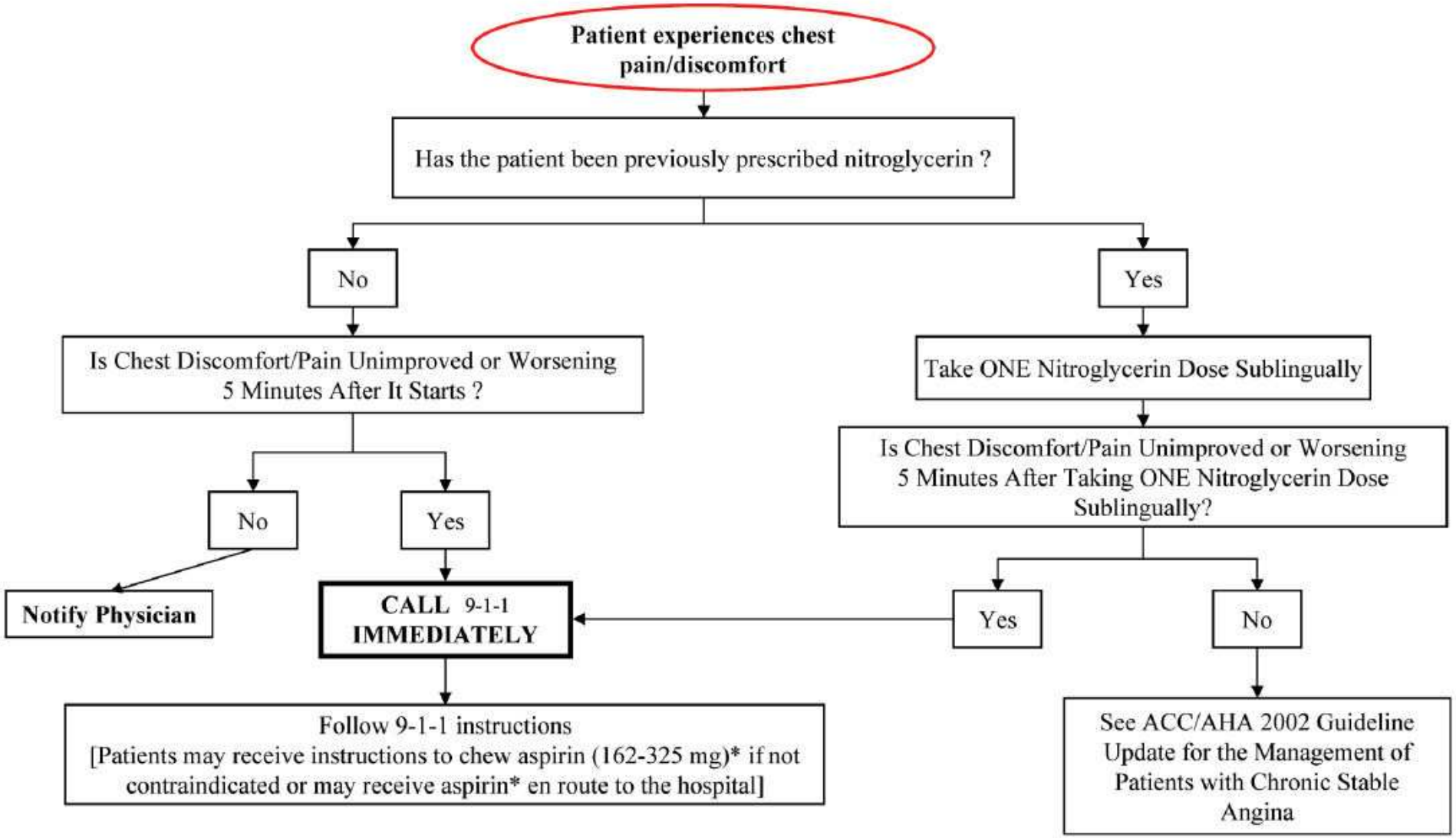
January 26, 2006

Epidemiology

- 1,680,000 unique discharges for ACS in 2001
- Applying the conservative estimate of 30% of the ACS patients who have STEMI from the National Registry of Myocardial Infarction [NRMI-4], we estimate 500 000 STEMI events per year in the U.S.
- However, there has been a steady decline in the mortality rate from STEMI over the last several decades.
- This appears to be due to a combination of a fall in the incidence of MI (replaced in part by an increase in the incidence of unstable angina) and a reduction in the case fatality rate once an MI has occurred.
- There has been a progressive increase in the proportion of patients who present with NSTEMI compared with STEMI.



ACC 2004 STEMI

ACS –Unstable Angina, NSTEMI, and STEMI

- Acute coronary syndrome
  - UA
  - NSTEMI
  - STEMI
- UA differentiated from NSTEMI by absence of biomarkers
  - Troponin, CPK/MB

TABLE 46–2 Revised Definition of Myocardial Infarction (MI)

Criteria for acute, evolving, or recent MI
Either one of the following criteria satisfies the diagnosis for an acute, evolving, or recent MI:
1. Typical rise and gradual fall (troponin) or more rapid rise and fall (CK-MB) of biochemical markers of myocardial necrosis with at least one of the following: a. Ischemic symptoms b. Development of pathologic Q waves on the ECG reading c. ECG changes indicative of ischemia (ST-segment elevation or depression) d. Coronary artery intervention (e.g., coronary angioplasty)
2. Pathological findings of an acute MI
Criteria for established MI
Either of the following criteria satisfies the diagnosis for established MI:
1. Development of new pathological Q waves on serial ECG readings. The patient may or may not remember previous symptoms. Biochemical markers of myocardial necrosis may have normalized, depending on the length of time that has passed since the infarct developed.
2. Pathological findings of a healed or healing MI

CK = creatine kinase; ECG = electrocardiographic.

From Alpert JS, Thygesen K, Antman E, et al: Myocardial infarction redefined—A consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. J Am Coll Cardiol 36:959, 2000.

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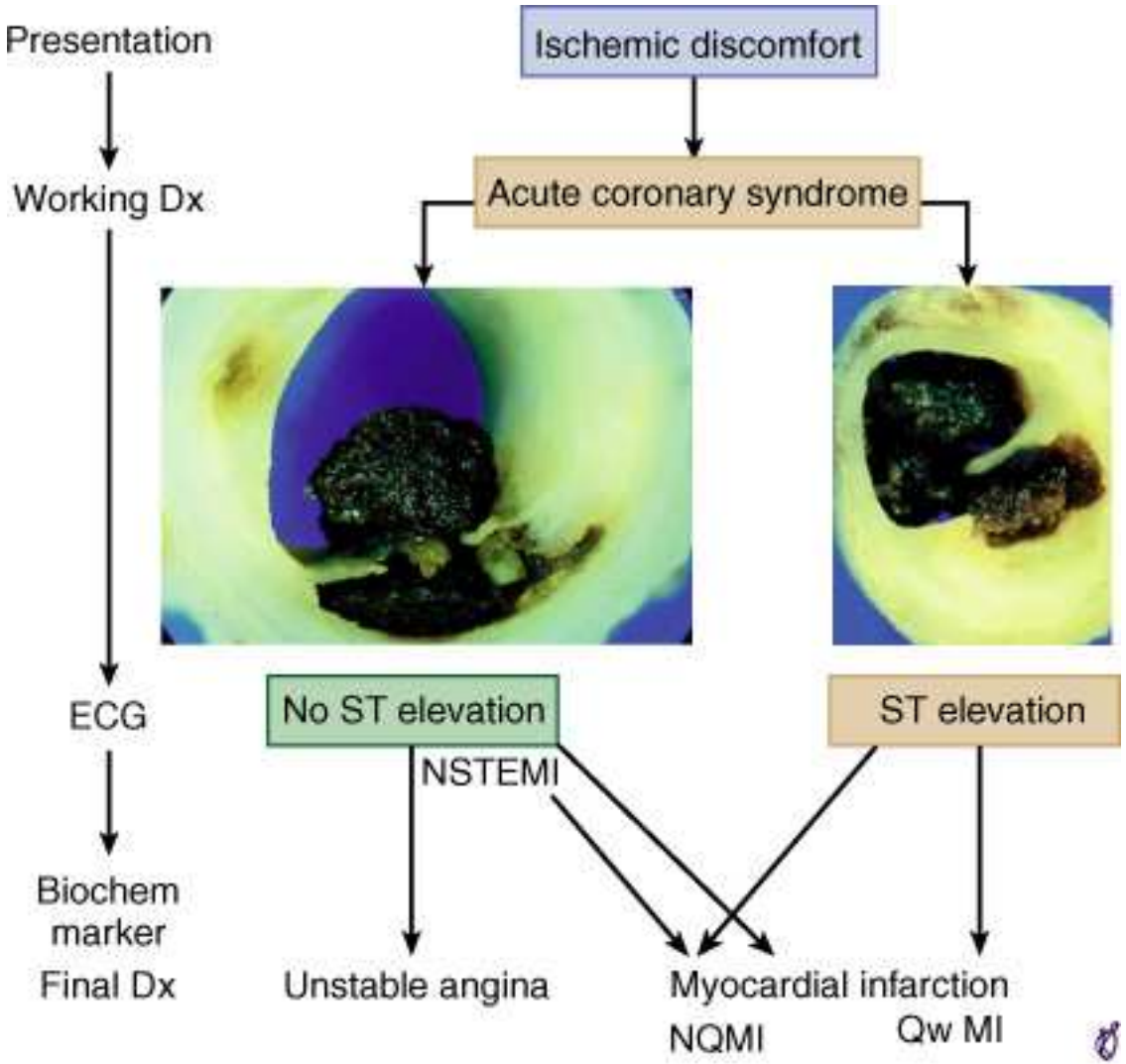
ACS



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Etiology of UA

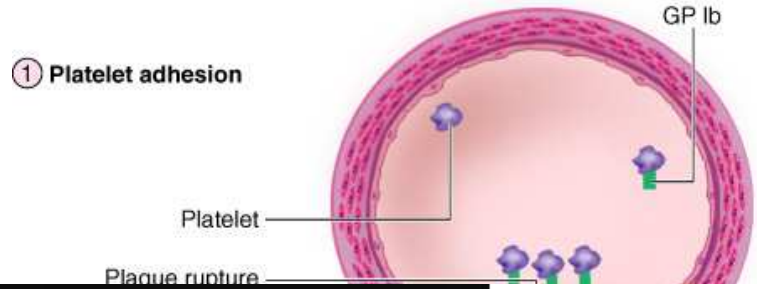
Table 2. Causes of UA*

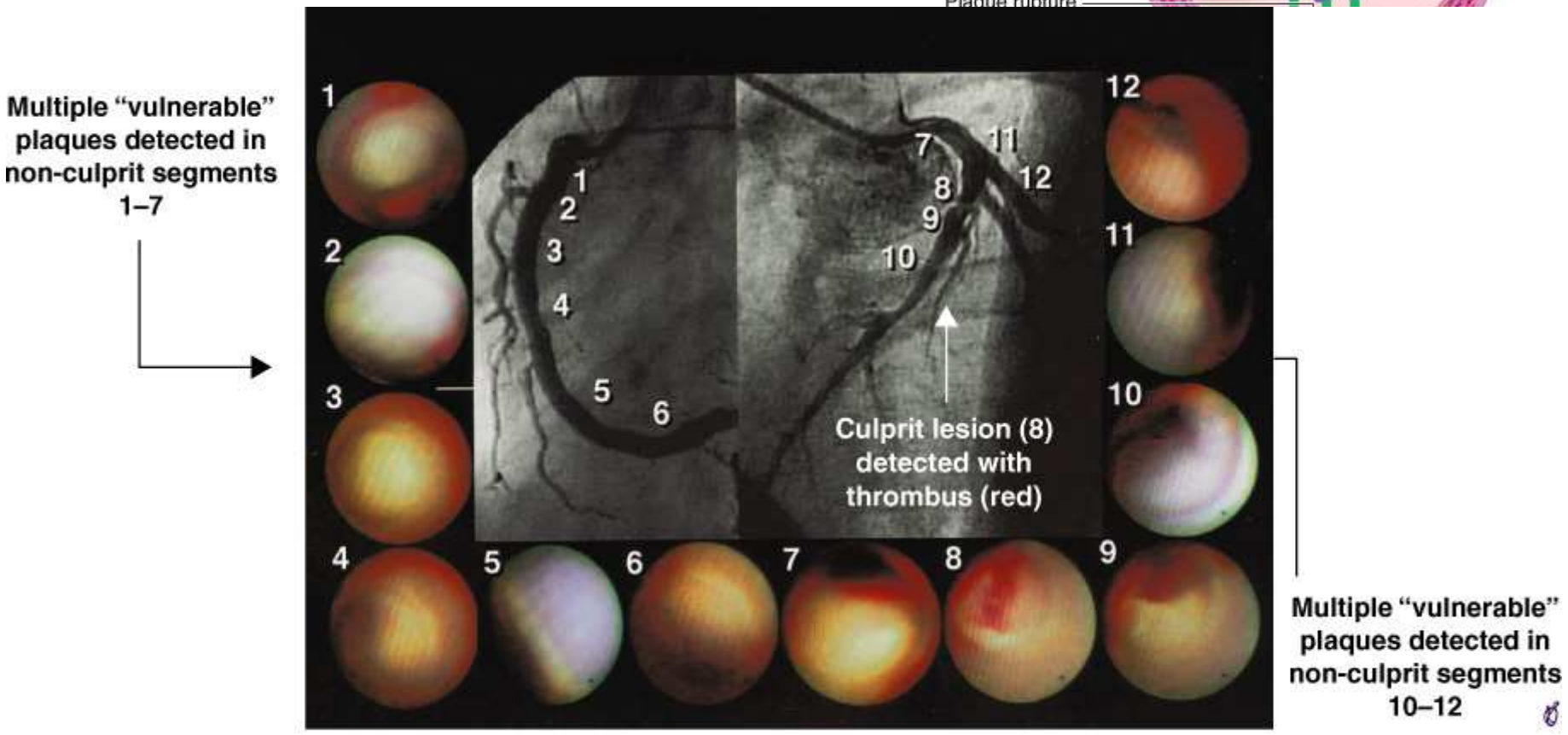
Nonocclusive thrombus on pre-existing plaque
Dynamic obstruction (coronary spasm or vasoconstriction)
Progressive mechanical obstruction
Inflammation and/or infection
Secondary UA

*These causes are not mutually exclusive; some patients have greater than or equal to 2 causes.

Reprinted with permission from Braunwald E. Unstable angina: an etiologic approach to management. Circulation 1998;98:2219–22.

Pathophysiology of UA/NSTEMI





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Symptoms related to ACS

Table 5. Likelihood That Signs and Symptoms Represent an ACS Secondary to CAD

Feature	High Likelihood *Any of the following:*	Intermediate Likelihood *Absence of high-likelihood features and presence of any of the following:*	Low Likelihood *Absence of high- or intermediate-likelihood features but may have:*
History	Chest or left arm pain or discomfort as chief symptom reproducing prior documented angina Known history of CAD, including MI	Chest or left arm pain or discomfort as chief symptom Age >70 years Male sex Diabetes mellitus	Probable ischemic symptoms in absence of any of the intermediate likelihood characteristics Recent cocaine use
Examination	Transient MR, hypotension, diaphoresis, pulmonary edema, or rales	Extracardiac vascular disease	Chest discomfort reproduced by palpation
ECG	New, or presumably new, transient ST-segment deviation (≥0.05 mV) or T-wave inversion (≥0.2 mV) with symptoms	Fixed Q waves Abnormal ST segments or T waves not documented to be new	T-wave flattening or inversion in leads with dominant R waves Normal ECG
Cardiac markers	Elevated cardiac TnI, TnT, or CK-MB	Normal	Normal

Braunwald E, Mark DB, Jones RH, et al. Unstable angina: diagnosis and management. Rockville, MD: Agency for Health Care Policy and Research and the National Heart, Lung, and Blood Institute, US Public Health Service, US Department of Health and Human Services; 1994; AHCPR Publication No. 94-0602.

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Physical Exam

Table 5. Brief Physical Examination in the Emergency Department

1. Airway, Breathing, Circulation (ABC)
2. Vital signs, general observation
3. Presence or absence of jugular venous distension
4. Pulmonary auscultation for rales
5. Cardiac auscultation for murmurs and gallops
6. Presence or absence of stroke
7. Presence or absence of pulses
8. Presence or absence of systemic hypoperfusion (cool, clammy, pale, ashen)

Implications of Physical Exam

- General: Restless agitated, anguished facies, clenched fist (Levine's sign)
- Skin: Cool, clammy, pale, ashen
- Low-grade fever: Nonspecific response to myocardial necrosis
- Hypertension, tachycardia: High sympathetic tone (anterior MI)
- Hypotension, bradycardia: High vagal tone (inferior-posterior MI)
- Small-volume pulses: Low cardiac output
- Fast, slow, or irregular pulse: Atrial or ventricular arrhythmias, heart block
- Paradoxical "ectopic" systolic impulse: LV dyskinesis, ventricular aneurysm (anterior MI)
- Soft S1: Decreased LV contractility; first-degree AV block (inferior MI)
- S4 gallop: Decreased LV compliance
- Paradoxically split S2: Severe LV dysfunction, LBBB, S3

Implications of Physical Exam

- Hypotension: Skin – cool, clammy, cyanotic; CNS – altered mental status; kidneys – oliguria (signs of cardiogenic shock)
- Jugular venous distension: with Kussmaul's sign, hypotension,
- RV S4 and S3 gallops, clear lungs (RV infarction)
- Systolic murmur of VSR: VSR (LSB, palpable thrill common). Differentiate from systolic murmur of MR: papillary muscle rupture
- Pericardial friction rub: Pericarditis (accompanies transmural MI) – late post-MI (Dressler's) syndrome
- Signs of cardiac tamponade, EM dissociation: Cardiac rupture
- Absent pulses and murmur of aortic regurgitation: Aortic dissection
- Screening Neurological Examination
- Cognitive disorientation: memory loss, dysarthria, aphasia, hemispatial neglect
- Motor: facial asymmetry, pronator drift, reflex symmetry, limb dysmetria

Differential Diagnosis of STEMI

Life-threatening

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

Other noncardiac

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

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

Labs

Table 9. Laboratory Evaluations for Management of ST-Elevation Myocardial Infarction

Serum biomarkers for cardiac damage (do not wait for results before implementing reperfusion strategy)
CBC with platelet count
INR
aPTT
Electrolytes and magnesium
BUN
Creatinine
Glucose
Serum lipids

CBC = complete blood count; INR = international normalized ratio; aPTT = activated partial thomboplastin time; BUN = blood urea nitrogen.

