

Acute Coronary Syndrome

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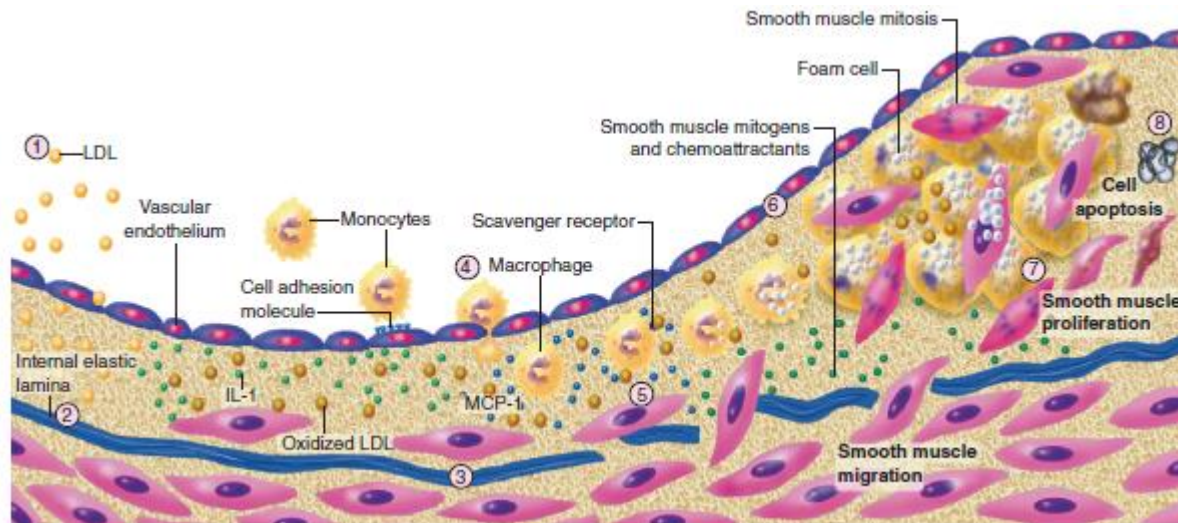
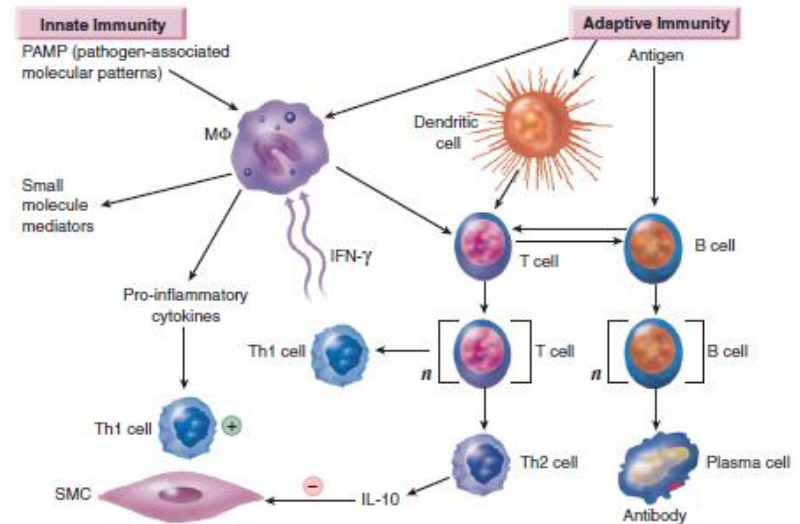
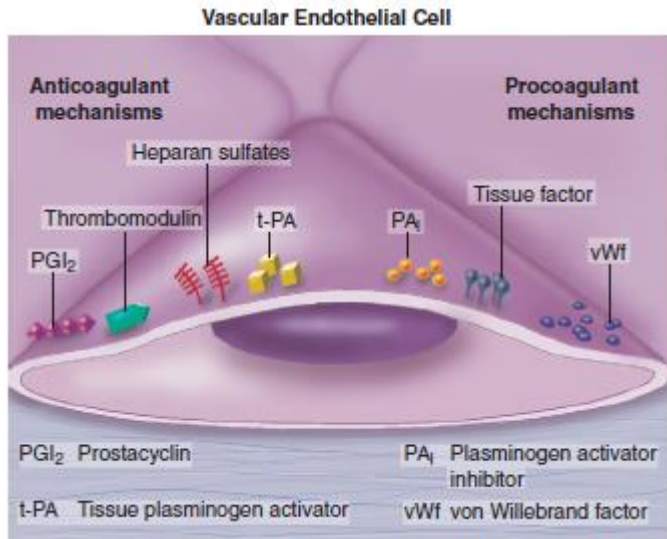
Contents

- Introduction
- Definition
- Clinical Features
- Diagnosis
- Management
- Critical Care in ACS

Perspective: Coronary artery disease

- Leading cause of mortality worldwide
- By 2020, will be first in the leading causes of disability
- Death rates declining for the past three decades in the west, these rates are **rising in India**.
- The prevalence of CAD has increased from 1.1% to 7.5% in the urban population and from 2.1% to 3.7% in the rural population
- CAD tends to occur at a **younger age** in Indians, with **more extensive angiographic involvement** contributed genetic, metabolic, conventional and nonconventional risk factors

Biology of Atherosclerosis



Universal definition of myocardial infarction

- Acute myocardial infarction (MI) defines **cardiomyocyte necrosis** in a **clinical setting** consistent with **acute myocardial ischaemia**
- A combination of **criteria** is required to meet the diagnosis of acute MI, namely the detection of an increase and/or decrease of a **cardiac biomarker**, preferably high-sensitivity cardiac troponin, with at least one value above the 99th percentile of the upper reference limit and **at least one of the following**:
 - (1) Symptoms of ischaemia.
 - (2) New or presumed new significant ST-T wave changes or left bundle branch block on 12-lead ECG.
 - (3) Development of pathological Q waves on ECG.
 - (4) Imaging evidence of new or presumed new loss of viable myocardium or regional wall motion abnormality.
 - (5) Intracoronary thrombus detected on angiography or autopsy.

Types of MI

Type 1: Spontaneous myocardial infarction

Spontaneous myocardial infarction related to atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection with resulting intraluminal thrombus in one or more of the coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis. The patient may have underlying severe CAD but on occasion non-obstructive or no CAD.

Type 2: Myocardial infarction secondary to an ischaemic imbalance

In instances of myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or demand, e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/brady-arrhythmias, anaemia, respiratory failure, hypotension, and hypertension with or without LVH.

Type 3: Myocardial infarction resulting in death when biomarker values are unavailable

Cardiac death with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or new LBBB, but death occurring before blood samples could be obtained, before cardiac biomarker could rise, or in rare cases cardiac biomarkers were not collected.

Type 4a: Myocardial infarction related to percutaneous coronary intervention (PCI)

Myocardial infarction associated with PCI is arbitrarily defined by elevation of cTn values $>5 \times 99^{\text{th}}$ percentile URL in patients with normal baseline values ($\leq 99^{\text{th}}$ percentile URL) or a rise of cTn values $>20\%$ if the baseline values are elevated and are stable or falling. In addition, either (i) symptoms suggestive of myocardial ischaemia, or (ii) new ischaemic ECG changes or new LBBB, or (iii) angiographic loss of patency of a major coronary artery or a side branch or persistent slow- or no-flow or embolization, or (iv) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required.

Type 4b: Myocardial infarction related to stent thrombosis

Myocardial infarction associated with stent thrombosis is detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac biomarkers values with at least one value above the 99^{th} percentile URL.

Type 5: Myocardial infarction related to coronary artery bypass grafting (CABG)

Myocardial infarction associated with CABG is arbitrarily defined by elevation of cardiac biomarker values $>10 \times 99^{\text{th}}$ percentile URL in patients with normal baseline cTn values ($\leq 99^{\text{th}}$ percentile URL). In addition, either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Myocardial Injury