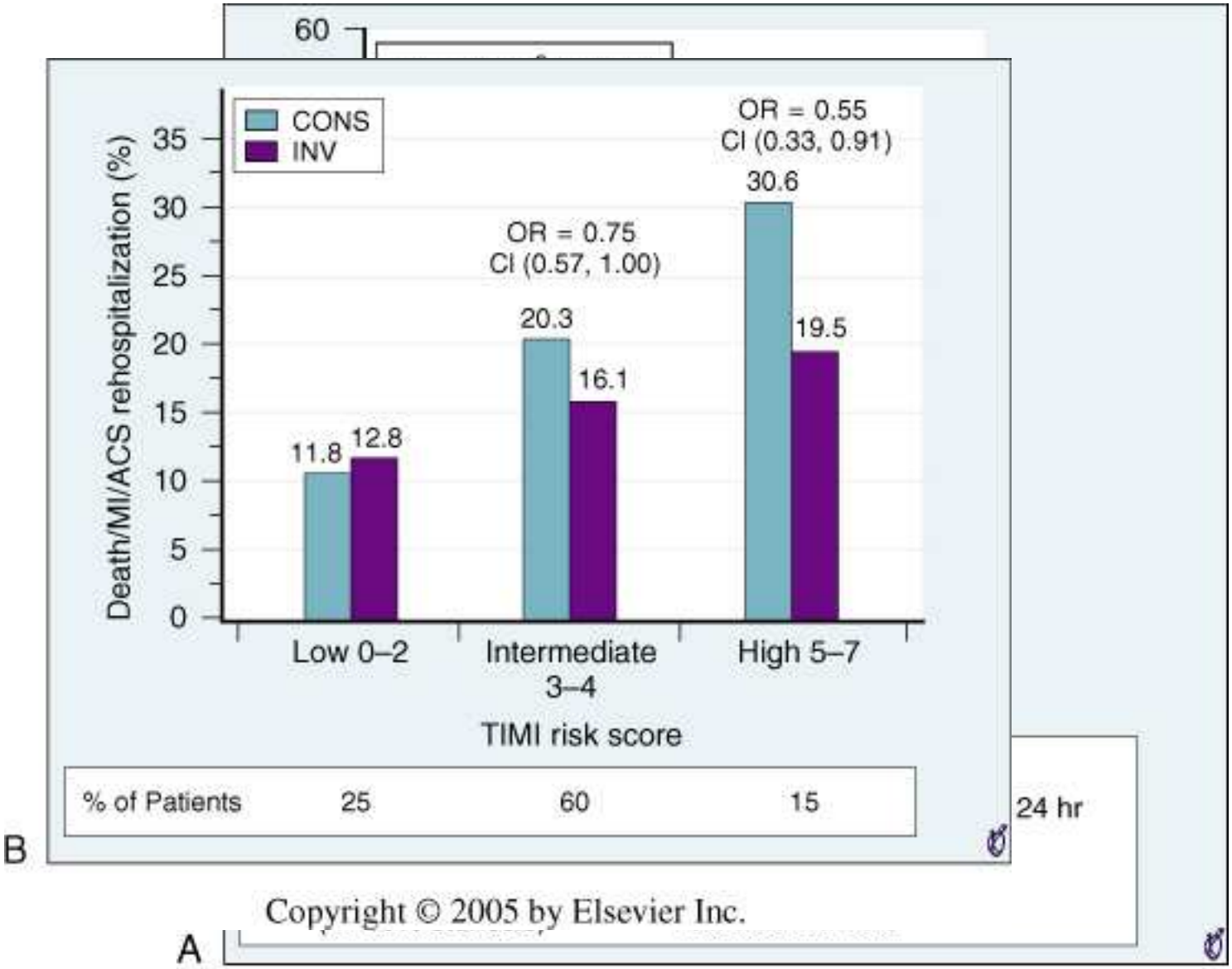
Biomarkers

Table 10. Molecular Biomarkers for the Evaluation of Patients With ST-Elevation Myocardial Infarction

Biomarker	Molecular Weight, Da	Range of Times to Initial Elevation, h	Mean Time to Peak Elevations (Nonreperfused)	Time to Return to Normal Range
Frequently used in clinical practice				
CK-MB	86 000	3-12 h	24 h	48-72 h
cTnI	23 500	3-12 h	24 h	5-10 d
cTnT	33 000	3-12 h	12 h–2 d	5-14 d
Infrequently used in clinical practice				
Myoglobin	17 800	1-4 h	6-7 h	24 h
CK-MB tissue isoform	86 000	2-6 h	18 h	Unknown
CK-MM tissue isoform	86 000	1-6 h	12 h	38 h

Da = Daltons; h = hours; CK-MB = MB isoenzyme of creatine kinase; cTnI = cardiac troponin I; cTnT = cardiac troponin T; CK-MM = MM isoenzyme of creatine

TIMI Risk Score for UA/NSTEMI



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Risk Stratification with UA

Table 6. Short-Term Risk of Death or Nonfatal MI in Patients With UA*

Feature	High Risk *At least 1 of the following features must be present:*	Intermediate Risk *No high-risk feature but must have 1 of the following:*	Low Risk *No high- or intermediate-risk feature but may have any of the following features:*
History	Accelerating tempo of ischemic symptoms in preceding 48 h	Prior MI, peripheral or cerebrovascular disease, or CABG, prior aspirin use	
Character of pain	Prolonged ongoing (>20 minutes) rest pain	Prolonged (>20 min) rest angina, now resolved, with moderate or high likelihood of CAD Rest angina (<20 min) or relieved with rest or sublingual NTG	New-onset or progressive CCS Class III or IV angina the past 2 weeks without prolonged (>20 min) rest pain but with moderate or high likelihood of CAD (see Table 5)
Clinical findings	Pulmonary edema, most likely due to ischemia New or worsening MR murmur S_3 or new/worsening rales Hypotension, bradycardia, tachycardia Age >75 years	Age >70 years	
ECG	Angina at rest with transient ST-segment changes >0.05 mV Bundle-branch block, new or presumed new Sustained ventricular tachycardia	T-wave inversions >0.2 mV Pathological Q waves	Normal or unchanged ECG during an episode of chest discomfort
Cardiac markers	Elevated (e.g., TnT or TnI >0.1 ng/mL)	Slightly elevated (e.g., TnT >0.01 but <0.1 ng/mL)	Normal

*Estimation of the short-term risks of death and nonfatal cardiac ischemic events in UA is a complex multivariable problem that cannot be fully specified in a table such as this; therefore, this table is meant to offer general guidance and illustration rather than rigid algorithms.

Adapted from AHCPR Clinical Practice Guideline No. 10, Unstable Angina: Diagnosis and Management, May 1994. Braunwald E, Mark DB, Jones RH, et al. Unstable angina: diagnosis and management. Rockville, MD: Agency for Health Care Policy and Research and the National Heart, Lung, and Blood Institute, US Public Health Service, US Department of Health and Human Services; 1994; AHCPR Publication No. 94-0602.

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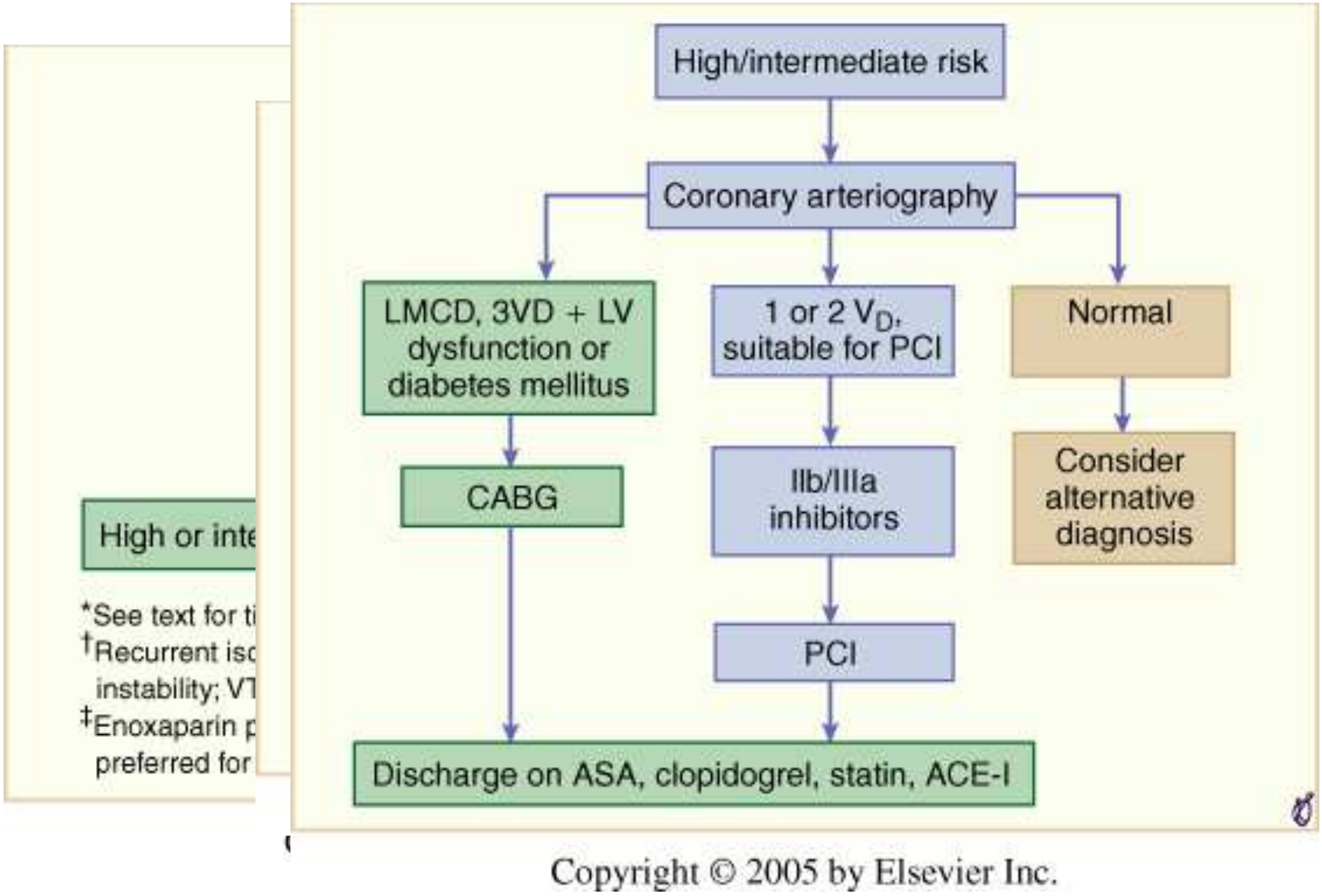
Evaluation of UA/NSTEMI



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Risk Stratification with UA

Class I

- **1. Noninvasive stress testing in low-risk patients (Table 6) who have been free of ischemia at rest or with low level activity and of CHF for a minimum of 12 to 24 h. (Level of Evidence: C)**
- **2. Noninvasive stress testing in patients at intermediate risk (Table 6) who have been free of ischemia at rest or with low-level activity and of CHF for a minimum of 2 or 3 days. (Level of Evidence: C)**

High Risk Noninvasive Results

High risk (>3% annual mortality rate)

1. Severe resting LV dysfunction (LVEF <0.35)
2. High-risk treadmill score (score ≤ –11)
3. Severe exercise LV dysfunction (exercise LVEF <0.35)
4. Stress-induced large perfusion defect (particularly if anterior)
5. Stress-induced multiple perfusion defects of moderate size
6. Large, fixed perfusion defect with LV dilation or increased lung uptake (thallium-201)
7. Stress-induced moderate perfusion defect with LV dilation or increased lung uptake (thallium-201)
8. Echocardiographic wall motion abnormality (involving >2 segments) developing at a low dose of dobutamine (≤ 10 mg · kg–1 · min–1) or at a low heart rate (<120 bpm)
9. Stress echocardiographic evidence of extensive ischemia

Intermediate and Low risk results

Intermediate risk (1–3% annual mortality rate)

1. Mild/moderate resting LV dysfunction (LVEF 0.35–0.49)
2. Intermediate-risk treadmill score (–11 < score <5)
3. Stress-induced moderate perfusion defect without LV dilation or increased lung intake (thallium-201)
4. Limited stress echocardiographic ischemia with a wall motion abnormality only at higher doses of dobutamine involving ≤2 segments

Low risk (<1% annual mortality rate)

1. Low-risk treadmill score (score ≥5)
2. Normal or small myocardial perfusion defect at rest or with stress
3. Normal stress echocardiographic wall motion or no change of limited resting wall motion abnormalities during stress

Table 18. Noninvasive Test Results That Predict High Risk for Adverse Outcome (LV Imaging)

Stress radionuclide ventriculography	Stress echocardiography
Exercise EF ≤0.50	Rest EF ≤0.35
Rest EF ≤0.35	Wall motion score index >1
Fall in EF ≥0.10	

Adapted from O'Rourke RA, Chatterjee K, Dodge HT, et al. Guidelines for clinical use of cardiac radionuclide imaging, December 1986: a report of the American College of Cardiology/American Heart Association Task Force on Assessment of Cardiovascular Procedures (Subcommittee on Nuclear Imaging). J Am Coll Cardiol 1986;8:1471–83; and Cheitlin MD, Alpert JS, Armstrong WF, et al. ACC/AHA guidelines for the clinical application of echocardiography. Circulation 1997;95: 1686–744.

Table 19. Noninvasive Test Results That Predict High Risk for Adverse Outcome on Stress Radionuclide Myocardial Perfusion Imaging

- Abnormal myocardial tracer distribution in >1 coronary artery region at rest or with stress or a large anterior defect that reperfuses
- Abnormal myocardial distribution with increased lung uptake
- Cardiac enlargement

Adapted from O'Rourke RA, Chatterjee K, Dodge HT, et al. Guidelines for clinical use of cardiac radionuclide imaging, December 1986: a report of the American College of Cardiology/American Heart Association Task Force on Assessment of Cardiovascular Procedures (Subcommittee on Nuclear Imaging). J Am Coll Cardiol 1986;8:1471– 83.

Early invasive therapy for UA/NSTEMI

Class I

1. An early invasive strategy in patients with UA/NSTEMI and any of the following high-risk indicators. (Level of Evidence: A).

a) Recurrent angina/ischemia at rest or with low level activities despite intensive anti-ischemic therapy

b) Elevated TnT or TnI

c) New or presumably new ST-segment depression

d) Recurrent angina/ischemia with CHF symptoms, an S3 gallop, pulmonary edema, worsening rales, or new or worsening MR

e) High-risk findings on noninvasive stress testing

f) Depressed LV systolic function (e.g., EF less than 0.40 on noninvasive study)

g) Hemodynamic instability

h) Sustained ventricular tachycardia

i) PCI within 6 months

j) Prior CABG

2. In the absence of these findings, either an early conservative or an early invasive strategy in hospitalized patients without contraindications for revascularization. (Level of Evidence: B)

Class IIa

An early invasive strategy in patients with repeated presentations for ACS despite therapy and without evidence for ongoing ischemia or high risk. (Level of Evidence: C)

Troponin correlates with mortality

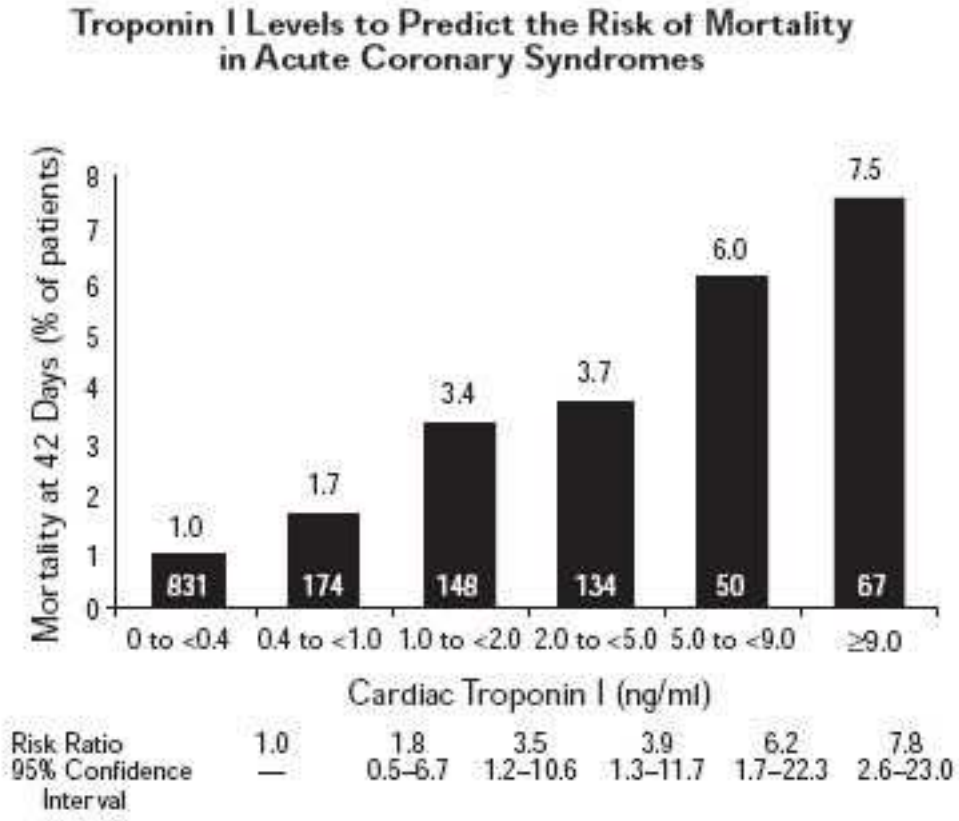

Figure 4. Relationship between cardiac troponin levels and risk of mortality in patients with ACS. Used with permission from Antman EM, Tanasijevic MJ, Thompson B, et al. Cardiac-specific troponin I levels to predict the risk of mortality in patients with acute coronary syndromes. N Engl J Med 1996;335:1342–9.