Plaque rupture with thrombus



MI Type 1

Vasospasm or endothelial dysfunction



MI Type 2

Fixed atherosclerosis and supply-demand imbalance

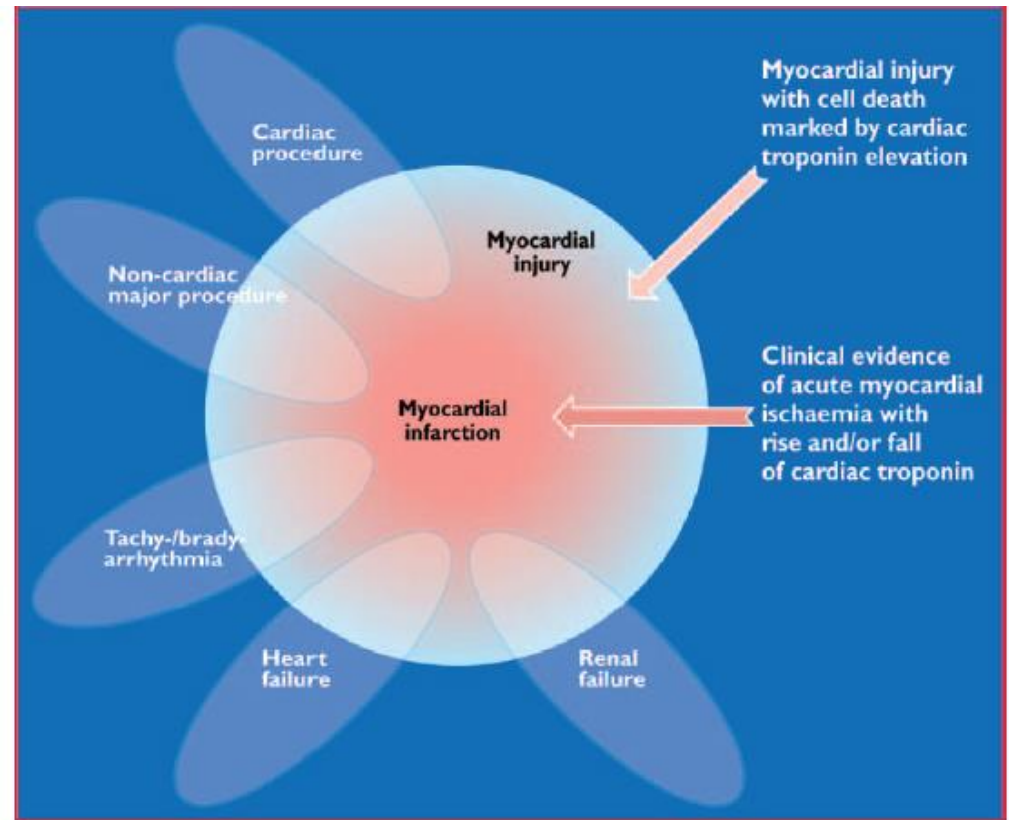


MI Type 2

Supply-demand imbalance alone

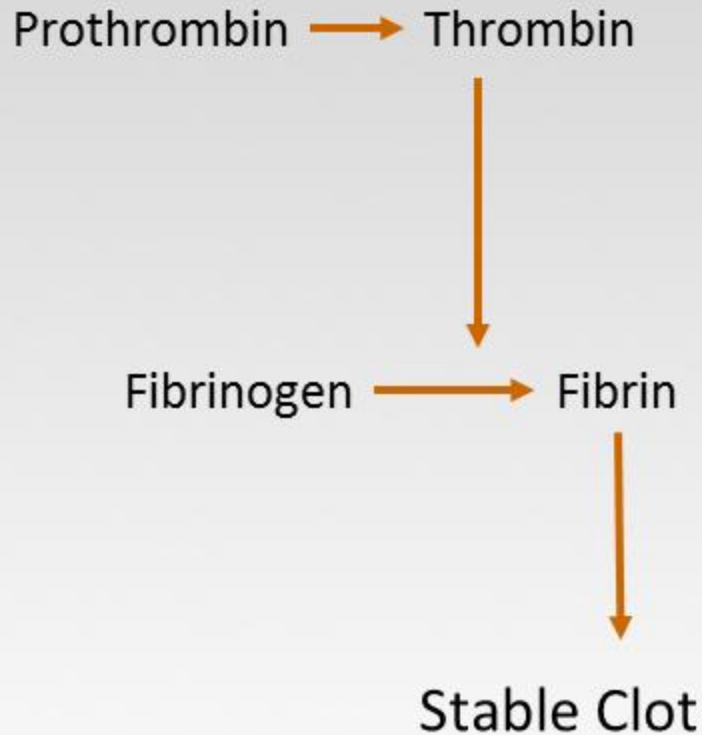


MI Type 2

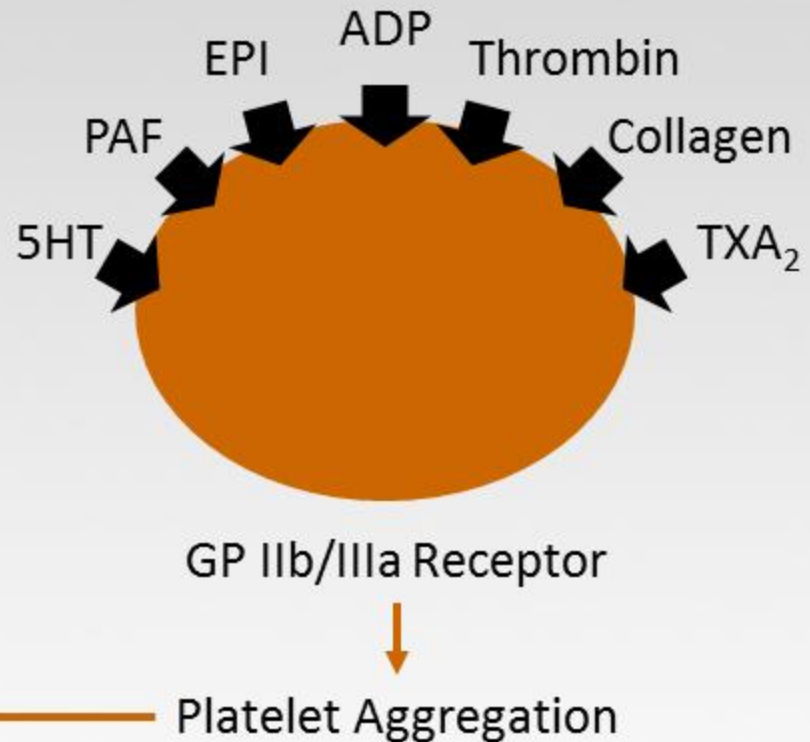


Clotting Cascade

Coagulation Cascade

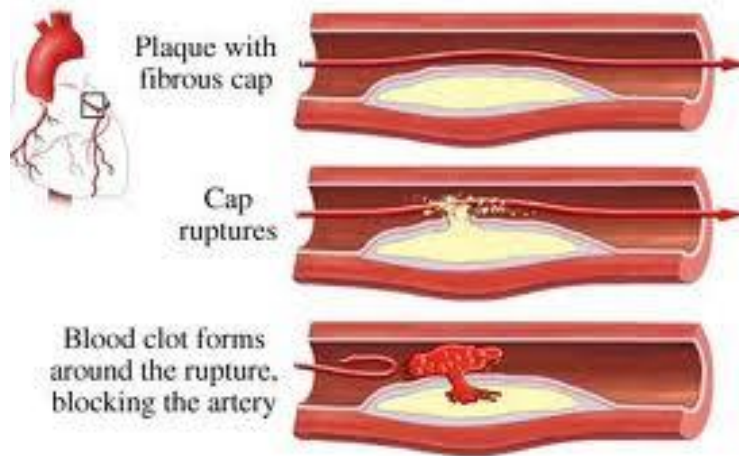


Platelet Cascade



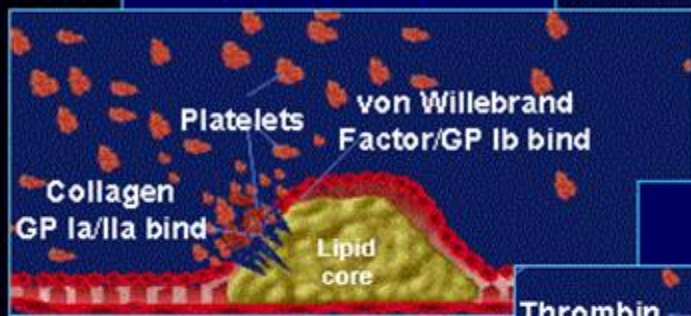
ADP = adenosine diphosphate; EPI = epinephrine; PAF = platelet-activating factor; 5HT = 5-hydroxytryptamine; TXA₂ = thromboxane A₂; GP = glycoprotein

The ruptured Plaque= ACS

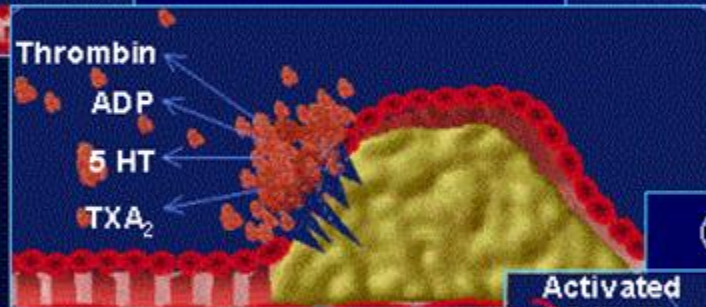


Cascade

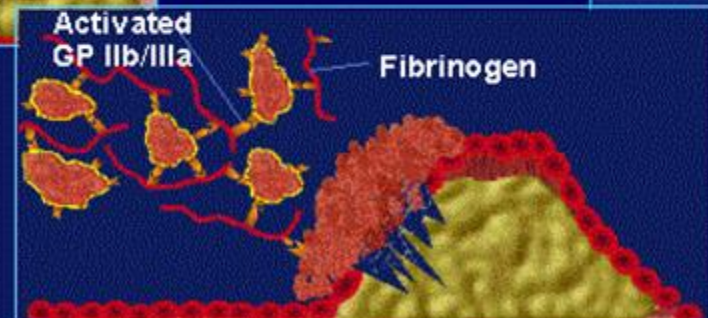
① Adhesion



② Activation



③ Aggregation



Handin RL. *Harrison's Principles of Internal Medicine*. Vol 1. 14th ed. NY, NY: McGraw-Hill; 1998:339-345.

Schafer AJ. *Am J Med*. 1996;101:199-209.