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Time Course of Biomarkers

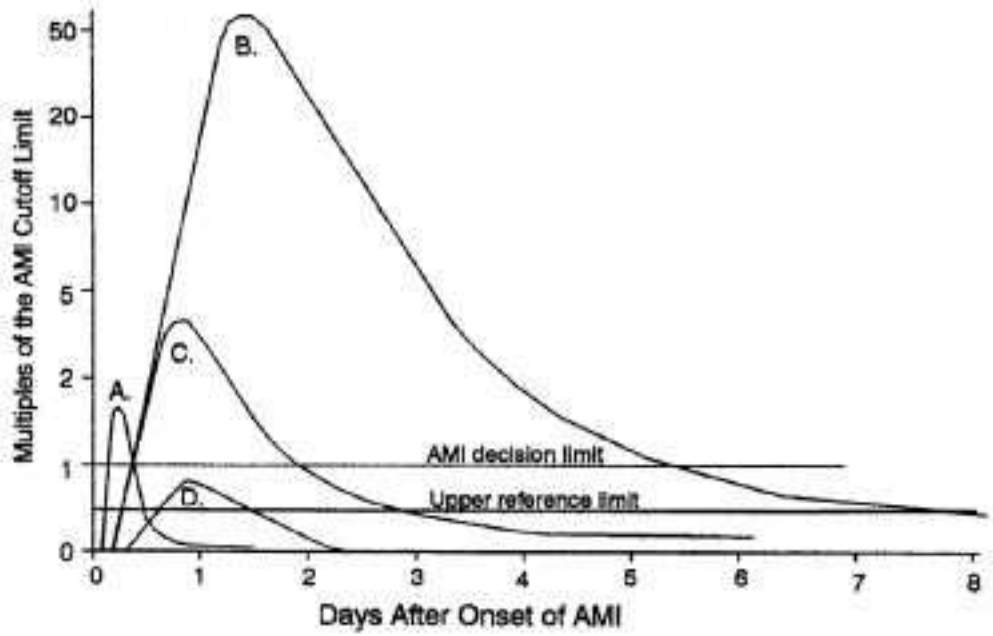
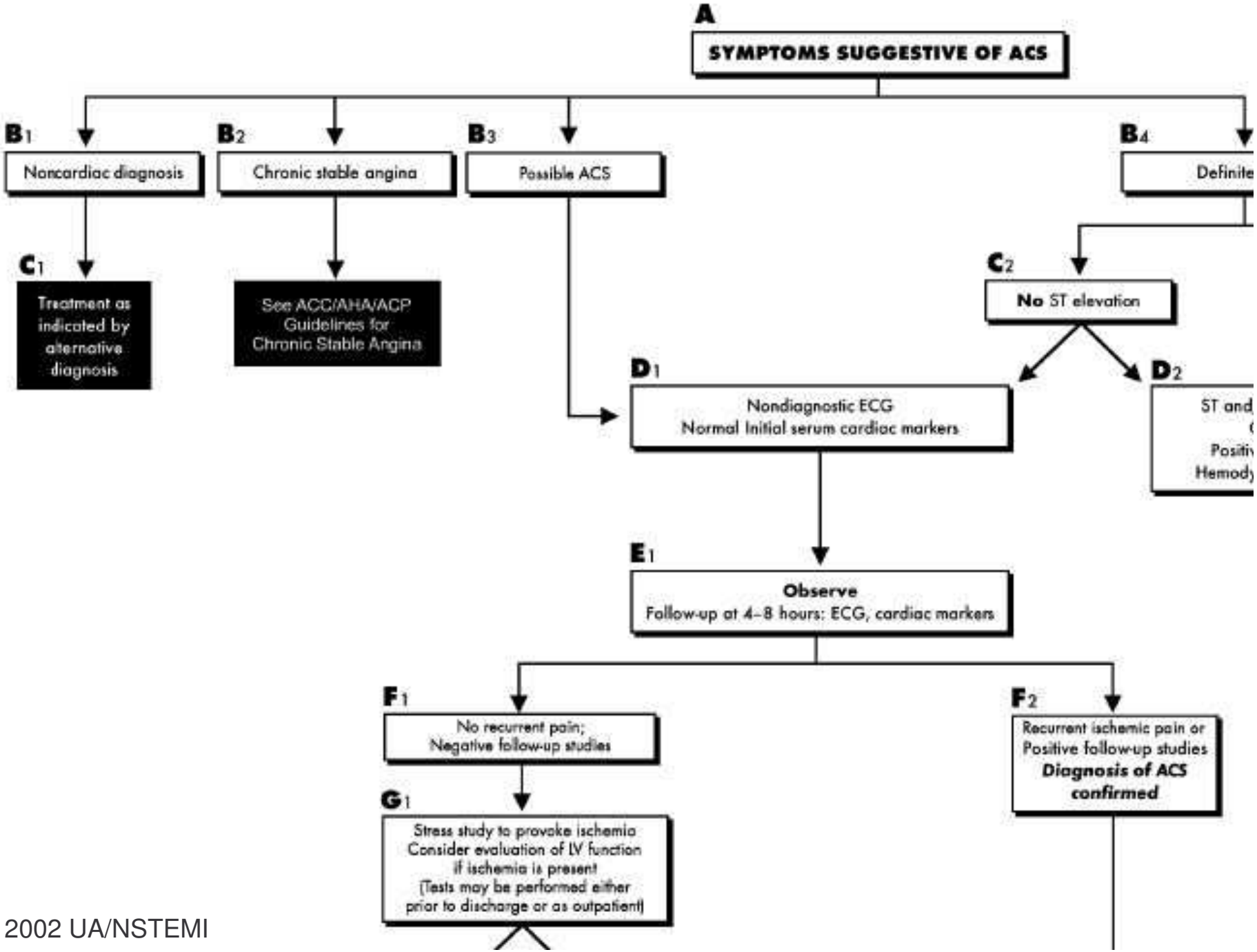


Figure 5. Plot of the appearance of cardiac markers in blood vs. time after onset of symptoms. Peak A, early release of myoglobin or CK-MB isoforms after AMI. Peak B, cardiac troponin after AMI. Peak C, CK-MB after AMI. Peak D, cardiac troponin after UA. Data are plotted on a relative scale, where 1.0 is set at the AMI cutoff concentration. Reprinted with permission from National Academy of Clinical Biochemistry, Washington, DC. Standards of laboratory practice: recommendations for use of cardiac markers in coronary artery disease. November 5, 1999.



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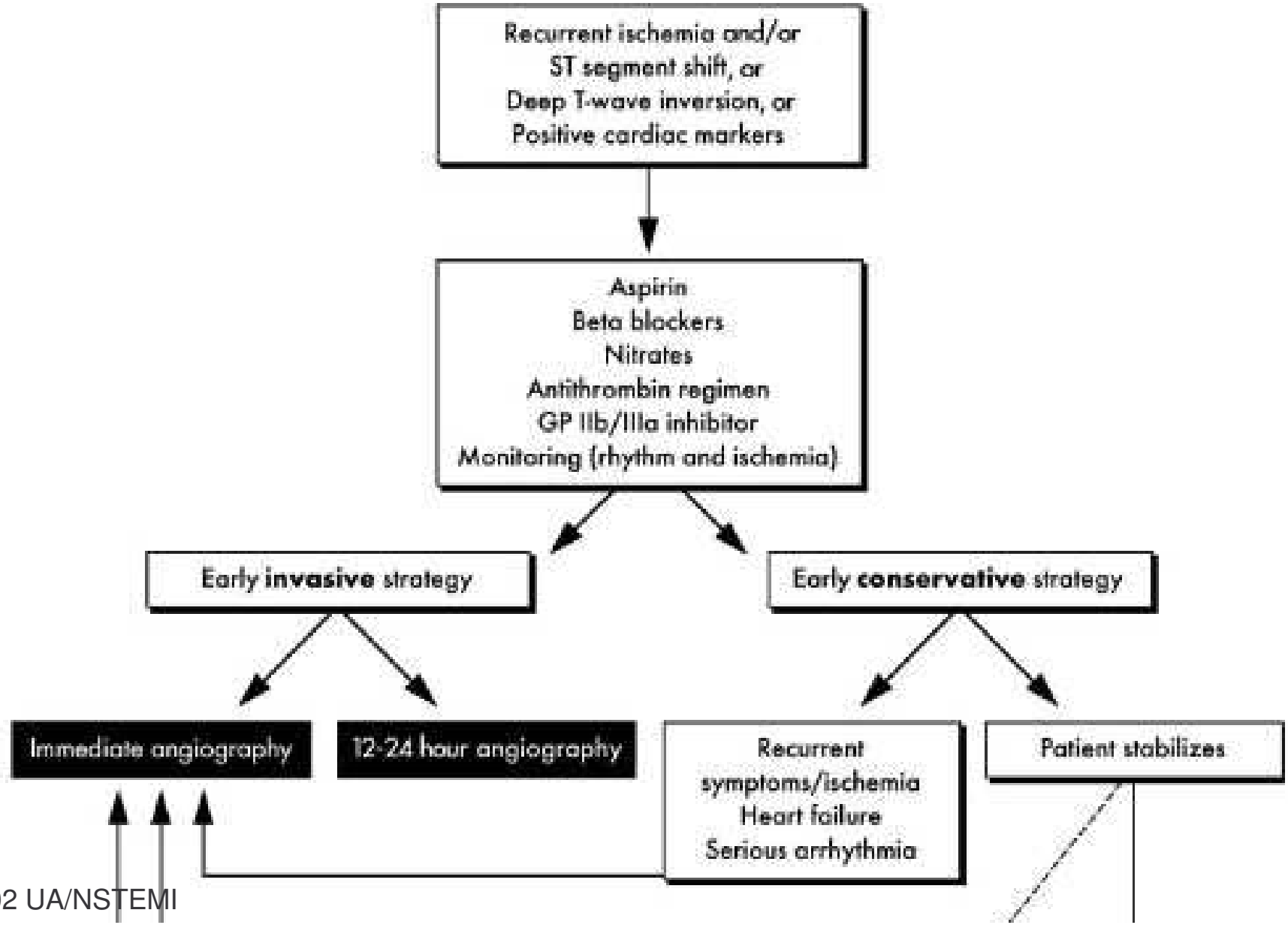
Acute Ischemia Pathway



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Treatment of UA/NSTEMI

Table 10. Class I Recommendations for Anti-Ischemic Therapy in the Presence or Absence of Continuing Ischemia or High-Risk Features*

Continuing Ischemia/Other Clinical High-Risk Features*	
Present	**Absent**
Bed rest with continuous ECG monitoring	
Supplemental O_2 to maintain Sao_2 >90%	
NTG IV	
Beta-blockers, oral or IV	Beta-blockers, oral
Morphine IV for pain, anxiety, pulmonary congestion	
IABP if ischemia or hemodynamic instability persists	
ACEI for control of hypertension or LV dysfunction, after MI	ACEI for control of hypertension or LV dysfunction, after MI

*Recurrent angina and/or ischemia-related ECG changes (greater than or equal to 0.05-mV ST-segment depression or bundle-branch block) at rest or low-level activities; or ischemia associated with CHF symptoms, S_3 gallop, or new or worsening mitral regurgitation; or hemodynamic instability or depressed LV function (EF <0.40 on noninvasive study); or malignant ventricular arrhythmia.

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Antiplatelet therapy for UA/NSTEMI

Table 14. Class I Recommendations for Antithrombotic Therapy*

Possible ACS	Likely/Definite ACS	Definite ACS With Continuing Ischemia or Other High-Risk Features† or Planned Intervention
Aspirin	Aspirin + Subcutaneous LMWH *or* IV heparin	Aspirin + IV heparin + IV platelet GP IIb/IIIa antagonist

*Clinical data on the combination of LMWH and platelet GP IIb/IIIa antagonists are lacking. Their combined use is not currently recommended.
†High-risk features are listed in Table 6; others include diabetes, recent MI, and elevated TnT or TnI.

- Plavix is also a class 1 recommendation for UA/NSTEMI, for both early invasive and conservative therapy

LMWH in UA

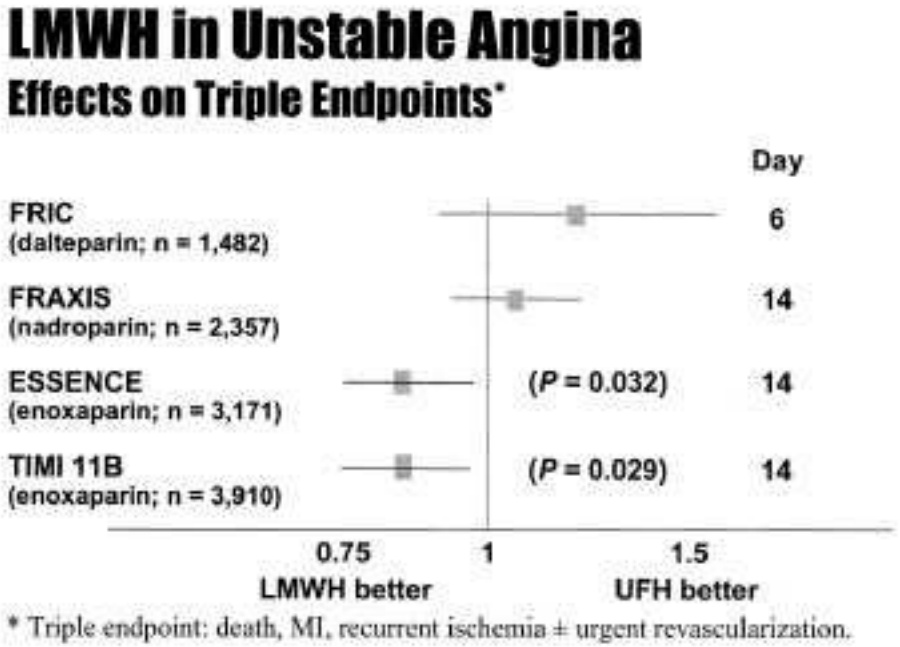

Figure 9. The use of LMWH in UA showing effects on the triple end points of death, MI, and recurrent ischemia with or without revascularization. Early (6-day) and intermediate outcomes of the 4 trials that compared LMWH and UFH: ESSENCE (169), TIMI 11B (170), FRIC (218), and FRAXIS (219). Nadroparine in FRAXIS was given for 14 days.

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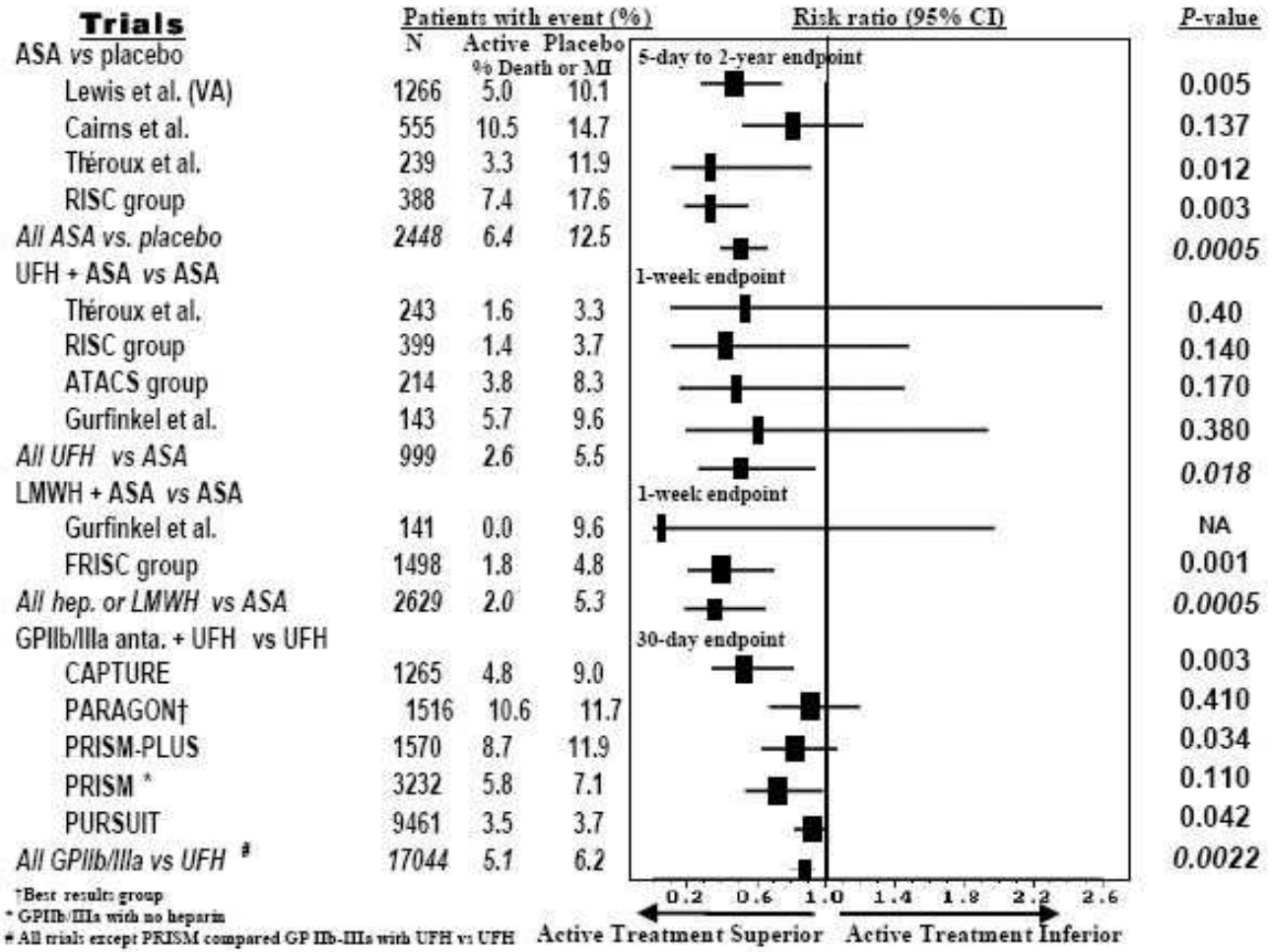

Figure 8. Summary of trials of antithrombotic therapy in UA. Meta-analysis of randomized trials in UA/NSTEMI that have compared ASA with placebo, the combination of UFH and ASA with ASA alone, the combination of an LMWH and ASA with ASA alone, and the combination of a platelet GP IIb/IIIa antagonist (anta.), UFH (hep.), and ASA with UFH plus ASA. The RR values, 95% CIs, and probability value for each trial are shown. The timing of the end point (death or MI) varied. Results with the platelet GP IIb/IIIa antagonists are reported at the 30-day time point. Incremental gain is observed from single therapy with ASA to double therapy with ASA and UFH and to triple antithrombotic therapy with ASA, UFH, and a platelet GP IIb/IIIa antagonist. In the CAPTURE trial, nearly all patients underwent PCI after 20 to 24 h per study design. From PURSUIT (10), PRISM-PLUS (21), Lewis et al. (175), Cairns et al. (176), Théroux et al. (177), RISC group (178), ATACS group (179), Gurfinkel et al. (180), FRISC group (181), CAPTURE (182), PARAGON (183), and PRISM (184).

ACS Outcomes

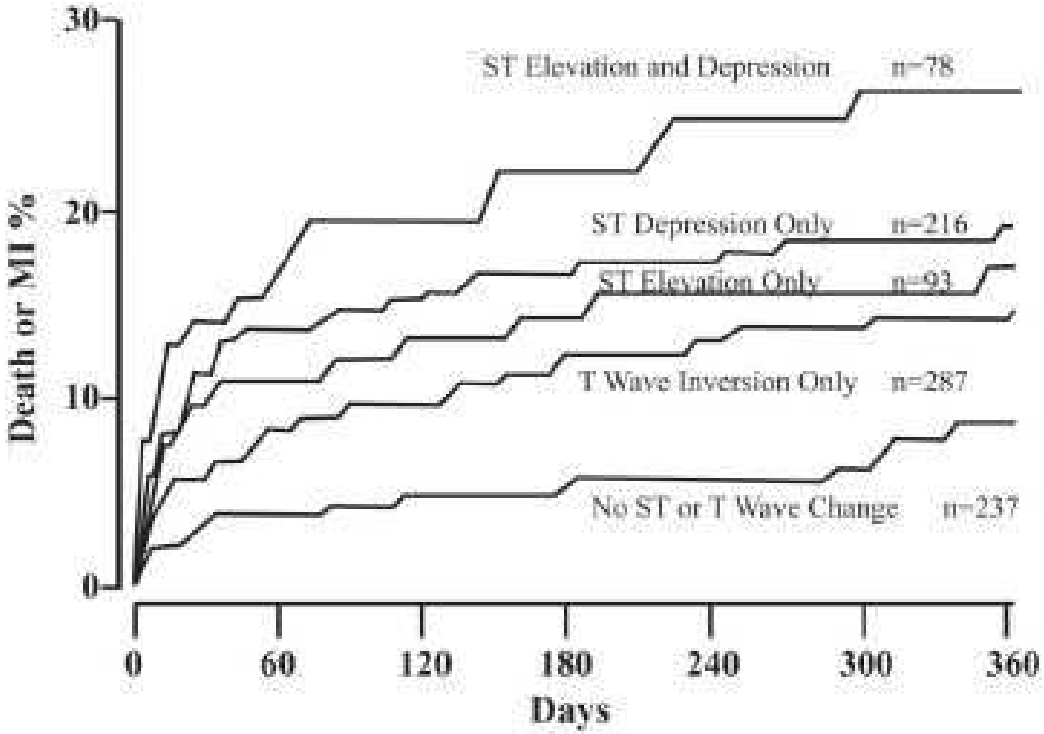

Figure 11. Adverse outcome by initial ECG in ACS. Adapted from Nyman I, Areskog M, Areskog NH, Swahn E, Wallentin L. Very early risk stratification by electrocardiogram at rest in men with suspected unstable coronary heart disease. The RISC Study Group. J Intern Med 1993;234:293–301.

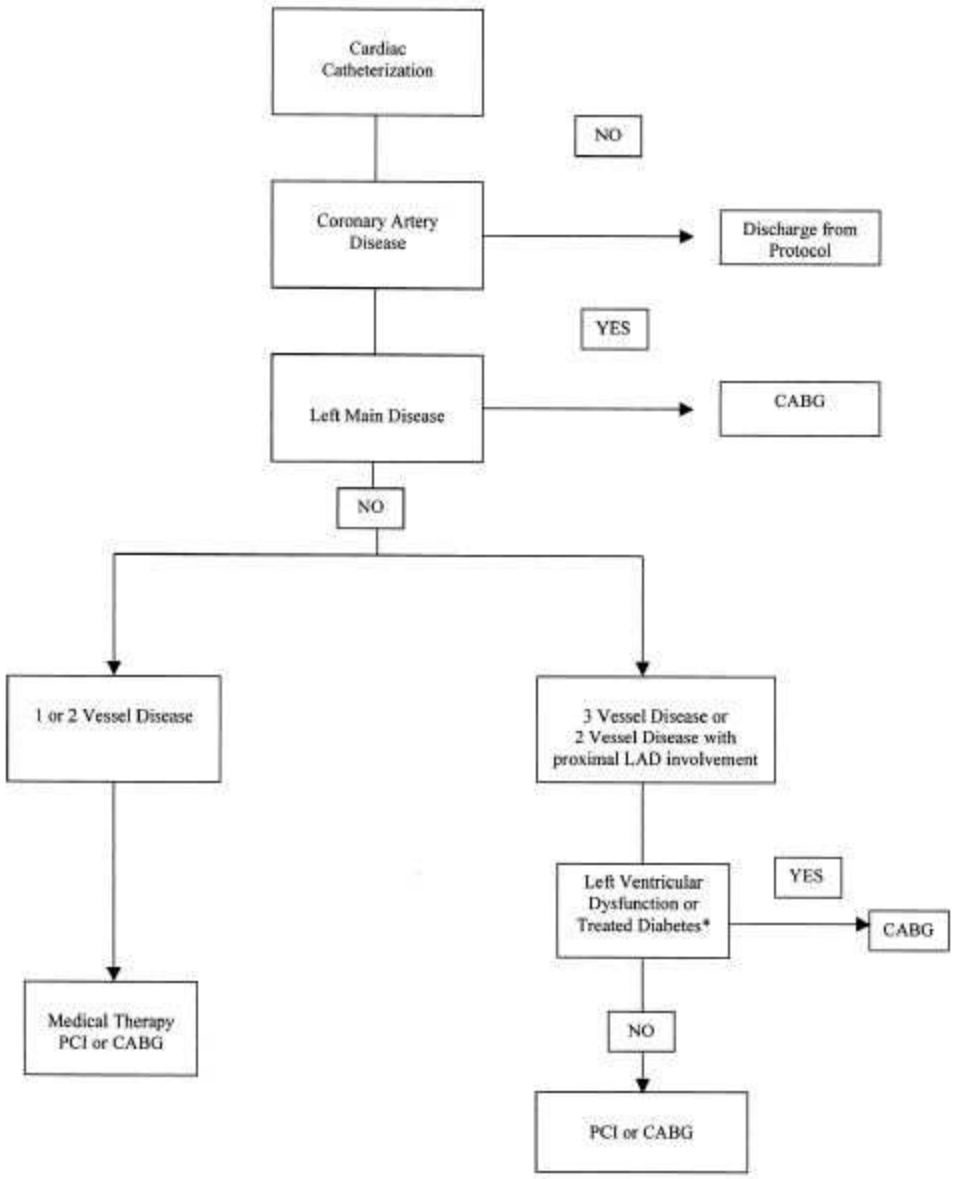

Figure 12. Revascularization strategy in UA/NSTEMI. *There is conflicting information about these patients. Most consider CABG to be preferable to PCI.

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Choice of Revascularization

Table 20. Mode of Coronary Revascularization for UA/NSTEMI

Extent of Disease	Treatment	Class/Level of Evidence
Left main disease,* candidate for CABG	CABG	I/A
	PCI	III/C
Left main disease, not candidate for CABG	PCI	IIb/C
Three-vessel disease with EF <0.50	CABG	I/A
Multivessel disease including proximal LAD with EF <0.50 or treated diabetes	CABG or PCI	I/A IIb/B
Multivessel disease with EF >0.50 and without diabetes	PCI	I/A
One- or 2-vessel disease without proximal LAD but with large areas of myocardial ischemia or high-risk criteria on noninvasive testing (see Table 17)	CABG or PCI	I/B
One-vessel disease with proximal LAD	CABG or PCI	IIa/B†
One- or 2-vessel disease without proximal LAD with small area of ischemia or no ischemia on noninvasive testing	CABG or PCI	III/C†
Insignificant coronary stenosis	CABG or PCI	III/C

*≥50% diameter stenosis.
†Class/level of evidence I/A if severe angina persists despite medical therapy.

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Medications following UA/NSTEMI

Table 21. Medications Used for Stabilized UA/NSTEMI

Anti-Ischemic and Antithrombotic/ Antiplatelet Agent	Drug Action	Class/Level of Evidence
Aspirin	Antiplatelet	I/A
Clopidogrel* or ticlopidine	Antiplatelet when aspirin is contraindicated	I/A
Beta-blockers	Anti-ischemic	I/A
ACEI	EF less than 0.40 or CHF EF greater than 0.40	I/A IIa/A
Nitrates	Antianginal	I/C For ischemic symptoms
Calcium antagonists (short-acting dihydropyridine antagonists should be avoided)	Antianginal	I For ischemic symptoms When beta-blockers are not successful (level of evidence: B) or contraindicated Or cause unacceptable side effects (level of evidence: C)
Warfarin low intensity with or without aspirin	Antithrombotic	IIb/B
Dipyridamole	Antiplatelet	III/A

Medications following UA/NSTEMI

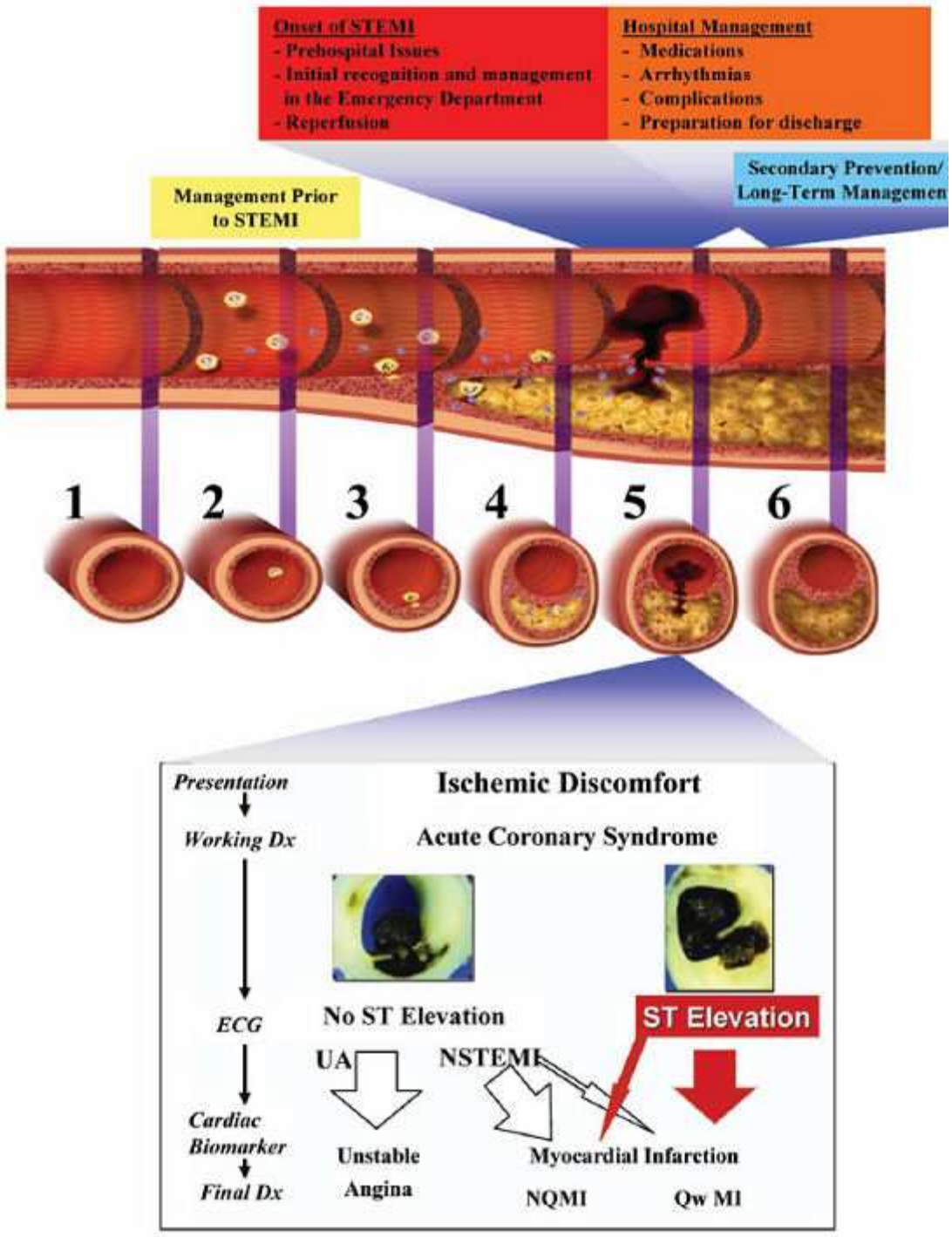
Agent	Risk Factor	Class/Level of Evidence
HMG-CoA reductase inhibitors	LDL cholesterol greater than 130 mg/dL	I/A
HMG-CoA reductase inhibitors	LDL cholesterol 100–130 mg/dL	IIa/B
Gemfibrozil	HDL cholesterol less than 40 mg/dL	IIa/B
Niacin	HDL cholesterol less than 40 mg/dL	IIa/B
Niacin or gemfibrozil	Triglycerides greater than 200 mg/dL	IIa/B
Folate	Elevated homocysteine	IIb/C
Antidepressant	Treatment of depression	IIb/C
Treatment of hypertension	Blood pressure greater than 135/85 mm Hg	I/A
HRT (initiation)†	Postmenopausal state	III/B
HRT (continuation)†	Postmenopausal state	IIa/C

ACEI indicates angiotensin-converting enzyme inhibitor; CHF, congestive heart failure; EF, ejection fraction; HDL, high-density lipoprotein; HRT, hormone replacement therapy; LDL, low-density lipoprotein; NSTEMI, non–ST-segment elevation myocardial infarction; UA, unstable angina.
*Preferred to ticlopidine.
†For risk reduction of coronary artery disease.

Antiplatelet agents following stenting

	Bare Metal Stent	Cypher (Sirolimus)	Taxus (Tacrolimus)
Aspirin 325	1 month	3 months	6 months
Plavix 75	1 month	3 months	6 months

Aspirin 81mg daily is recommended indefinitely following stenting
Plavix use is variable, with some practitioners continuing plavix for 1 year following drug eluting stent



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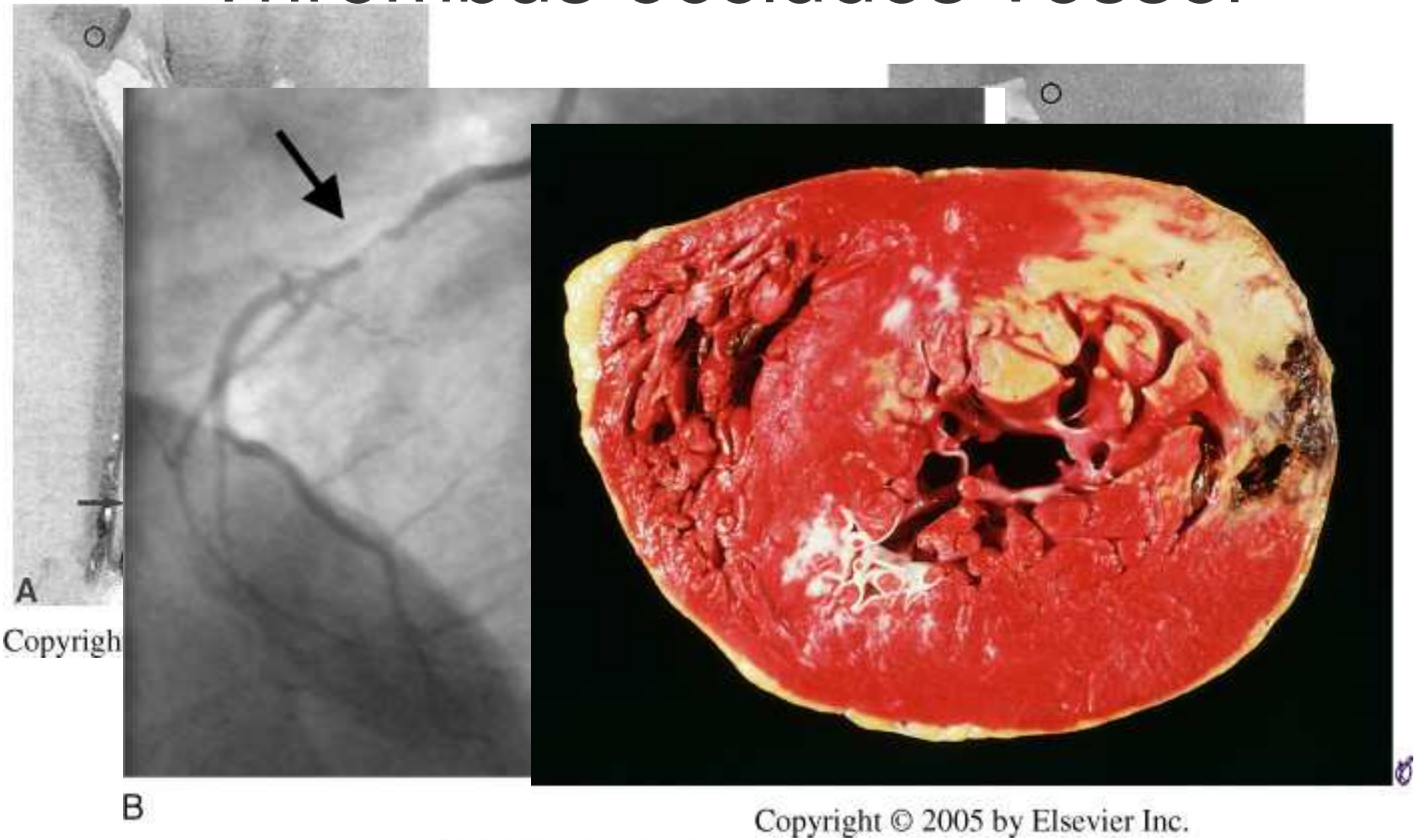
Thrombus occludes vessel