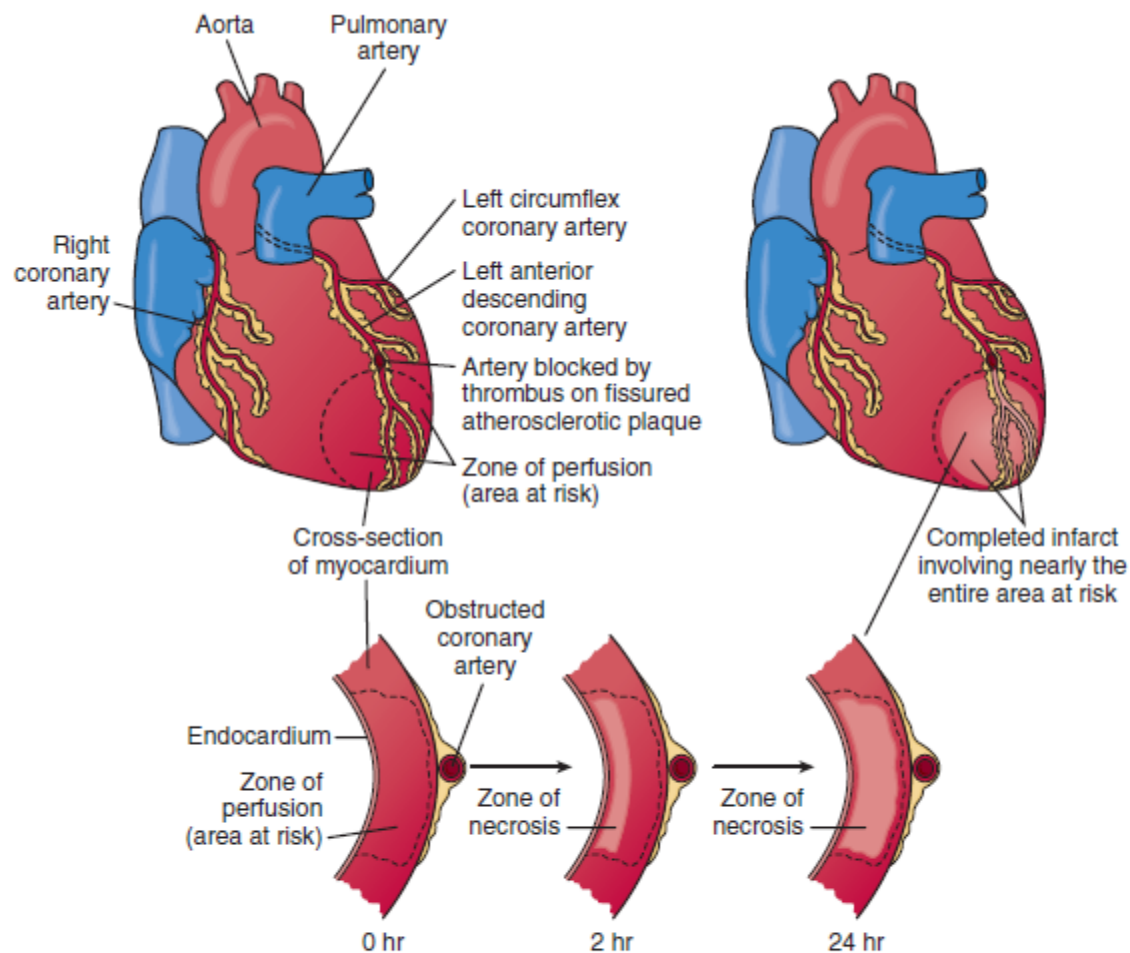


FIGURE 54-3 Schematic representation of the progression of myocardial necrosis after coronary artery occlusion. Necrosis begins in a small zone of the myocardium beneath the endocardial surface in the center of the ischemic zone. This entire region of myocardium (dashed outline) depends on the occluded vessel for perfusion and is the area at risk. A narrow zone of myocardium immediately beneath the endocardium is spared from necrosis because it can be oxygenated by diffusion from the ventricle. (From Schoen FJ: *The heart*. In Kumar V, Abbas AK, Fausto N, Aster J [eds]: *Robbins and Cotran Pathologic Basis of Disease*. 8th ed. Philadelphia, WB Saunders, 2010, pp 529-587.)

Clinical Features

TABLE 53-1 Common Causes of Acute Chest Pain

SYSTEM	SYNDROME	CLINICAL DESCRIPTION	KEY DISTINGUISHING FEATURES
Cardiac	Angina	Retrosternal chest pressure, burning, or heaviness; radiating occasionally to neck, jaw, epigastrium, shoulders, left arm	Precipitated by exercise, cold weather, or emotional stress; duration 2-10 min
	Rest or unstable angina	Same as angina, but may be more severe	Typically <20 min; lower tolerance for exertion; crescendo pattern
	Acute myocardial infarction	Same as angina, but may be more severe	Sudden onset, usually lasting ≥30 min; often associated with shortness of breath, weakness, nausea, vomiting
	Pericarditis	Sharp, pleuritic pain aggravated by changes in position; highly variable duration	Pericardial friction rub
Vascular	Aortic dissection	Excruciating, ripping pain of sudden onset in anterior of chest, often radiating to back	Marked severity of unrelenting pain; usually occurs in setting of hypertension or underlying connective tissue disorder such as Marfan syndrome
	Pulmonary embolism	Sudden onset of dyspnea and pain, usually pleuritic with pulmonary infarction	Dyspnea, tachypnea, tachycardia, signs of right heart failure
	Pulmonary hypertension	Substernal chest pressure, exacerbated by exertion	Pain associated with dyspnea and signs of pulmonary hypertension
Pulmonary	Pleuritis and/or pneumonia	Pleuritic pain, usually brief, over involved area	Pain pleuritic and lateral to midline, associated with dyspnea
	Tracheobronchitis	Burning discomfort in midline	Midline location, associated with coughing
	Spontaneous pneumothorax	Sudden onset of unilateral pleuritic pain, with dyspnea	Abrupt onset of dyspnea and pain
Gastrointestinal	Esophageal reflux	Burning substernal and epigastric discomfort, 10-60 min in duration	Aggravated by large meal and postprandial recumbency; relieved by antacid
	Peptic ulcer	Prolonged epigastric or substernal burning	Relieved by antacid or food
	Gallbladder disease	Prolonged epigastric or right upper quadrant pain	Unprovoked or following meal
	Pancreatitis	Prolonged, intense epigastric and substernal pain	Risk factors including alcohol, hypertriglyceridemia, medications
Musculoskeletal	Costochondritis	Sudden onset of intense fleeting pain	May be reproduced by pressure over affected joint; occasionally, swelling and inflammation over costochondral joint
	Cervical disc disease	Sudden onset of fleeting pain	May be reproduced with movement of neck
	Trauma or strain	Constant pain	Reproduced by palpation or movement of chest wall or arms
Infectious	Herpes zoster	Prolonged burning pain in dermatomal distribution	Vesicular rash, dermatomal distribution
Psychological	Panic disorder	Chest tightness or aching, often accompanied by dyspnea and lasting 30 minutes or more, unrelated to exertion or movement	Patient may have other evidence of emotional disorder

History

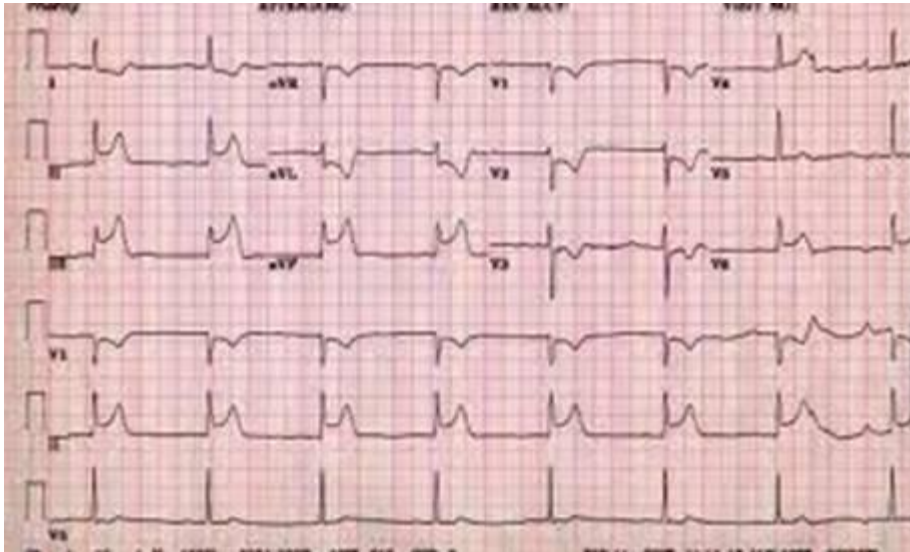
- Characterize symptoms
 - Typical angina
 - Substernal chest discomfort of characteristic quality and duration
 - Pressure, squeezing, radiation, diaphoresis, nausea
 - Provoked by emotional stress or exertion
 - Reduced with rest, nitroglycerine
 - Atypical angina
 - 2 or fewer of above
 - Non-cardiac chest pain
 - 1 or fewer of above
- **P** – Palliative/provocative factors
- **Q** – Quality of discomfort
- **R** – Radiation
- **S** – Symptoms associated with discomfort
- **T** - Timing

Physical Exam

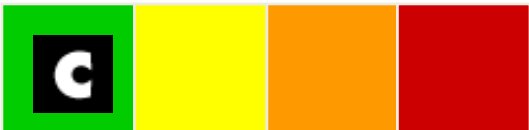
- Measurement of vital signs
 - Blood pressure
 - Pulse
 - Pulse oximetry
 - Temperature
- Focused assessment
 - Cardiovascular system
 - Respiratory system
 - Mental status
- Increased risk with:
 - Hypotension
 - Tachycardia
 - Pulmonary rales/pulmonary edema
 - New murmurs/heart sounds
 - Diminished peripheral pulses

Clinical Data

ECG



- 12-lead ECG should be obtained **WITHIN 10 MINUTES**
- Interpret for:
 - T-wave inversions
 - ST depression
 - ST elevation
 - Left bundle branch block



12 – lead ECG within 10 minutes

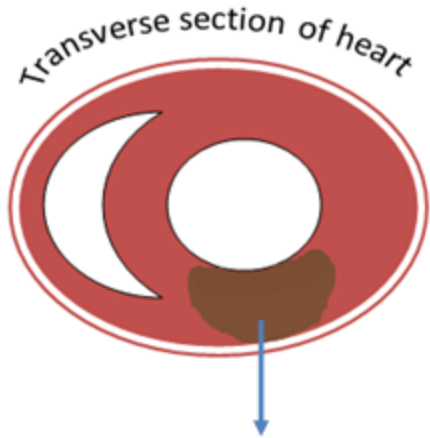
TABLE 53-3 Likelihood That Signs and Symptoms Represent an Acute Coronary Syndrome