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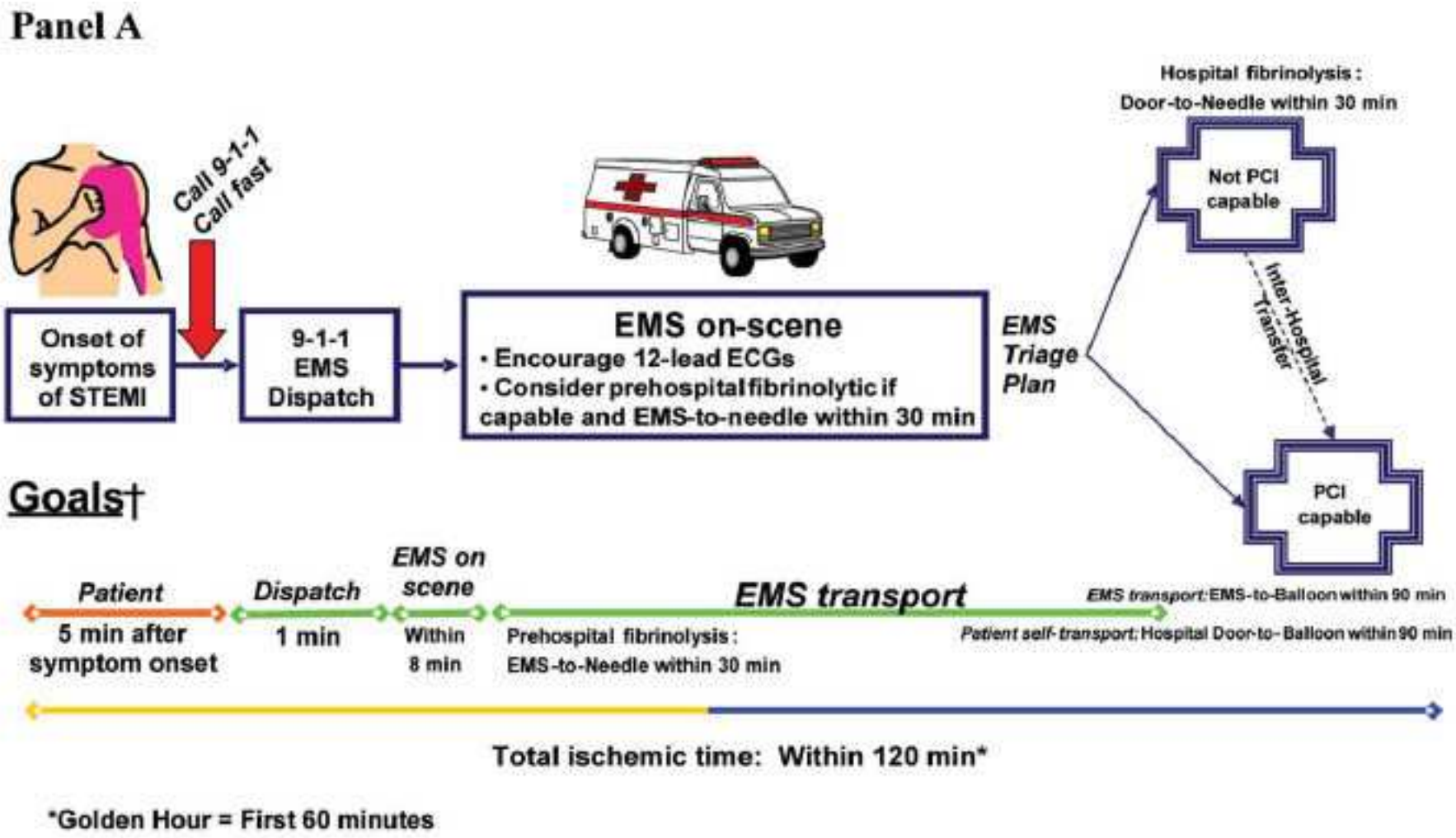
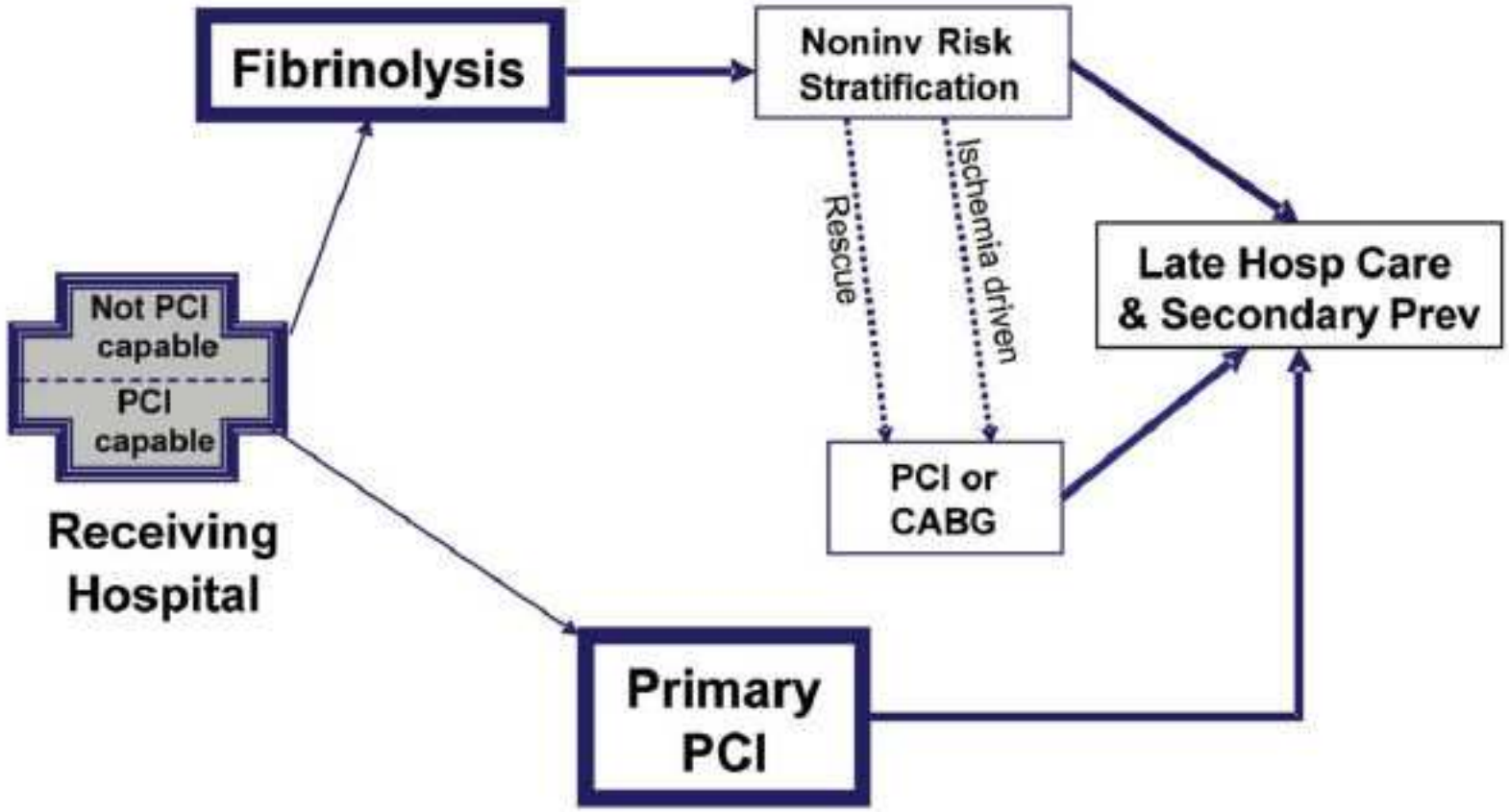
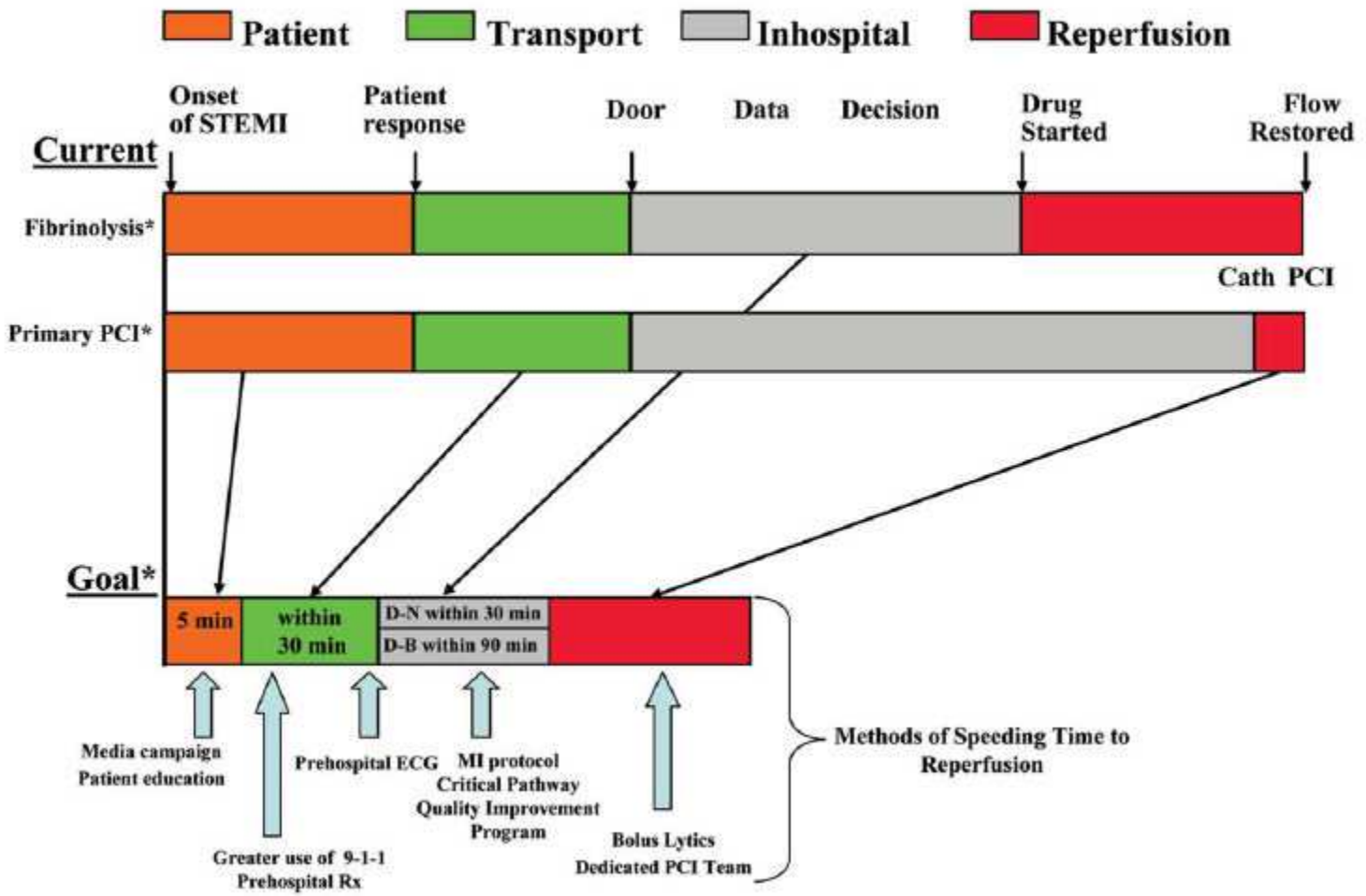
Thrombus Formation

- Angiographic evidence of coronary thrombus formation may be seen in more than 90% of patients with STEMI but in only 1% of patients with stable angina and about 35% to 75% of patients with unstable angina or NSTEMI







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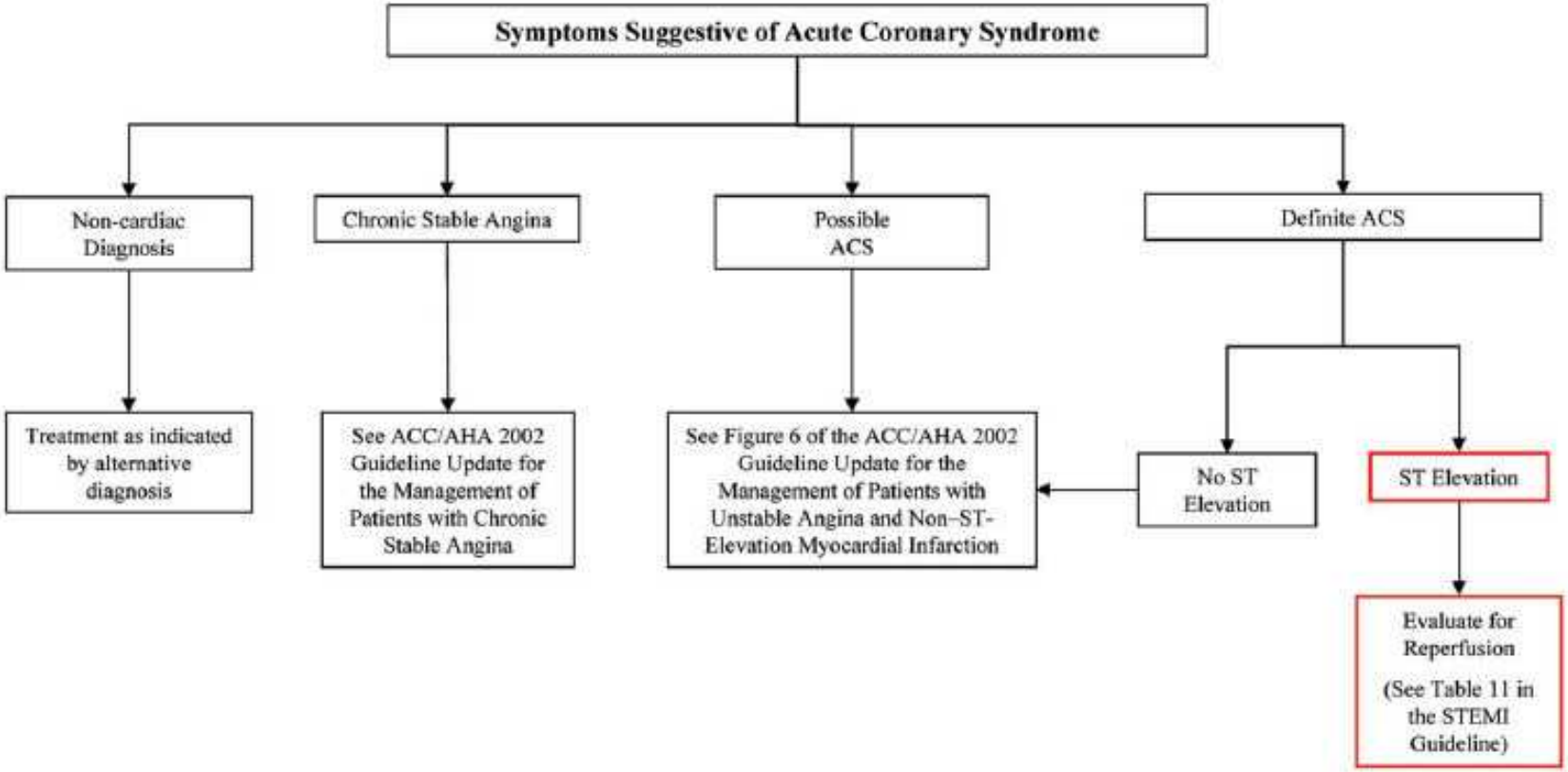
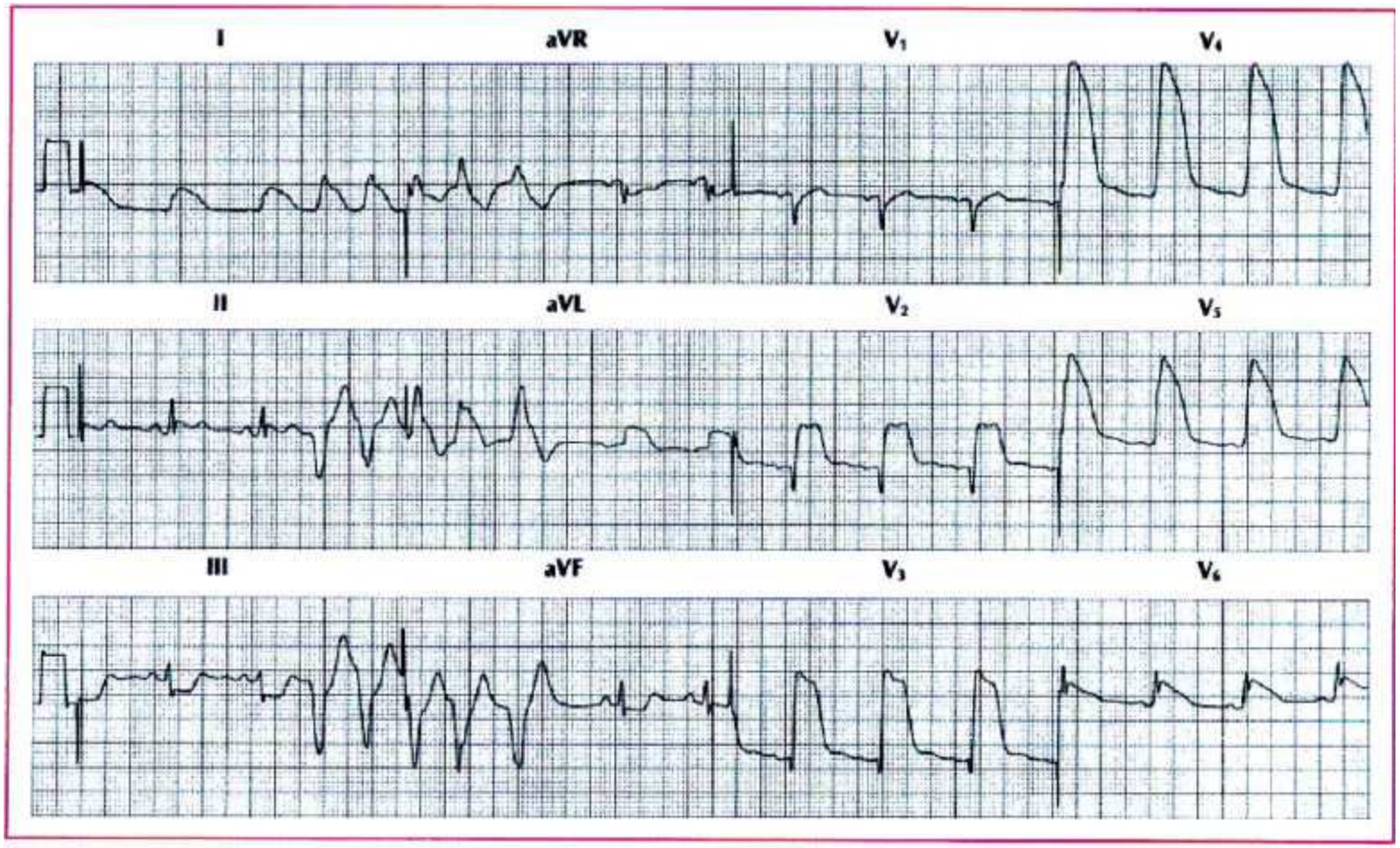


Figure 9. Algorithm for evaluation and management of patients suspected of having acute coronary syndrome. STEMI = ST-elevation myocardial infarction. Modified from Braunwald et al. J Am Coll Cardiol 2000;36:970-1062 (4).