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 ANY OF THE FOLLOWING
History	• Chest or left arm pain or discomfort as chief symptom reproducing documented angina prior • Known history of coronary artery disease, including MI	• Chest or left arm pain or discomfort as chief symptom • Age > 70 yr • Male sex • Diabetes mellitus	• Probable ischemic symptoms in absence of any of the intermediate-likelihood characteristics • Recent cocaine use
Examination	• Transient mitral regurgitation murmur, hypotension, diaphoresis, pulmonary edema, or rales	• Extracardiac vascular disease	• Chest discomfort reproduced by palpation
Electrocardiogram	• New, or presumably new, transient ST-segment deviation (≥ 0.1 mV) or T wave inversion (≥ 0.2 mV) in multiple precordial leads	• Fixed Q waves • ST-segment depression 0.05-0.1 mV or T wave inversion > 0.1 mV	• T wave flattening or inversion < 0.1 mV in leads with dominant R waves • Normal ECG
Cardiac markers	• Elevated cardiac TnI, TnT, or CK-MB	• Normal	• Normal

Unstable Angina

- Occurs at rest and prolonged, usually lasting >20 minutes
- New onset angina that limits activity
- Increasing angina: Pain that occurs more frequently, lasts longer periods or is increasingly limiting the patients activity
- No myocardial death
- May present with nonspecific or transient ST segment depressions or elevations
- Normal cardiac enzymes

STEMI vs. NSTEMI

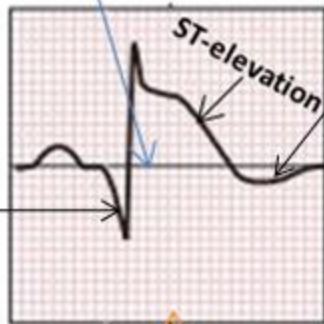


Full thickness damage

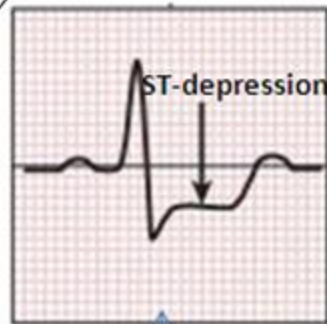
Pathological Q-wave

Isoelectric line

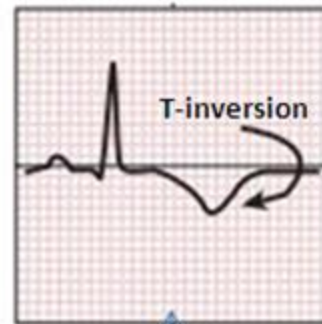
T-wave inversion



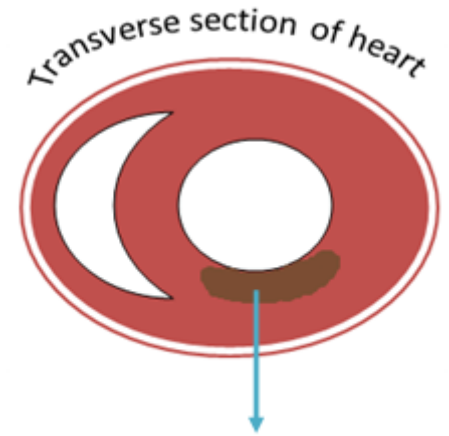
ST-segment elevation in stemi



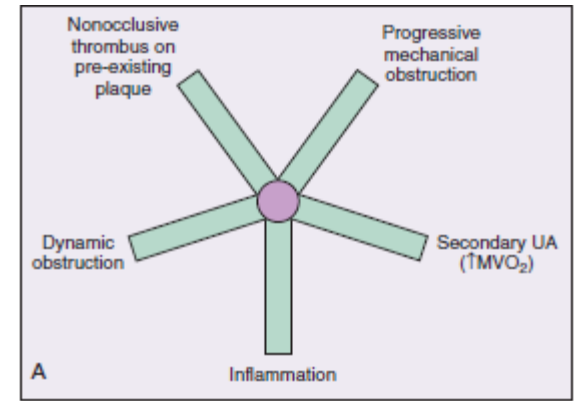
ST-segment depression in nstemi



T-wave inversion in nstemi



Partial thickness damage



STEMI vs. NSTEMI

STEMI

- Total occlusion of vessel
- Cardiac enzymes positive
- Complications more
- Pharmacotherapy same
- Early reperfusion
- Short term mortality higher

NSTEMI

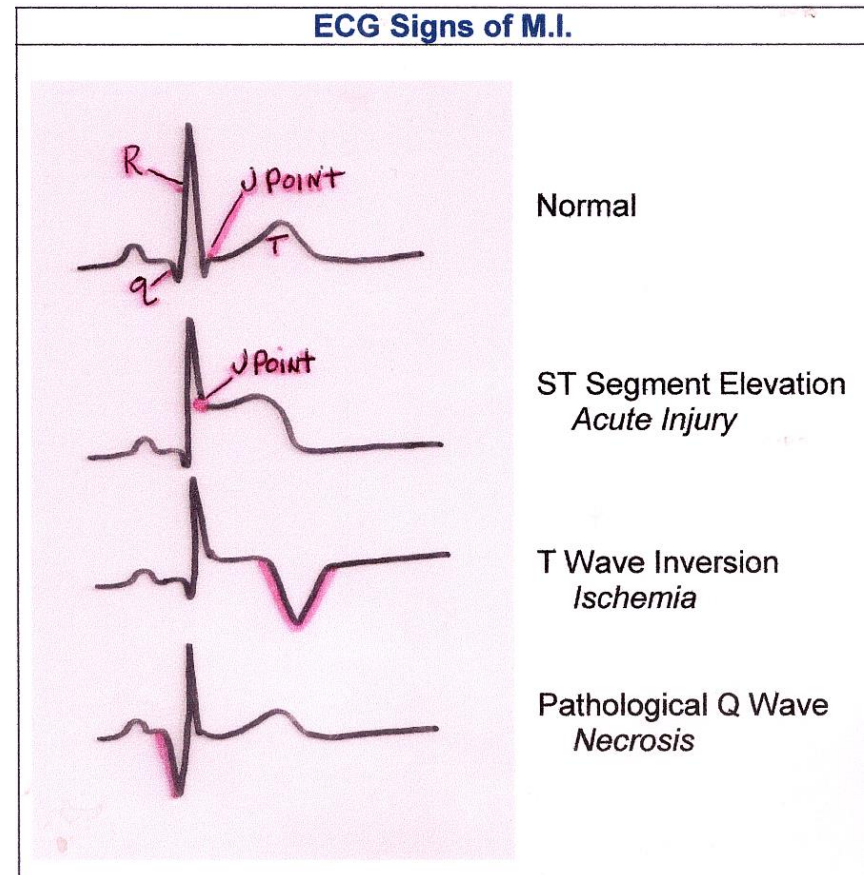
- Subtotal / endocardial involvement of vessels
- Cardiac enzymes positive
- Complications less than STEMI
- Pharmacotherapy same
- Thrombolysis harmful
- Short term mortality lower

Diagnosis

- **ECG**
- **Cardiac Biomarkers**
- Imaging: 2D-Echo (POC)
- Triple rule out: CT
Coronary artery disease
Pulmonary Embolism
Aortic Dissection

ECG

- ST Elevation
 - $> 0.1\text{mV}$ or 2mm
 - Present in 2 or more contiguous leads
 - ECG change most diagnostic of acute MI
 - Increased mortality with increased number of involved leads

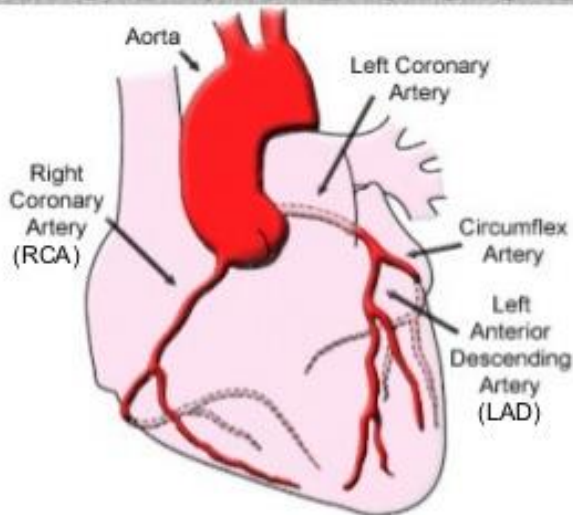
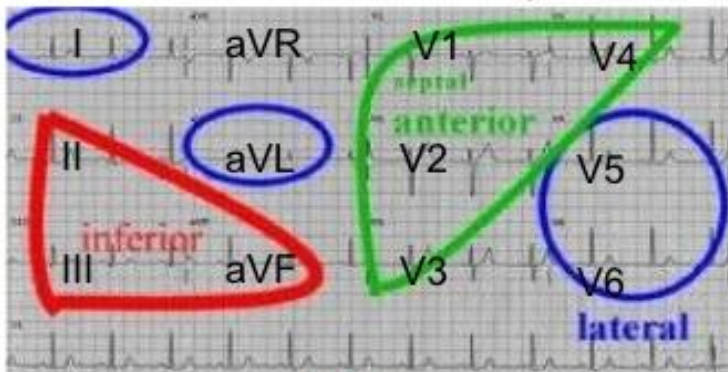


ECG: Localization

ST-SEGMENT

Localizing MI

Look at ST changes, Q wave in all leads. Grouping the leads into anatomical location, we have this:

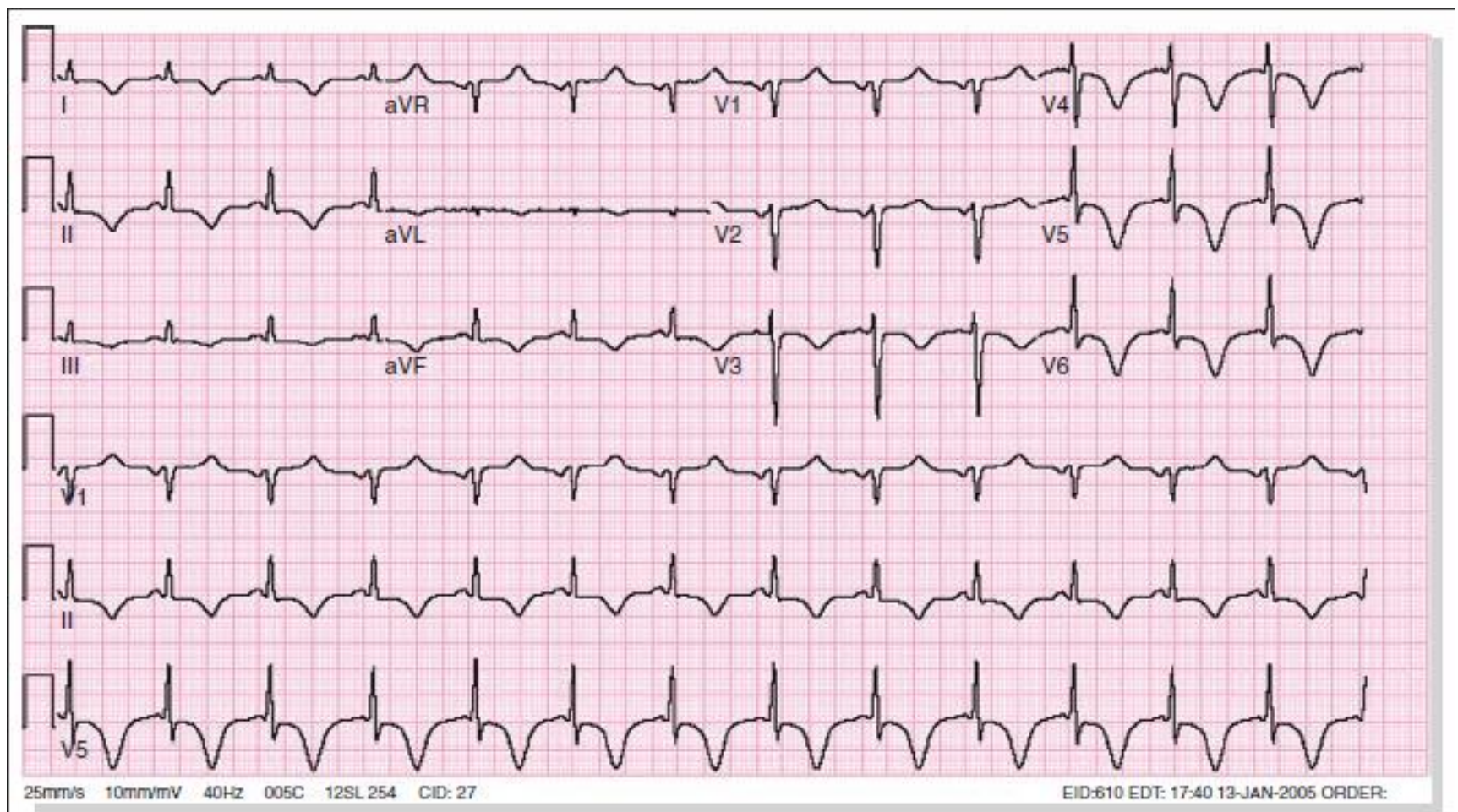


Ischaemic change can be attributed to different coronary arteries supplying the area.

Location of MI	Lead with ST changes	Affected coronary artery
Anterior	V1, V2, V3, V4	LAD
Septum	V1, V2	LAD
left lateral	I, aVL, V5, V6	Left circumflex
inferior	II, III, aVF	RCA
Right atrium	aVR, V1	RCA
*Posterior	Posterior chest leads	RCA
*Right ventricle	Right sided leads	RCA

*To help identify MI, right sided and posterior leads can be applied

NSTEMI



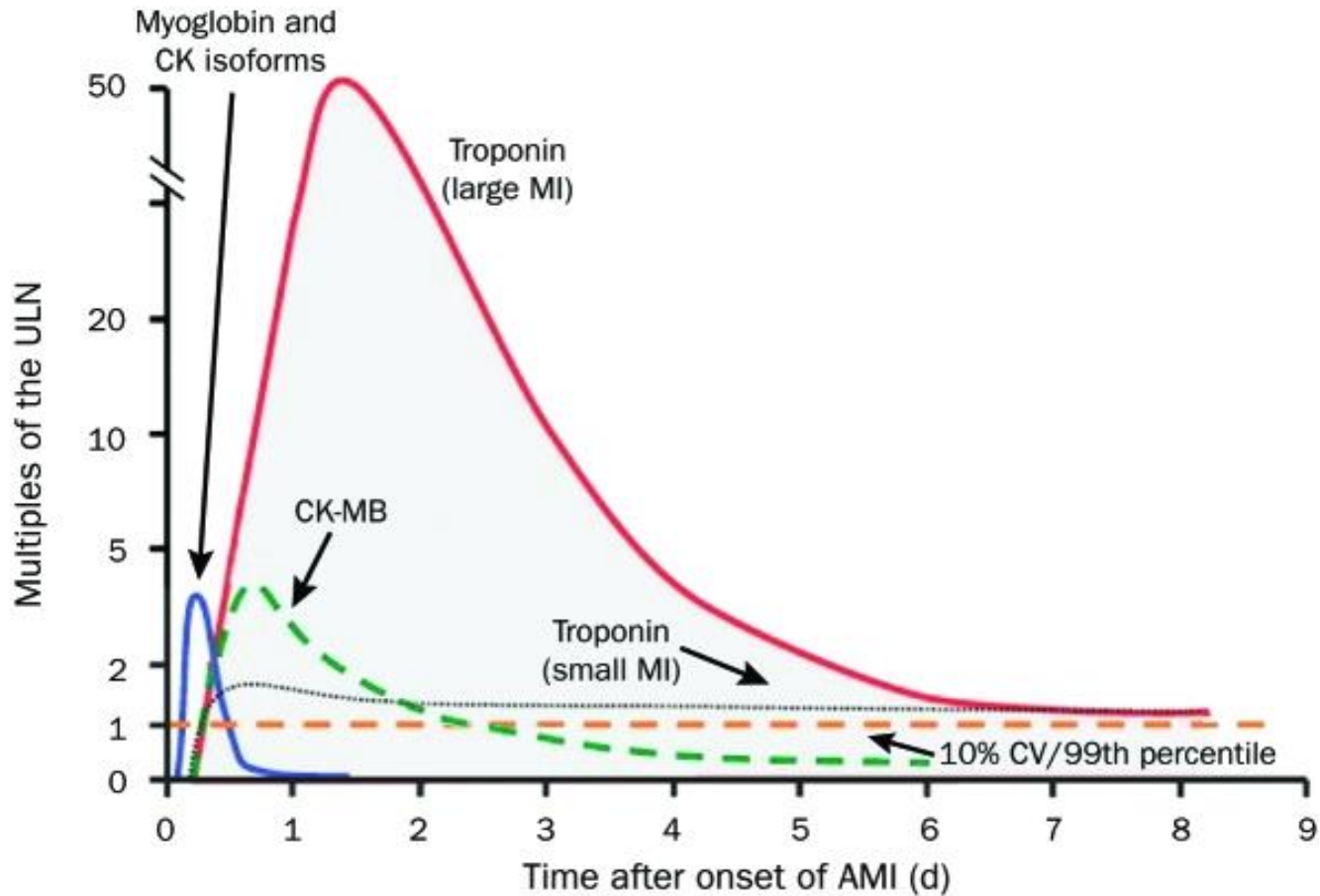
Cardiac Biomarkers

Marker	Advantages	Disadvantages	POC test?	Comment	Clinical recommendation
Cardiac troponins	1. Powerful tool for risk stratification 2. Greater sensitivity and specificity than CK-MB 3. Detection of recent MI up to 2 wk after onset 4. Useful for selection of therapy 5. Detection of reperfusion	1. Low sensitivity in very early phase of MI (<6 h after symptom onset) and requires repeated measurement at 8 to 12 h, if results are negative 2. Limited ability to detect late minor reinfarction	Yes	Data on diagnostic performance and potential therapeutic implications increasingly available from clinical trials	Useful as a single test for efficiently diagnosing NSTEMI (including minor myocardial damage), with serial measurements Clinicians should familiarize themselves with diagnostic "cutoffs" used in their local hospital laboratory
CK-MB	1. Rapid, cost-efficient, accurate assays 2. Ability to detect early reinfarction	1. Loss of specificity in setting of skeletal muscle disease or injury, including surgery 2. Low sensitivity during very early MI (<6 h after symptom onset) or later after symptom onset (>36 h) and for minor myocardial damage (detectable with troponins)	Yes	Familiar to most clinicians	Previous standard and still acceptable diagnostic test in most clinical circumstances
Myoglobin	1. High sensitivity 2. Useful in early detection of MI 3. Detection of reperfusion 4. Most useful in ruling out MI	1. Very low specificity in setting of skeletal muscle injury or disease 2. Rapid return to normal range limits sensitivity for later presentations	Yes	More convenient early marker than CK-MB isoforms because of greater availability of assays for myoglobin; rapid-release kinetics make myoglobin useful for noninvasive monitoring of reperfusion in patients with established MI	

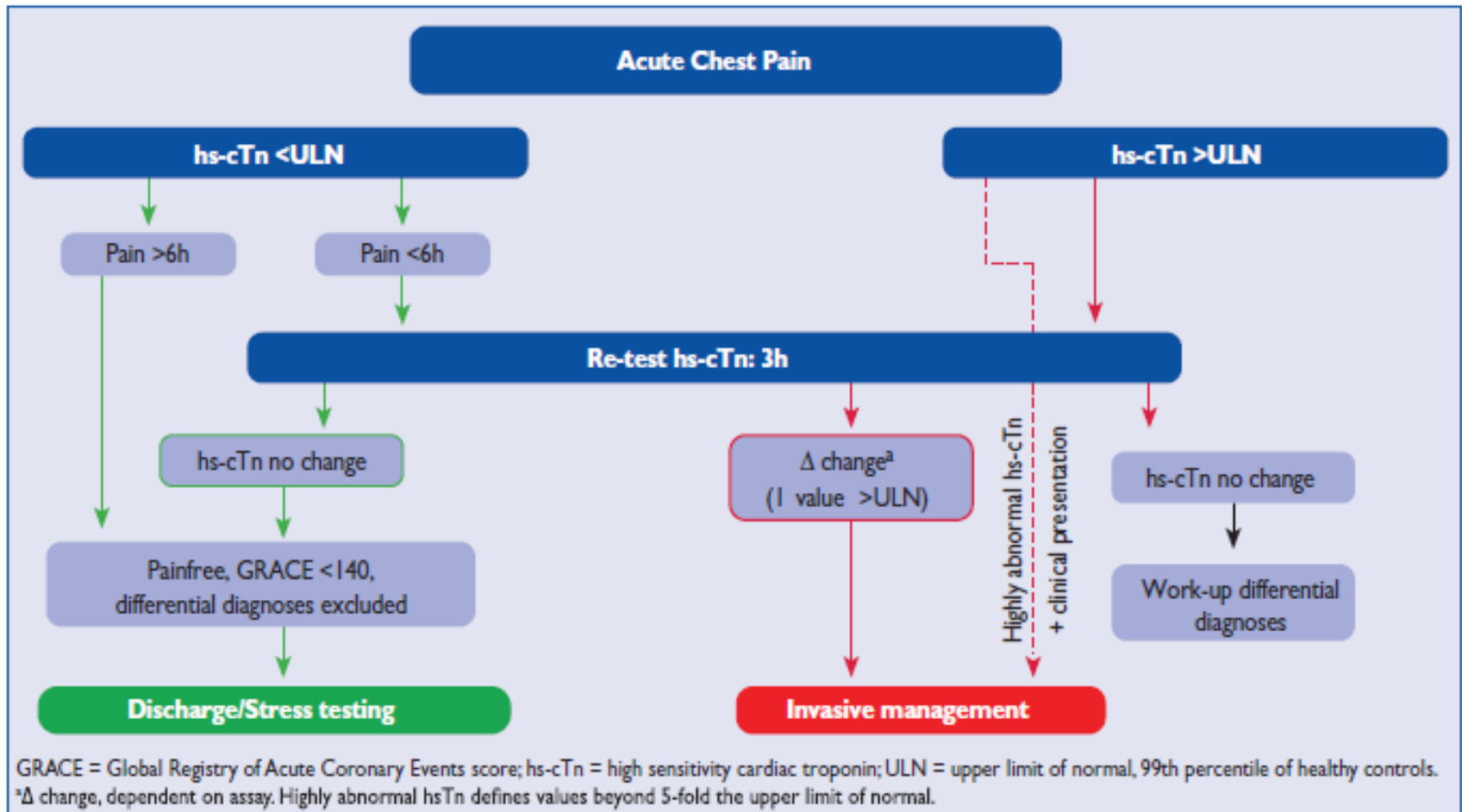
ACS = acute coronary syndrome; CK-MB = muscle and brain fraction of creatine kinase; ECG = electrocardiography; MI = myocardial infarction; NSTEMI = non-ST-segment elevation MI; POC = point-of-care.

From *J Am Coll Cardiol*;