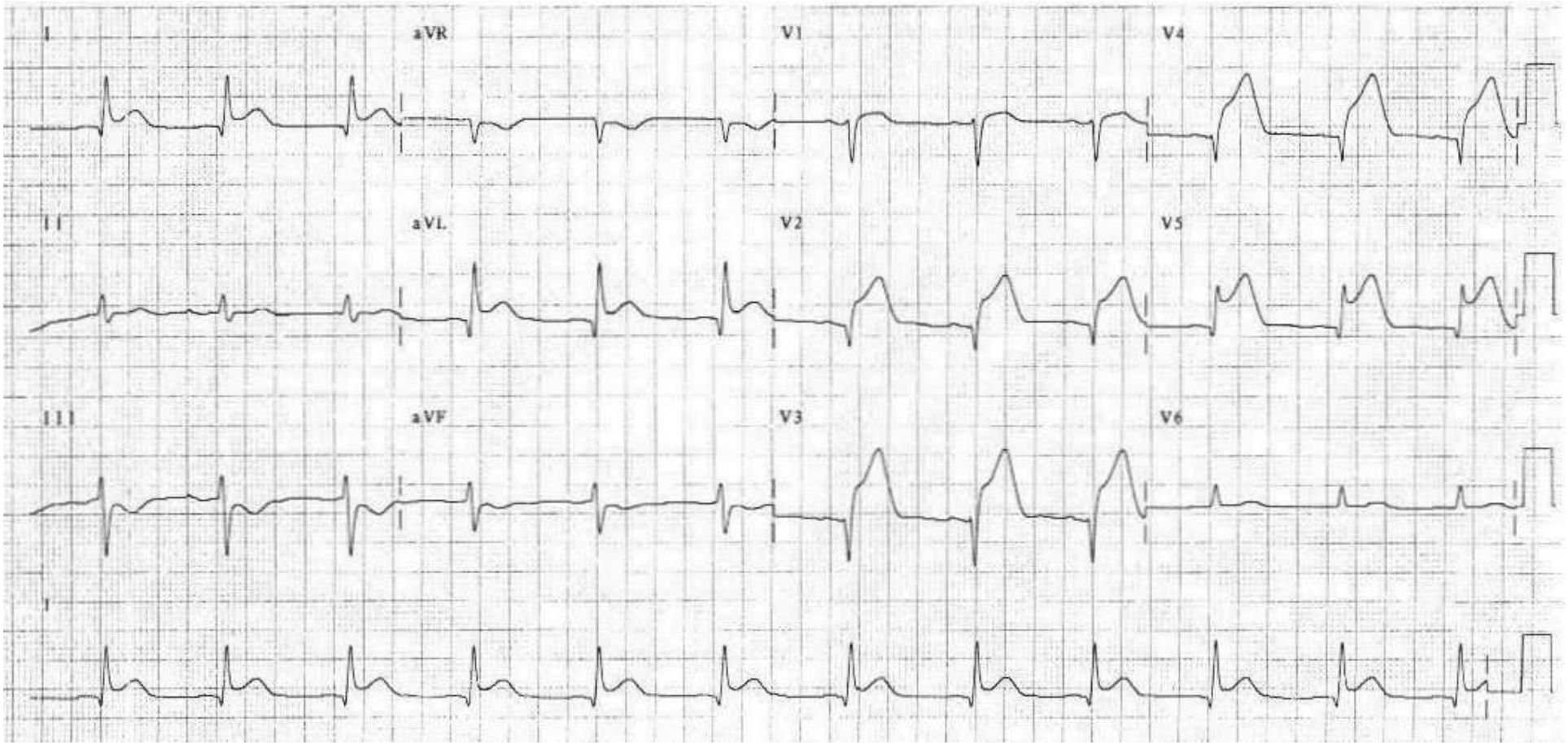
EKG Criteria for STEMI

- New (or presumed new) LBBB
- 0.1 mm ST elevation
  - 0.2mm V1-V4 may reduce false positive anteroseptal infarction
- 0.1 mm ST depression with tall R waves and upright T waves in V1-V4 (Posterior Infarct, V7-V8 may show ST elevation)

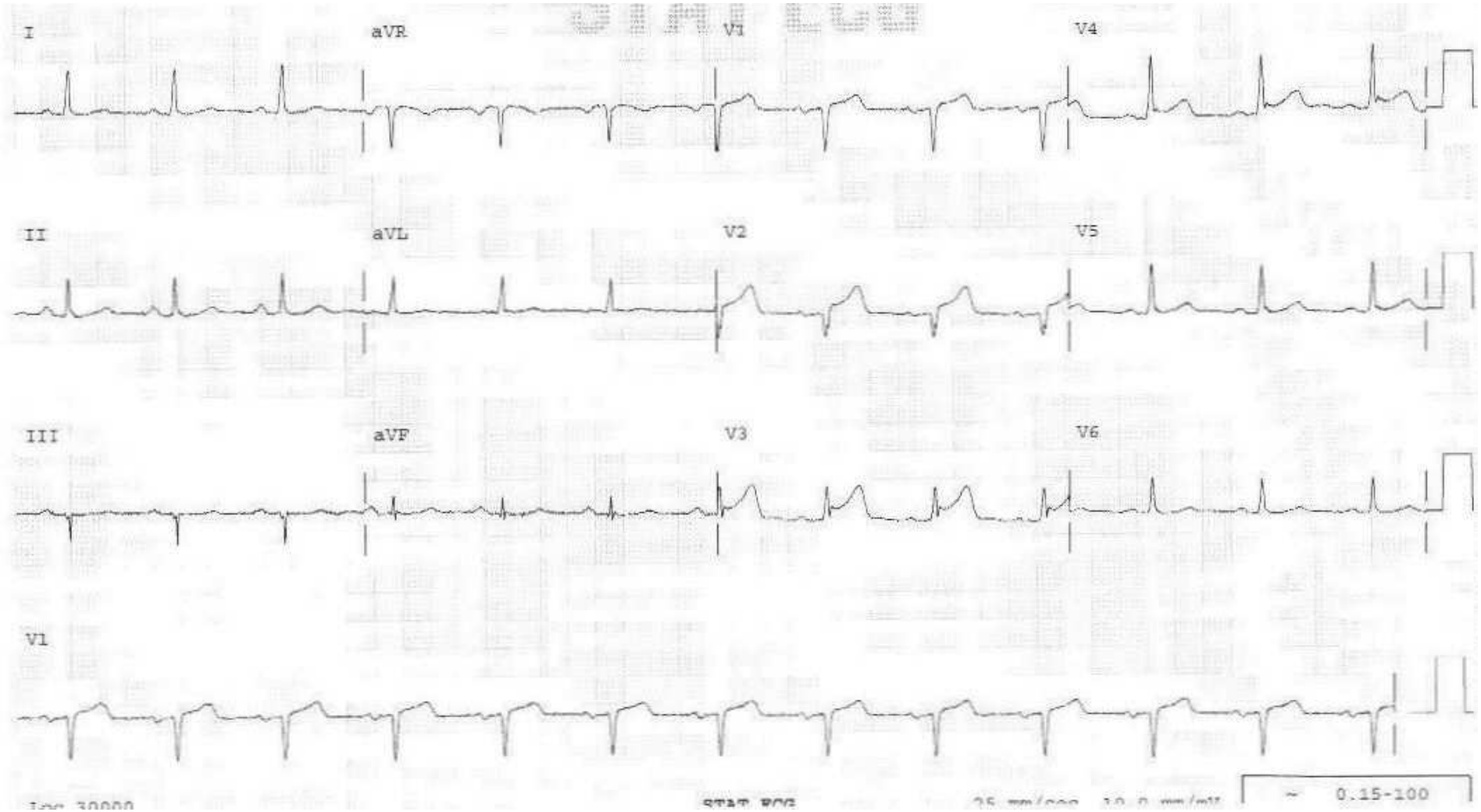
STEMI on EKG



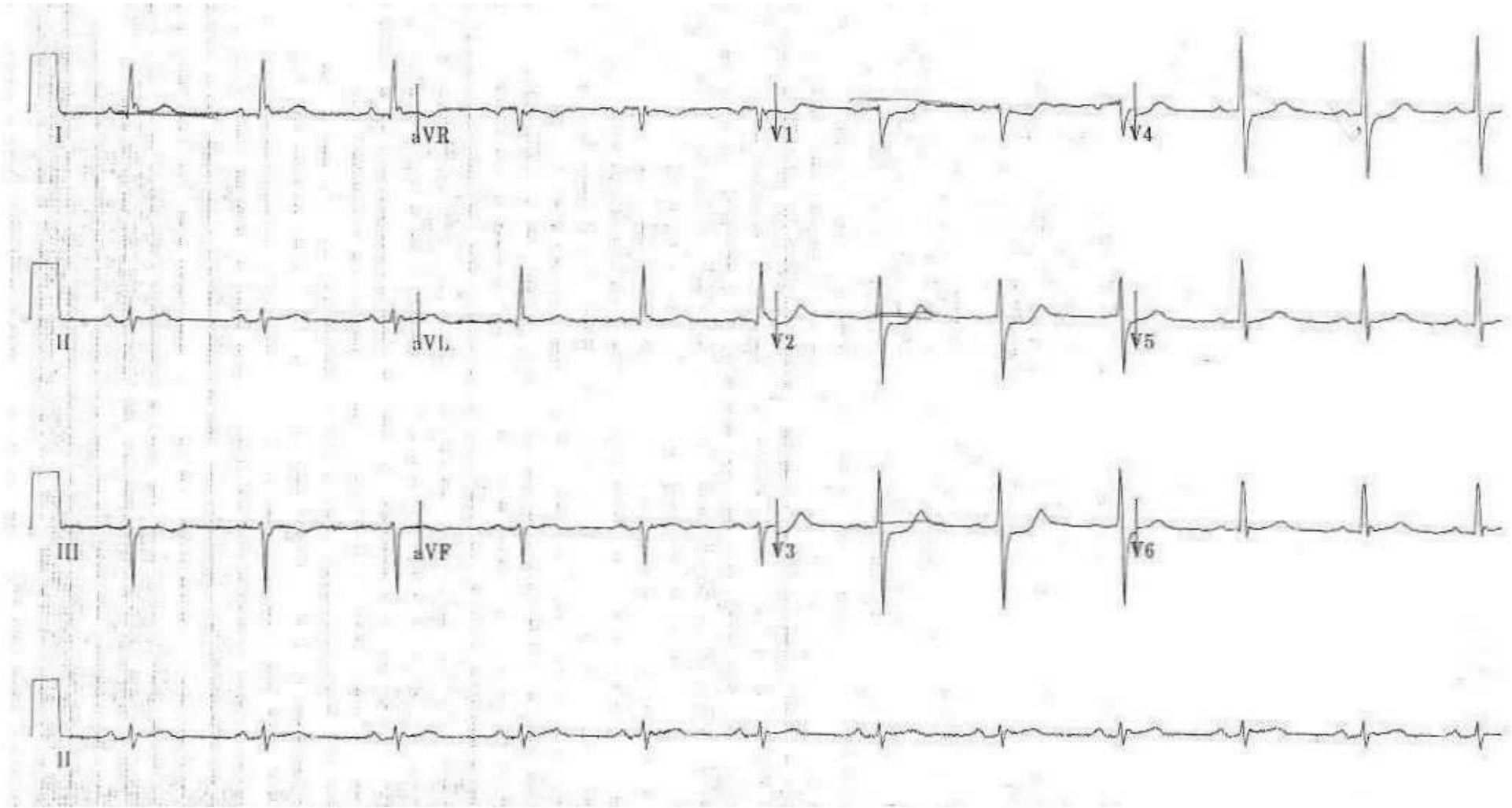
ACC 2004 STEMI



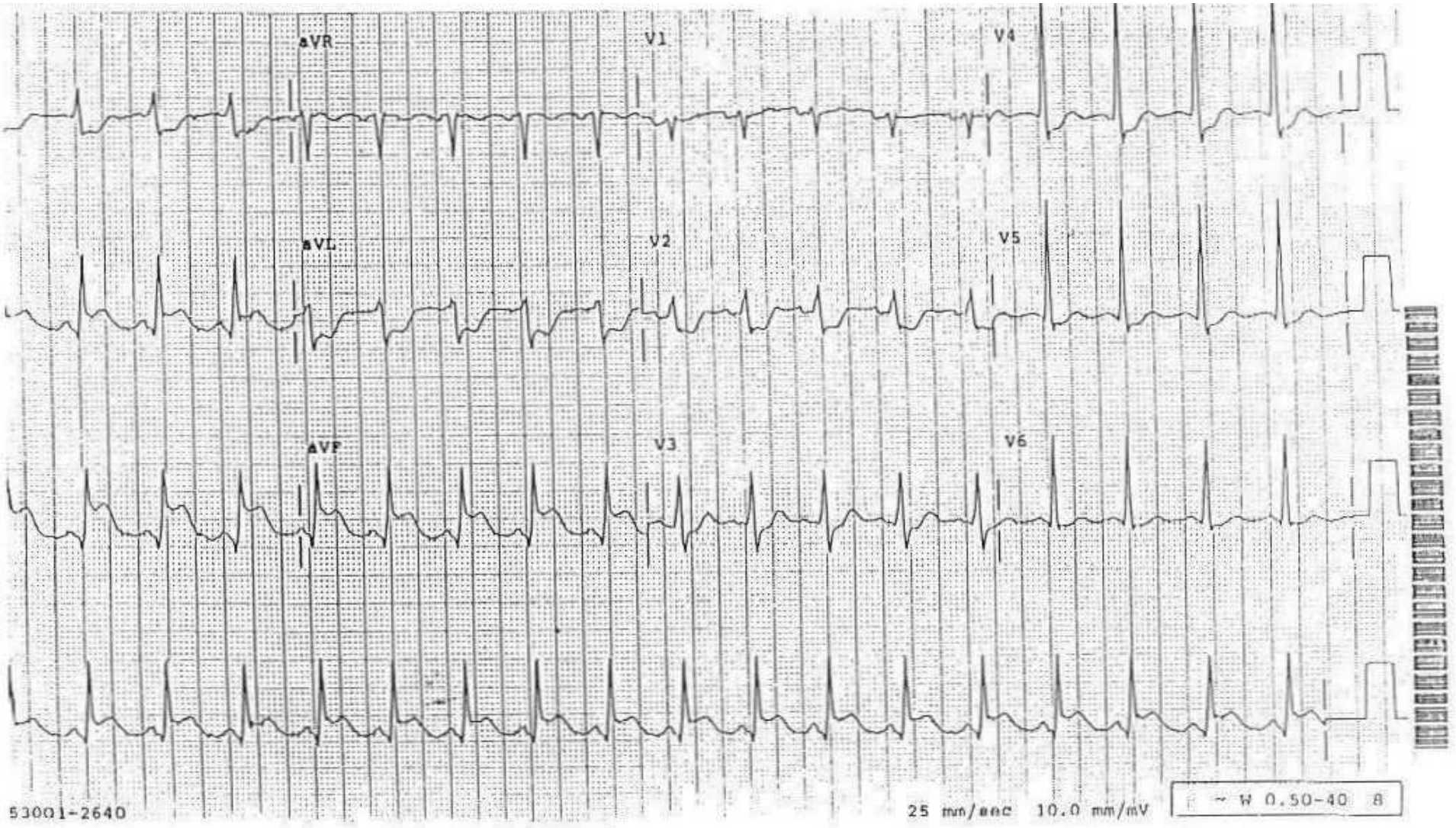
Anterior Injury, 100% LAD occlusion



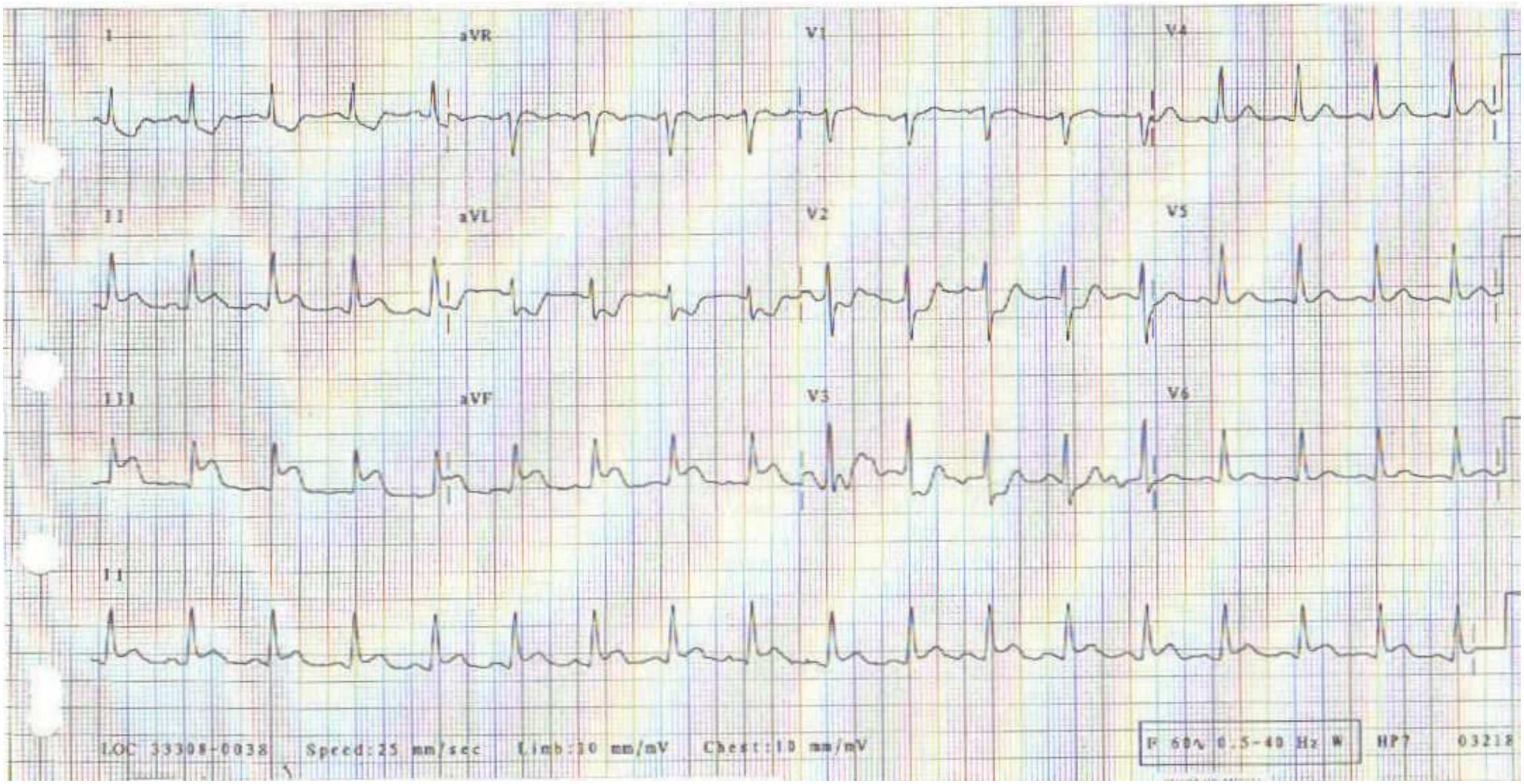
Anteroseptal Injury, 100% mid LAD occlusion



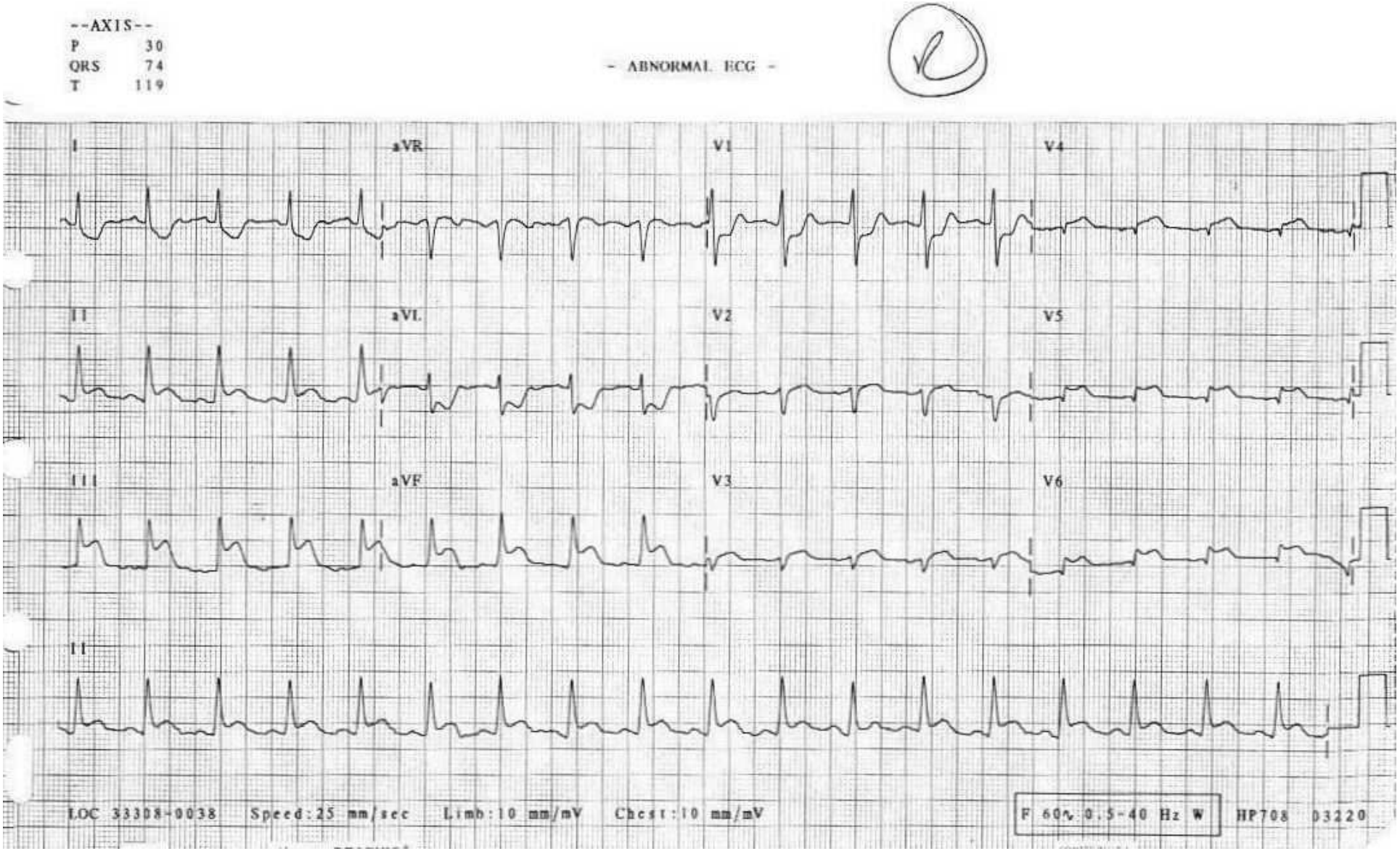
Posterior injury



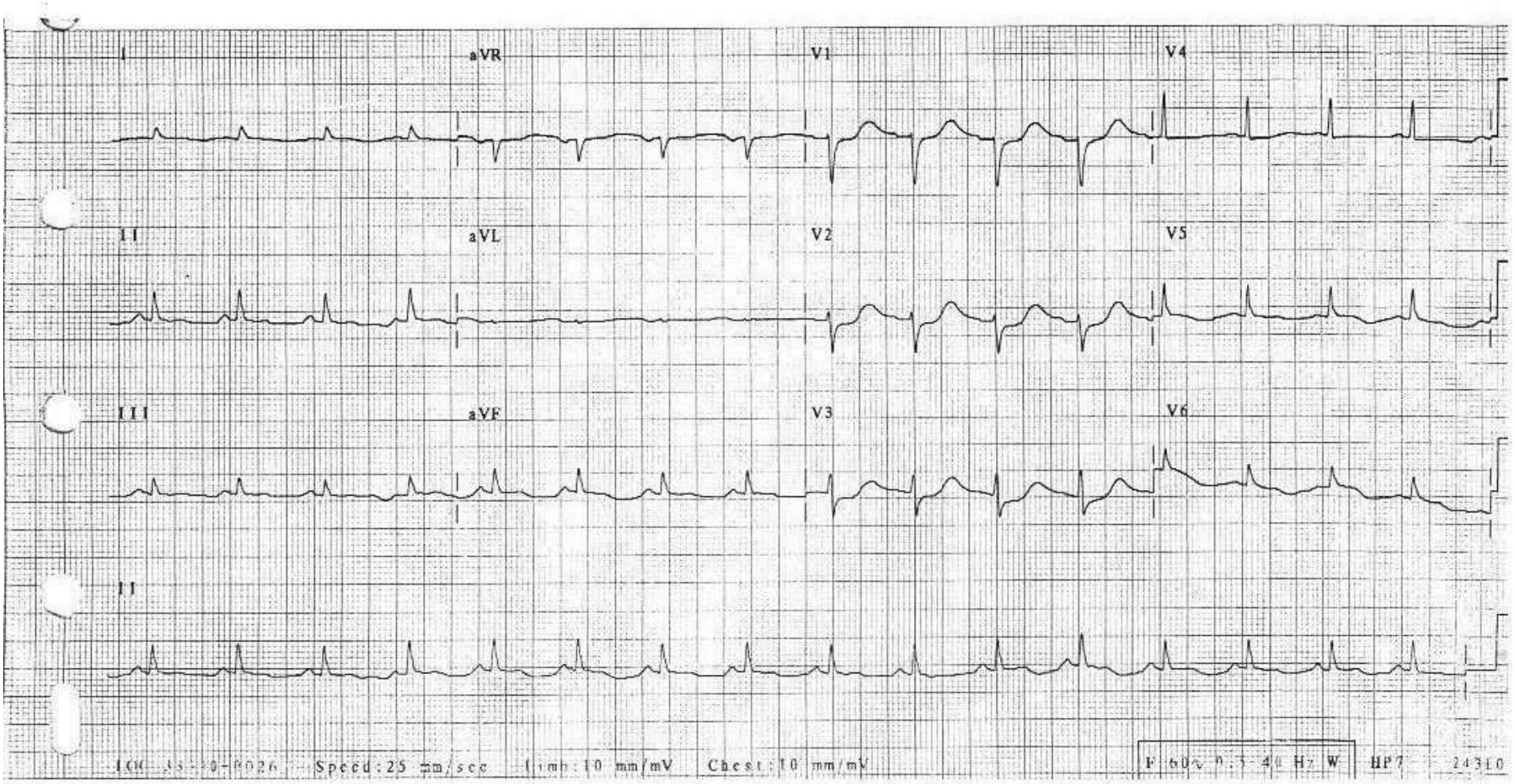
Inferior Injury



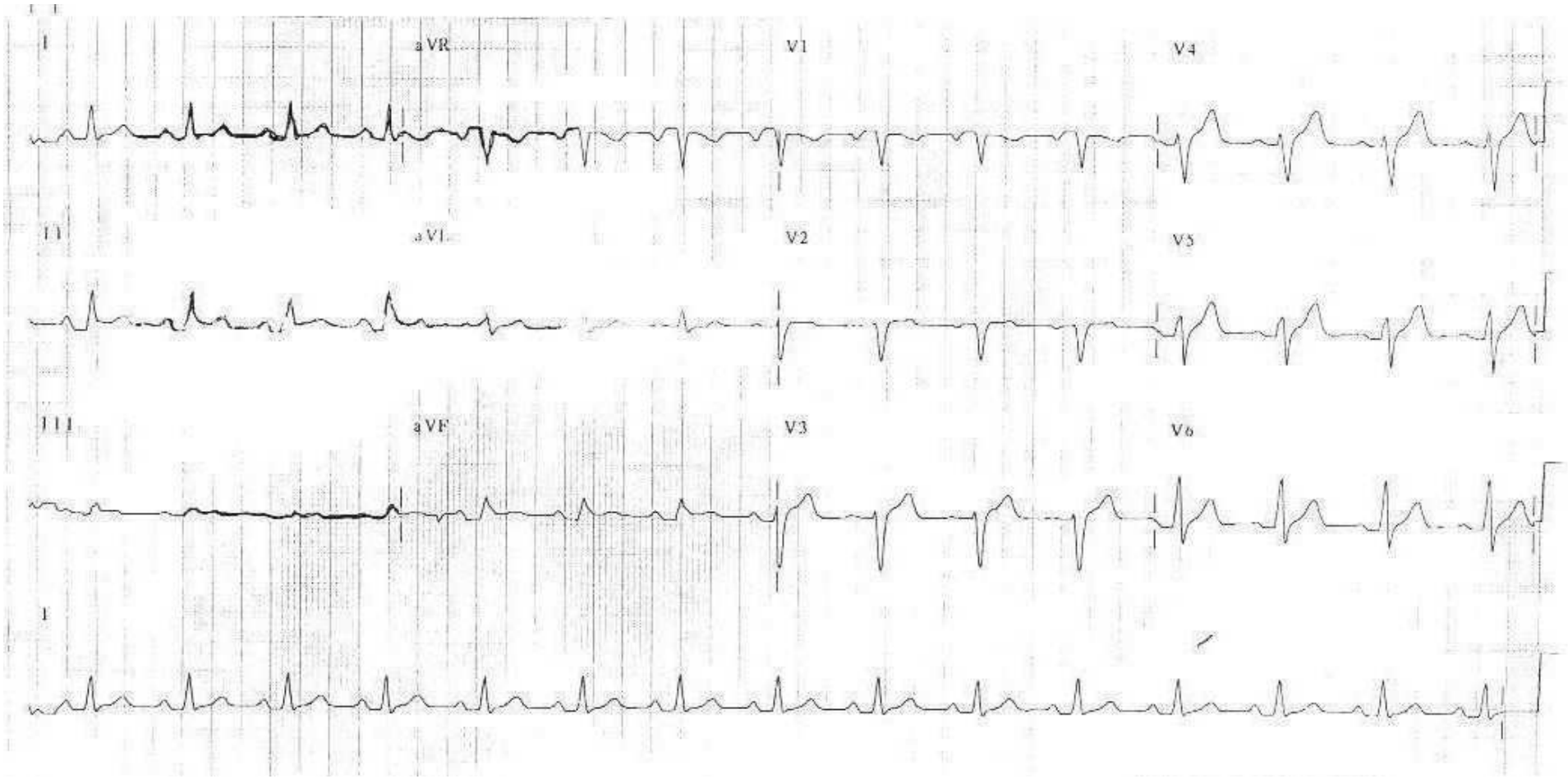
Inferior Injury……



Right Ventricle Injury – 1mm ST elevation in V4R



Posterior Injury – 99% Circumflex, 90% RCA



Pericarditis – note PR depression in II

EKG Criteria for STEMI with pre-existing LBBB

- ST elevation greater than or equal to 0.1 mV in leads with a positive QRS
- ST depression greater than or equal to 0.1 mV in V1 to V3
- ST elevation greater than or equal to 0.5 mV in leads with a negative QRS

Killip Class and Mortality

Table 7. Percent Mortality by Killip Class*

Killip Class	Killip and Kimball (Inhospital)	Fibrinolytic Trials (30 Days)			
		GISSI-1 (157)		International Study Group:	ASSENT-2:
		Placebo	Fibrinolytic	Fibrinolytic (354)	Fibrinolytic (28)
I	6	7	6	5	5
II	17	20	16	18	13
III	38	39	33	32	26
IV	81	70	70	72	56†

Class I = no rales, no S_3; Class II = rales less than 50%; Class III = pulmonary edema; Class IV = cardiogenic shock.
*Values cited are subject to survivor bias.
†Highly selected group of patients.
Modified with permission from Topol. Textbook of Cardiovascular Medicine. 2nd ed. Philadelphia, PA: Lippincott-Williams & Wilkins; 2002:438 (219).

Guideline Use of Biomarkers for STEMI

Class I

1. Cardiac-specific troponins should be used as the optimum biomarkers for the evaluation of patients with STEMI who have coexistent skeletal muscle injury. *(Level of Evidence: C)*

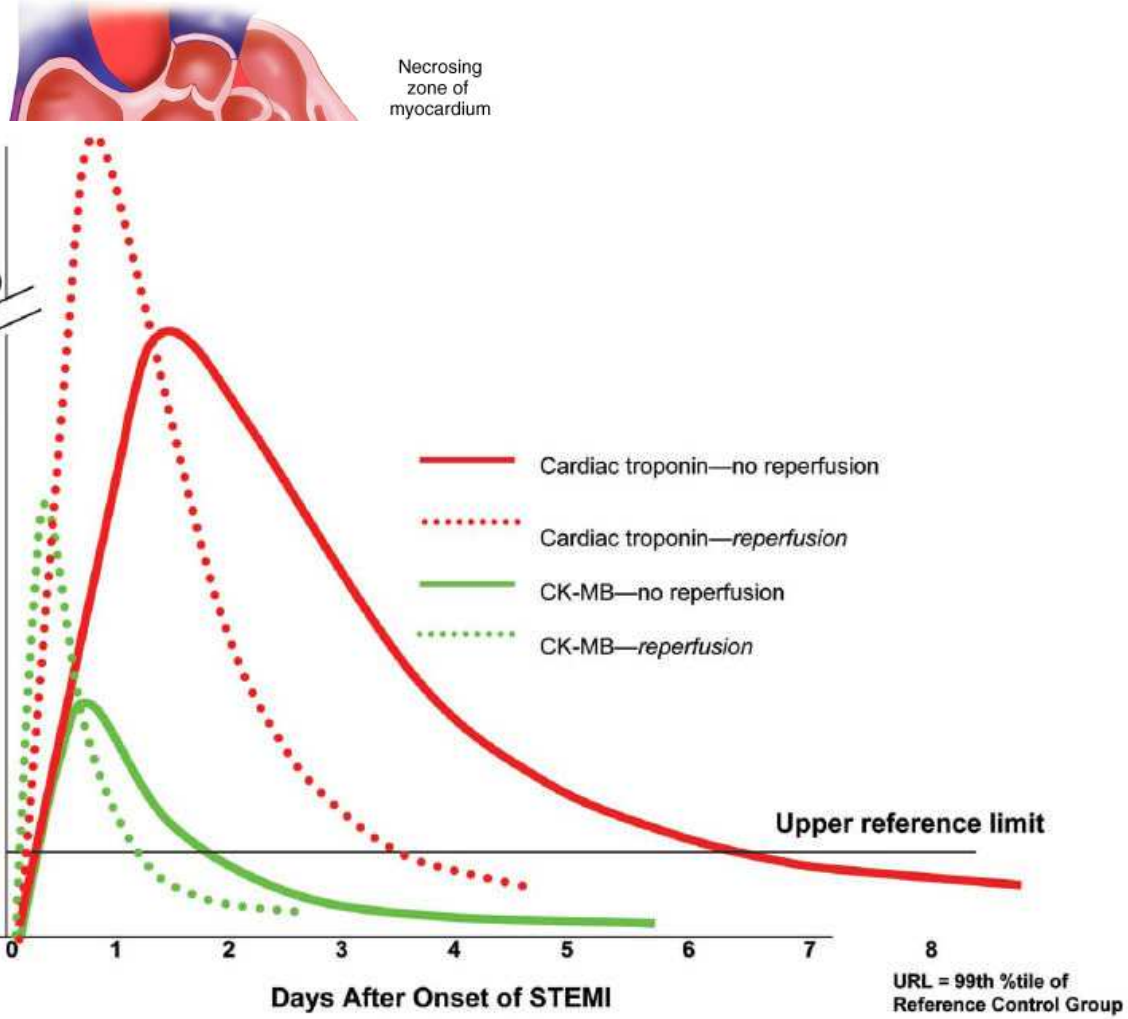
2. For patients with ST elevation on the 12-lead ECG and symptoms of STEMI, reperfusion therapy should be initiated as soon as possible and is not contingent on a biomarker assay. *(Level of Evidence: C)*