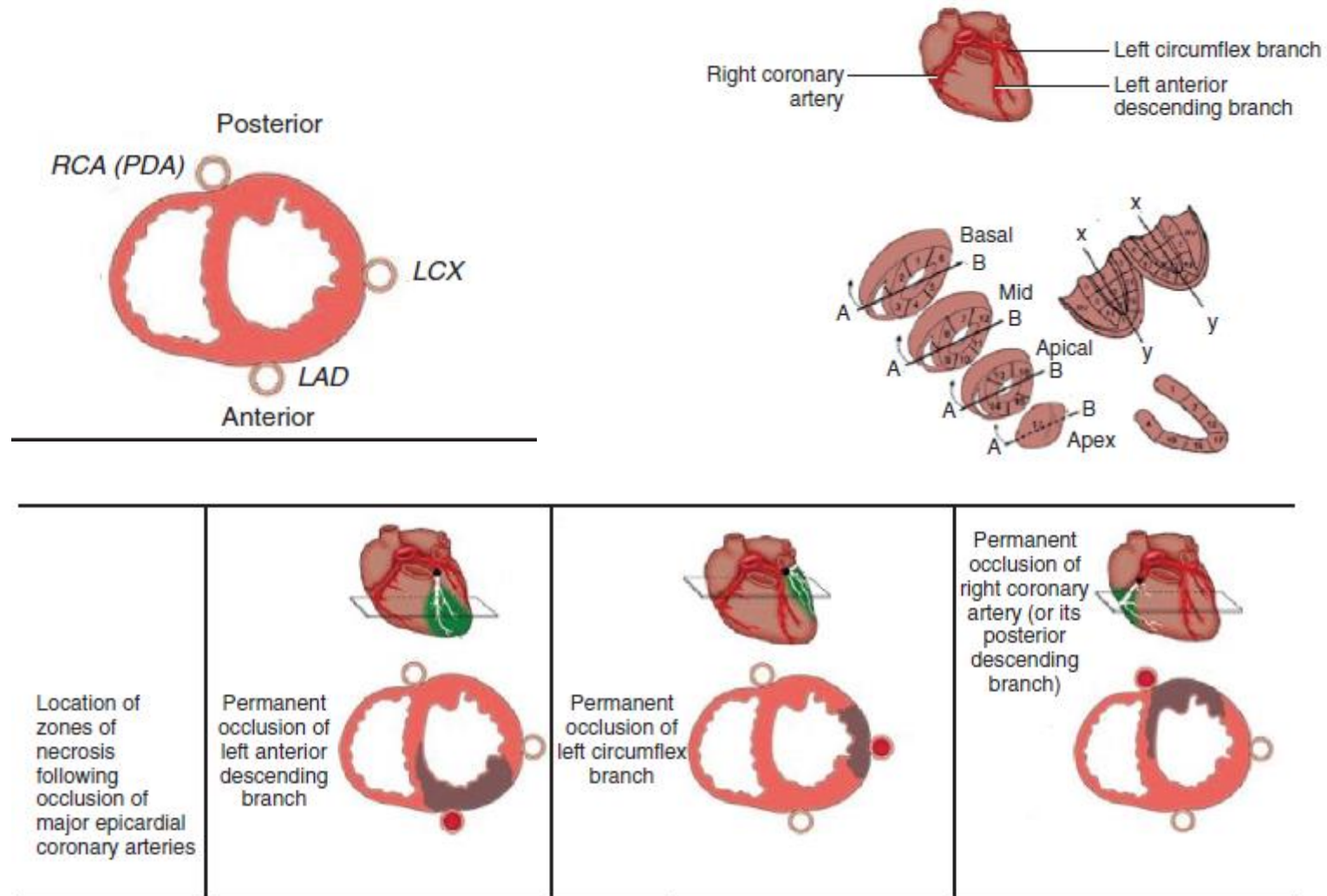
Cardiac Biomarkers



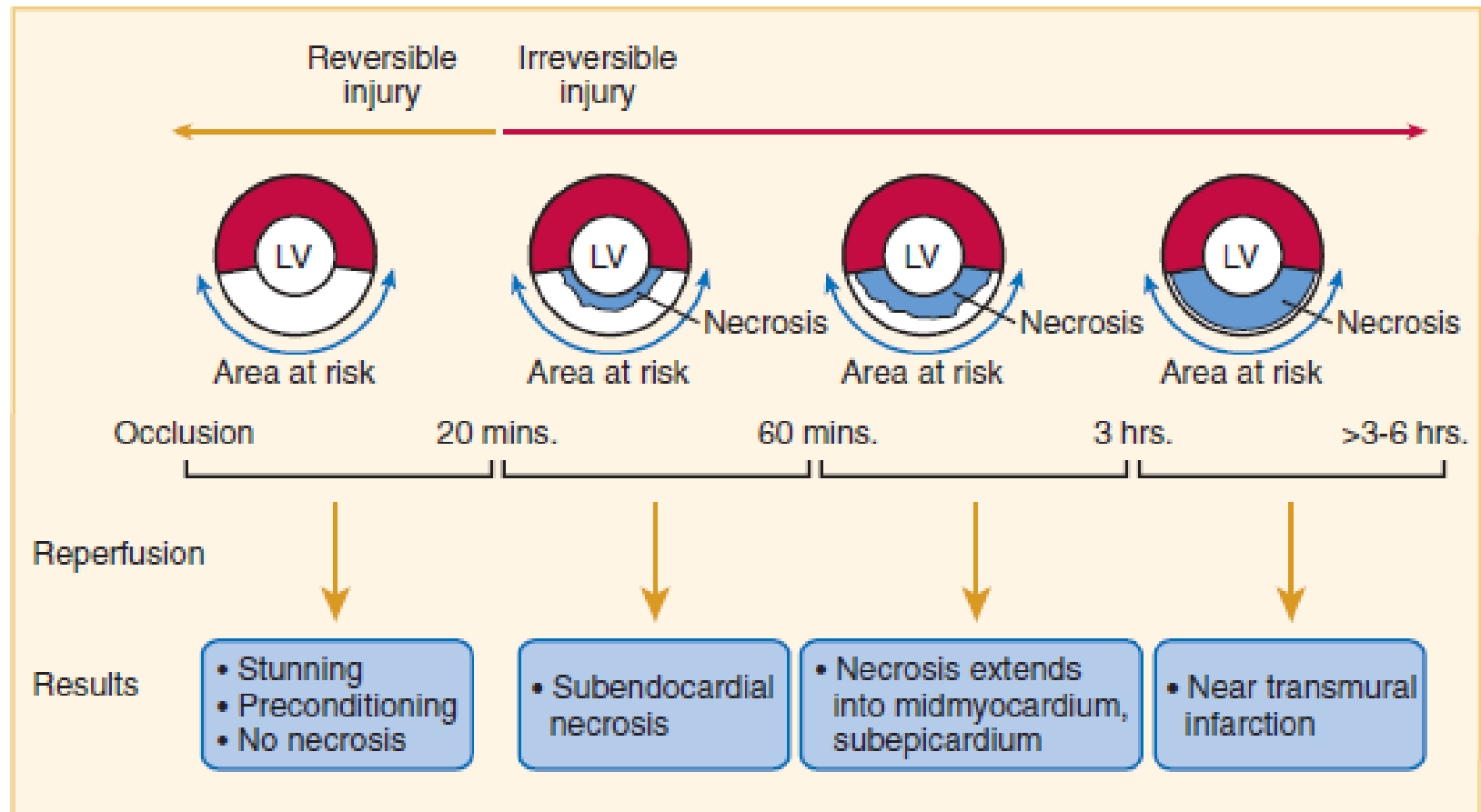
High sensitivity troponin assays



Sites of Occlusion with Zone of Necrosis



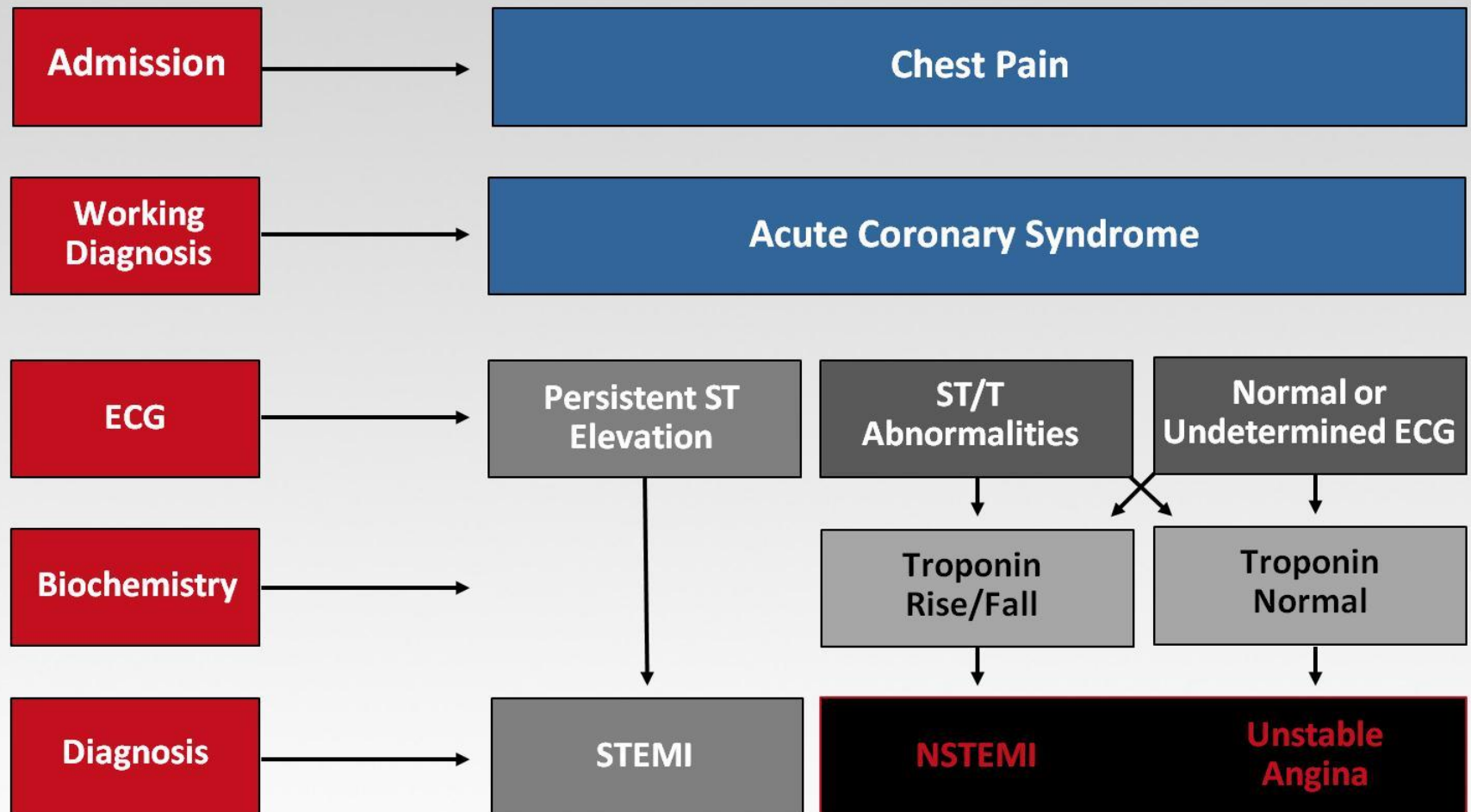
Time is Muscle!



Management

- Approach
- Risk Stratification
- STEMI
- NSTEMI/UA
- Pharmacotherapy

Evaluation of Chest Pain



Routine Measures

Drug	Dosing Regimen and Comments
Oxygen	Dosing: O₂ by nasal prongs or face mask to maintain SaO₂ ≥94% Comment: O₂ therapy should be used cautiously because it promotes coronary vasoconstriction and generates toxic O₂ metabolites.
Nitroglycerin	Dosing: For chest pain, 0.4 mg SL or by mouth spray, and repeat every 5 min x 2 if needed. For recurrent pain, CHF, or high BP, infuse at 5 µg/min and increase by 5–10 µg/min every 5 min to desired effect or to dose rate of 200 µg/min. Comment: Avoid in patients with RV infarction and for 24 hr following R_x for erectile dysfunction.
Morphine	Dosing: 2–4 mg IV with increments of 2–8 mg IV every 5–15 min as needed. Comment: Morphine-induced respiratory depression is uncommon in acute coronary syndromes.
Aspirin	Dosing: 162–325 mg (chewable form) initially, then 75–162 mg (enteric-coated tablets) daily. Comment: Initial dose should be chewed to enhance buccal absorption.
β-Blockers	Dosing: <i>Atenolol</i>: 10 mg IV, then 100 mg PO daily, or <i>Metoprolol</i>: 5 mg IV every 5 min for 3 doses, then 50 mg PO q 6 h for 48 hr, then 100 mg PO BID. Comment: Do NOT use in cocaine-induced chest pain or MI.

Risk Stratification- ACS

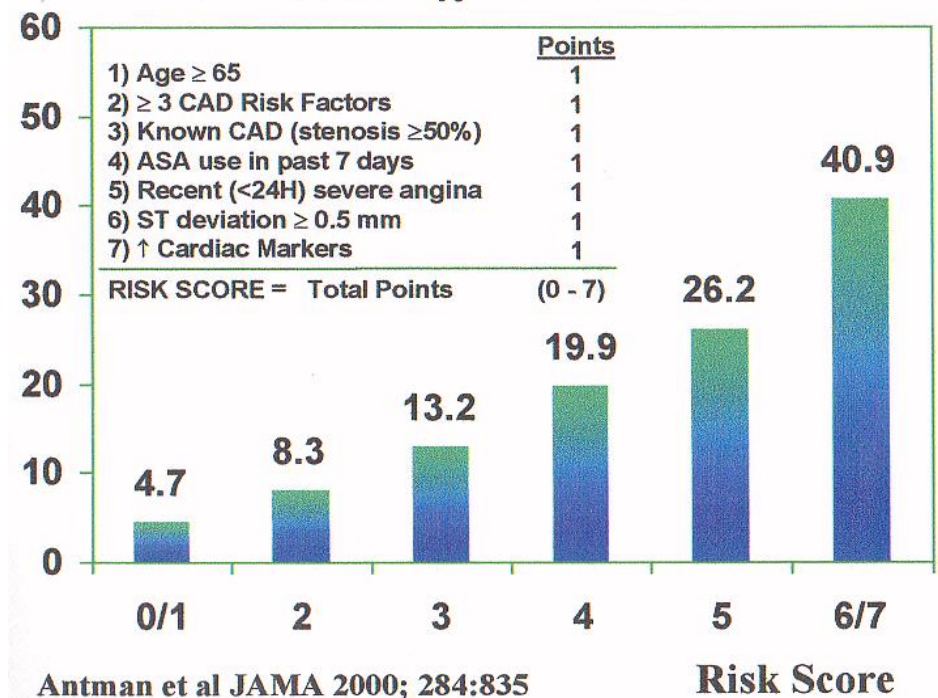
The screenshot shows the GRACE ACS Risk Model calculator. The header features the GRACE logo (Global Registry of Acute Coronary Events) and the title 'ACS Risk Model'. Below the header, there are two tabs: 'At Admission (in-hospital/to 6 months)' and 'At Discharge (to 6 months)'. The 'At Admission' tab is active. On the left, there are five input fields: Age (60-69), HR (70-89), SBP (160-199), Creat. (2.0-3.99), and CHF (I (no CHF)). On the right, there are three checkboxes: 'Cardiac arrest at admission' (unchecked), 'ST-segment deviation' (checked), and 'Elevated cardiac enzymes/markers' (checked). Below these, there is a table showing the probability of death and death or MI. At the bottom, there are three buttons: 'SI Units', 'Reset', and 'Display Score'.