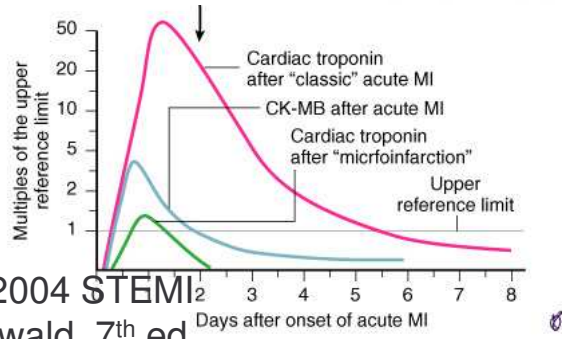
Class IIa

Serial biomarker measurements can be useful to provide supportive noninvasive evidence of reperfusion of the infarct artery after fibrinolytic therapy in patients not undergoing angiography within the first 24 hours after fibrinolytic therapy. *(Level of Evidence:B)*

Class III

Serial biomarker measurements should not be relied upon to diagnose reinfarction within the first 18 hours after the onset of STEMI. *(Level of Evidence: C)*





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Management of STEMI

Class 1 Recommendations (in absence of Contraindication)

- Oxygen
- Nitroglycerin (in absence of hypotension or recent PDE-5 inhibitor use [24 hours viagra, levitra; 48 hours for cialis]
- Morphine (2-8mg IV q 5-15min)
- ASA (162-325mg, chewed)
- Beta Blocker (oral preferred vs IV)
- Reperfusion

PCI vs Lysis - Early

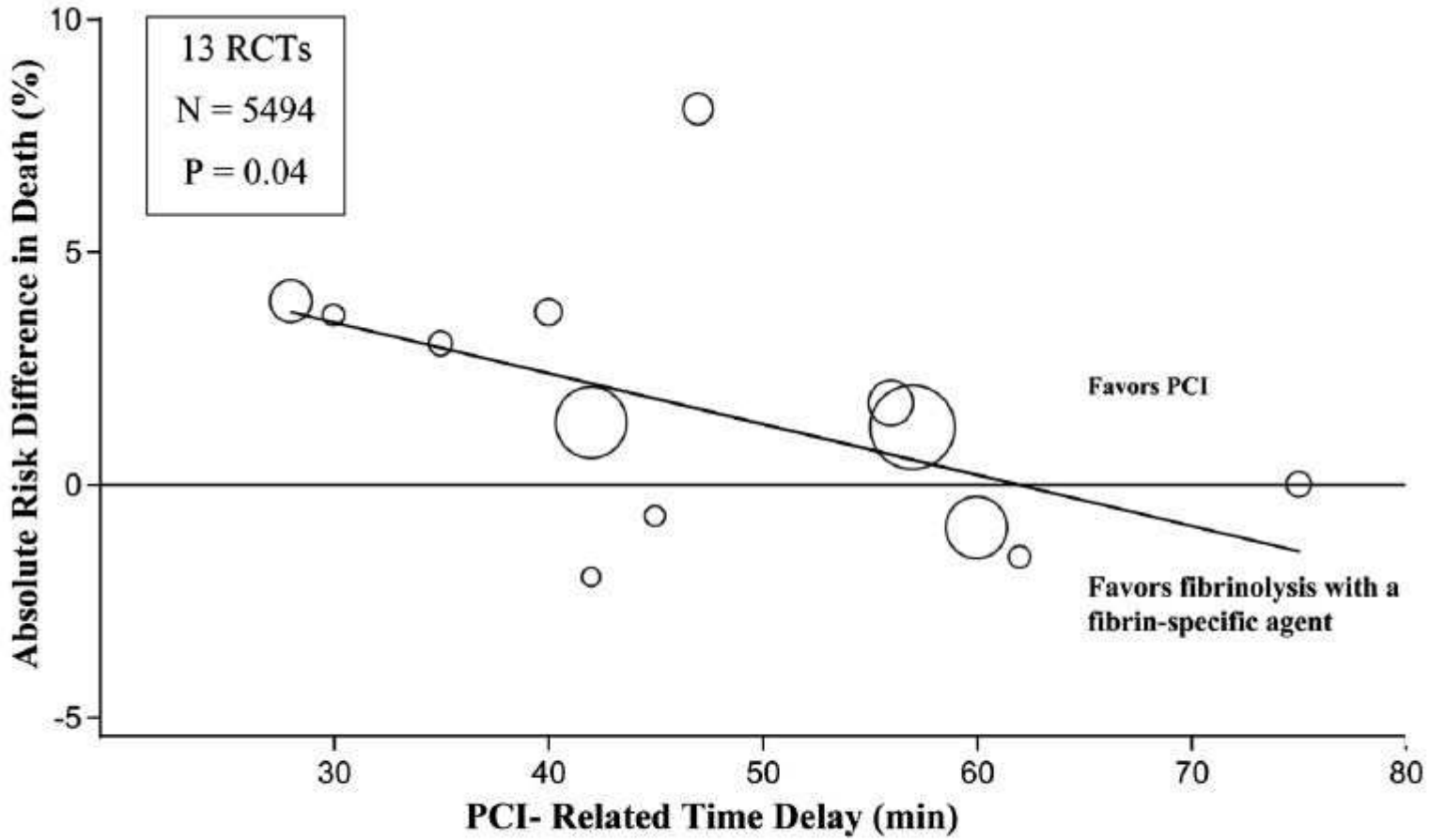

Figure 15. PCI versus lysis with fibrin-specific agents:is timing (almost) everything? RCT = randomized controlled trial; N = Number of patients; PCI = percutaneous coronary intervention. Modified from Nallamothu and Bates. Am J Cardiol 2003;92:824-6 (305). Copyright 2003, with permission from Excerpta Medica, Inc.

Choosing Lysis vs PCI

Fibrinolysis is generally preferred if (See Section 6.3.1.6.3.1):

- *Early Presentation (less than or equal to 3 hours from symptom onset and delay to invasive strategy) (see below)*
- *Invasive Strategy is not an option*
  - Catheterization lab occupied/not available
  - Vascular access difficulties
  - Lack of access to a skilled PCI lab †‡
- *Delay to Invasive Strategy*
  - Prolonged transport
  - (Door-to-Balloon) – (Door-to-Needle) is greater than 1 hour *§
  - Medical Contact-to-Balloon or Door-to-Balloon is greater than 90 minutes

An Invasive Strategy is generally preferred if (See Section 6.3.1.6.4.2):

- *Skilled PCI lab available with surgical backup* †‡
  - Medical Contact-to-Balloon or Door-to-Balloon is less than 90 minutes
  - (Door-to-Balloon) – (Door-to-Needle) is less than 1 hour *
- *High Risk from STEMI*
  - Cardiogenic shock
  - Killip class is greater than or equal to 3
- *Contraindications to fibrinolysis including increased risk of bleeding and ICH*
- *Late Presentation*
  - The symptom onset was greater than 3 hours ago
- *Diagnosis of STEMI is in doubt*

STEMI = ST-elevation myocardial infarction; PCI = percutaneous coronary intervention; ICH = intracranial hemorrhage.
*Applies to fibrin-specific agents (See Figure 15).
†Operator experience greater than a total of 75 Primary PCI cases/year.
‡Team experience greater than a total of 36 Primary PCI cases/year.
§This calculation implies that the estimated delay to the implementation of the invasive strategy is greater than one hour versus initiation of fibrinolytic therapy immediately with a fibrin-specific agent.

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Absolute lytic contraindications

Table 12. Contraindications and Cautions for Fibrinolysis in ST-Elevation Myocardial Infarction*

Absolute contraindications

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

Relative contraindications

Relative contraindications

- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP greater than 180 mm Hg or DBP greater than 110 mm Hg)†
- History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (greater than 10 minutes) CPR or major surgery (less than 3 weeks)
- Recent (within 2-4 weeks) internal bleeding
- Noncompressible vascular punctures
- For streptokinase/anistreplase: prior exposure (more than 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

Time to Balloon impacts mortality

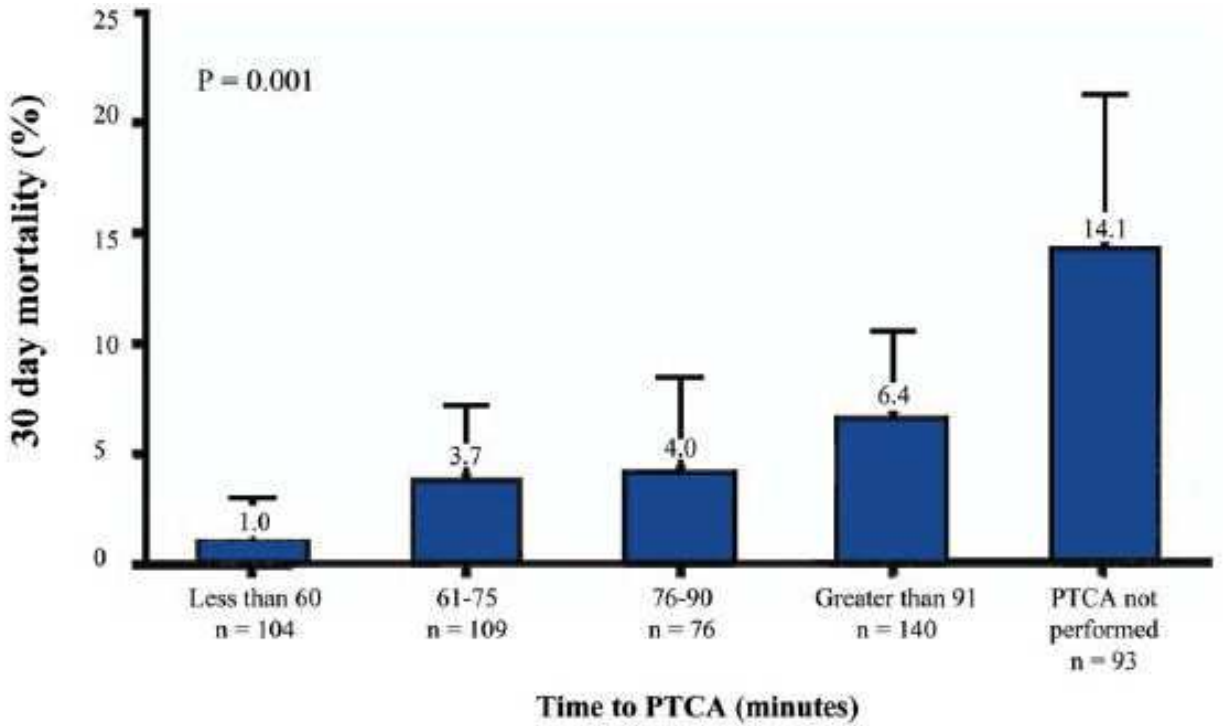
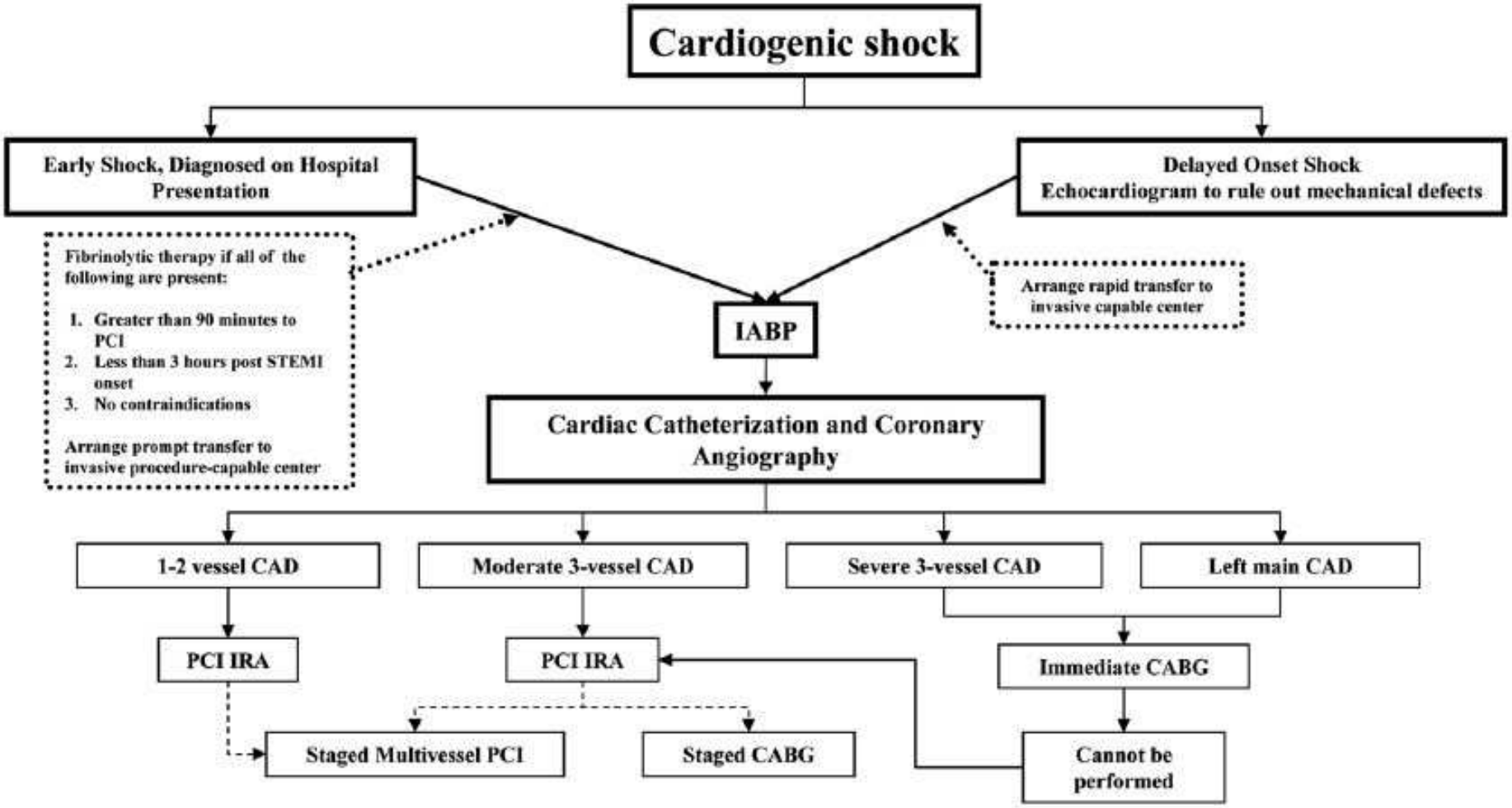


Figure 22. Relationship between 30-day mortality and time from study enrollment to first balloon inflation. Patients assigned to angioplasty in whom angioplasty was not performed are also shown. PTCA = percutaneous transluminal coronary angioplasty. Reprinted with permission from Berger et al. Circulation 1999;100:14-20 (294).

Cardiogenic Shock



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Basic Thrombolytic Therapy

- Given up to 12 hours after symptom onset
- Aspirin
- Fibrin Specific Lytics
  - rPA (10 units x 2, separated by 30 minutes)
  - tnK (weight based single bolus 30-50mg)
- Heparin (used with fibrin specific lytics)
  - 60 unit/kg bolus (NTE 4000 units)
  - 12 unit/kg/hour infusion (NTE 1000 units/hour)
    - Use for 24-48? Hours
- Plavix? (New data from Commit/Clarity trials)

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COMMIT collaborative group. Addition of clopidogrel to aspirin in 45 852 patients with acute myocardial infarction: randomised placebo-controlled trial. *Lancet* 2005; 366:1607-1621.

Sabatine MS, Cannon CP, Gibson CM, et al for the CLARITY-TIMI 28 Investigators. Addition of clopidogrel to aspirin and fibrinolytic therapy for myocardial infarction with ST-segment elevation. *N Engl J Med* 2005; 352:1179-1189

Basic Primary PCI

- ASA
- Heparin
  - 50-70 unit/kg bolus with 2b-3a
    - Goal ACT 200-250 at time of PCI
  - 70-100 unit/kg bolus without 2b-3a
    - Goal ACT 250-300 at time of PCI
- Glycoprotein 2b-3a
  - Reopro preferred
    - (0.25mg/kg bolus, 0.125 mcg/kg/min infusion)
- Plavix

Ancillary medications

- ACE inhibitors
  - Oral ACE inhibitor, if BP allows, useful for anterior infarctions or LVEF <=40%; start within 24 hours of STEMI
- Aldosterone antagnosits (epleronone, aldactone)
  - Class 1 indication for Post MI LV dysfunction (LVEF <=40) AND either Diabetes or CHF
- Statins – not mentioned as initial treatment in guidelines

Faded Fads

- IV ACE inhibitor is discouraged
- IV insulin recommended only for hyperglycemia (GIK is out of favor)
- Magnesium useful to correct hypomagnesemia or empiric treatment of torsades (no routine supplementation)
- Sublingual nifedipine is contraindicated for all purposes

References

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- Braunwald E, Antman EM, Beasley JW, Califf RM, Cheitlin MD, Hochman JS, Jones RH, Kereiakes D, Kupersmith J, Levin TN, Pepine CJ, Schaeffer JW, Smith EE III, Steward DE, Théroux P. ACC/AHA 2002 guideline update for the management of patients with unstable angina and non–ST-segment elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Unstable Angina). 2002.
- Brauwald E, Zipes DP, Libby P, Bonow R. Braunwald's Heart Disease, 7th edition. W.B. Saunders Company; 2004.