Probability of	Death	Death or MI
In-hospital	2%	17%
To 6 months	7%	31%

TIMI Risk Score

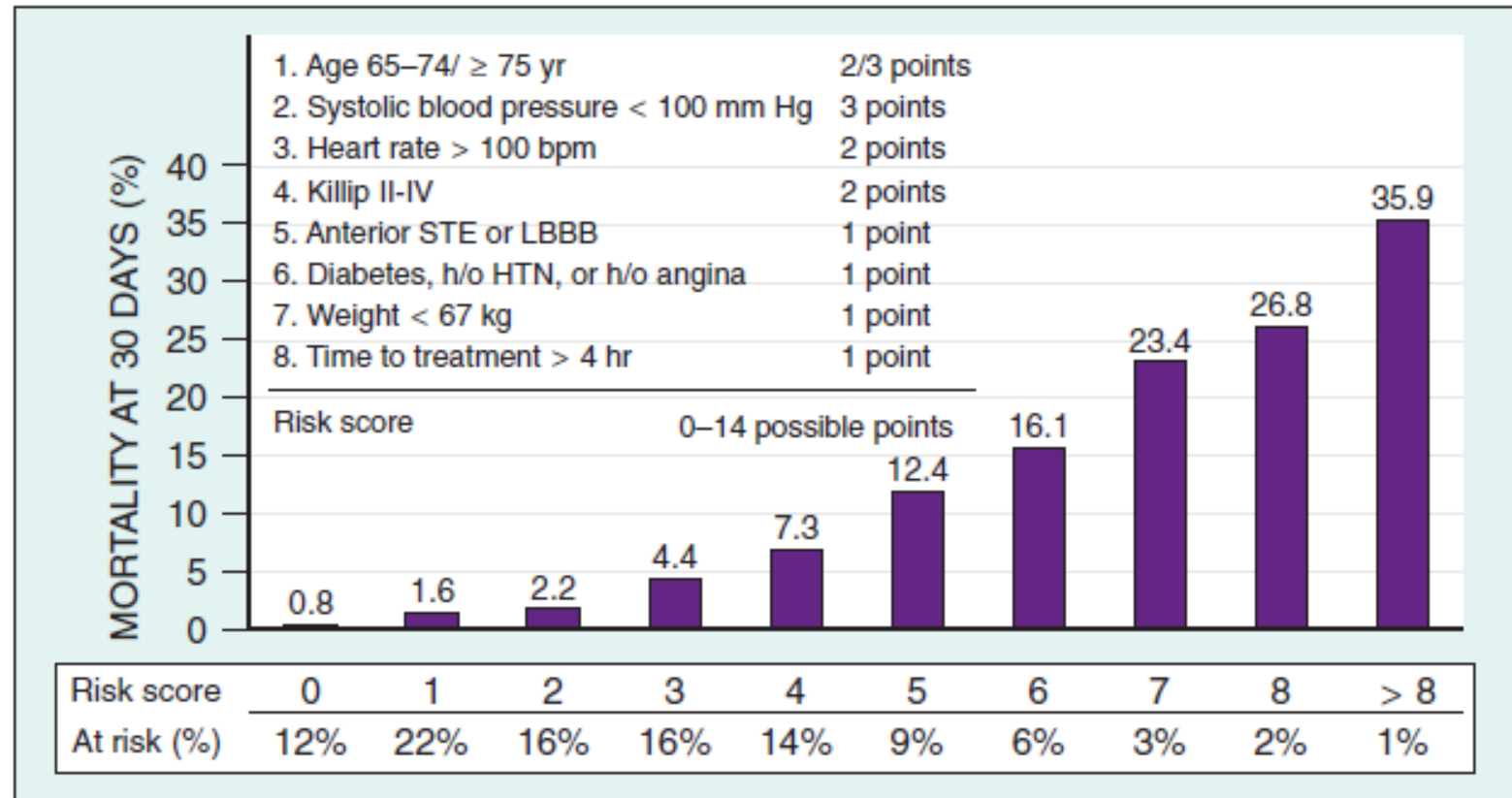
Predictor of adverse outcomes in those presenting with ACS (NSTEMI/UA)

- Age > 65
- More than 3 cardiac risk factors
- Prior angiographic coronary obstruction
- ST segment deviation
- More than 2 angina events within the previous 24 hours
- Use of aspirin within previous 7 days
- Elevated cardiac markers



TIMI Risk Score

Predictor of adverse outcomes in those presenting with ACS (STEMI)



STEMI- Reperfusion Therapy

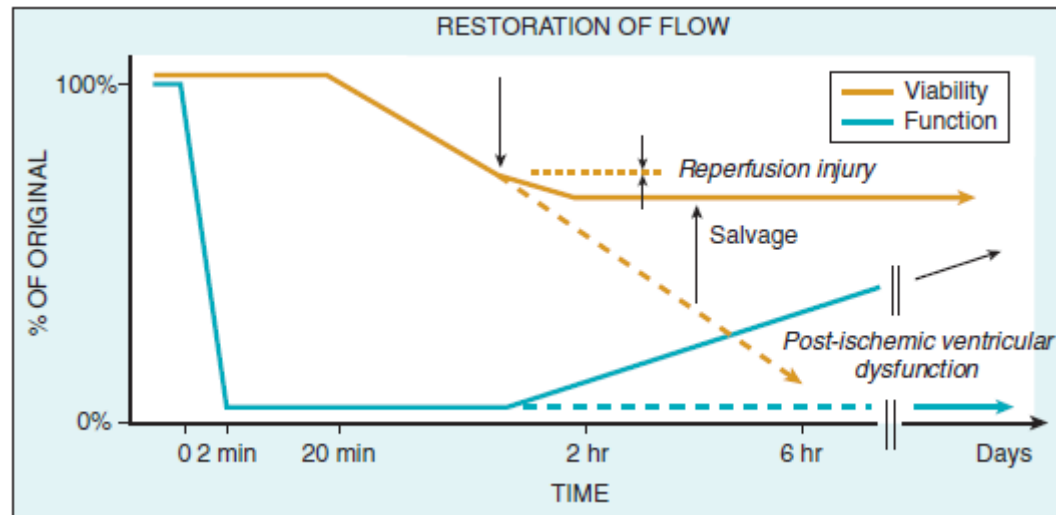
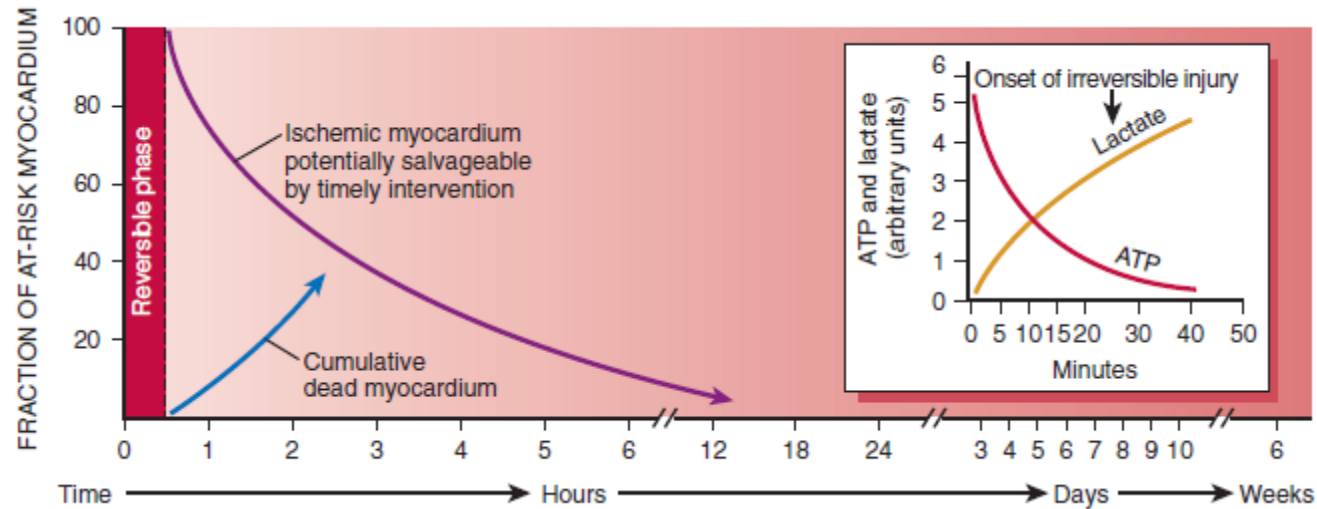


TABLE 55-4 Assessment of Reperfusion Options for STEMI Patients

Step 1: Assess time and risk

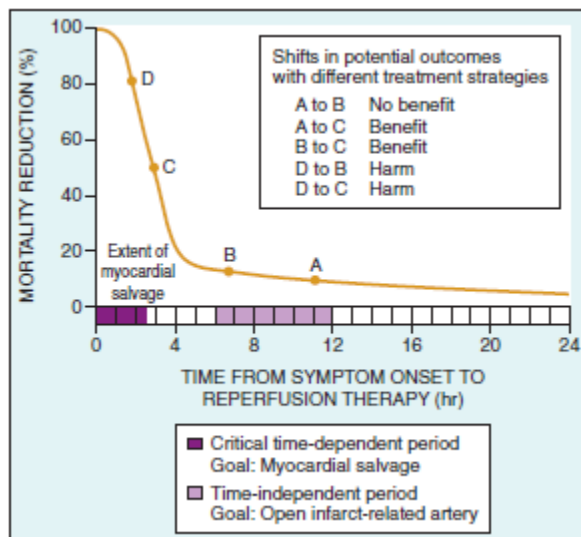
- Time since onset of symptoms
- Risk of STEMI
- Risk of fibrinolysis
- Time required for transport to a skilled PCI laboratory

Step 2: Determine if fibrinolysis or invasive strategy is preferred.

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

Fibrinolysis is generally preferred if:

- Early presentation (≤ 3 hr from symptom onset and delay to invasive strategy; see below)
- Invasive strategy is not an option:
 - Catheterization laboratory occupied or not available
 - Vascular access difficulties
 - Lack of access to a skilled PCI laboratory^{*†}
- Delay to invasive strategy:
 - Prolonged transport
 - (Door-to-balloon)–(door-to-needle) more than 1 hr^{‡§}
 - Medical contact-to-balloon or door-to-balloon more than 90 min

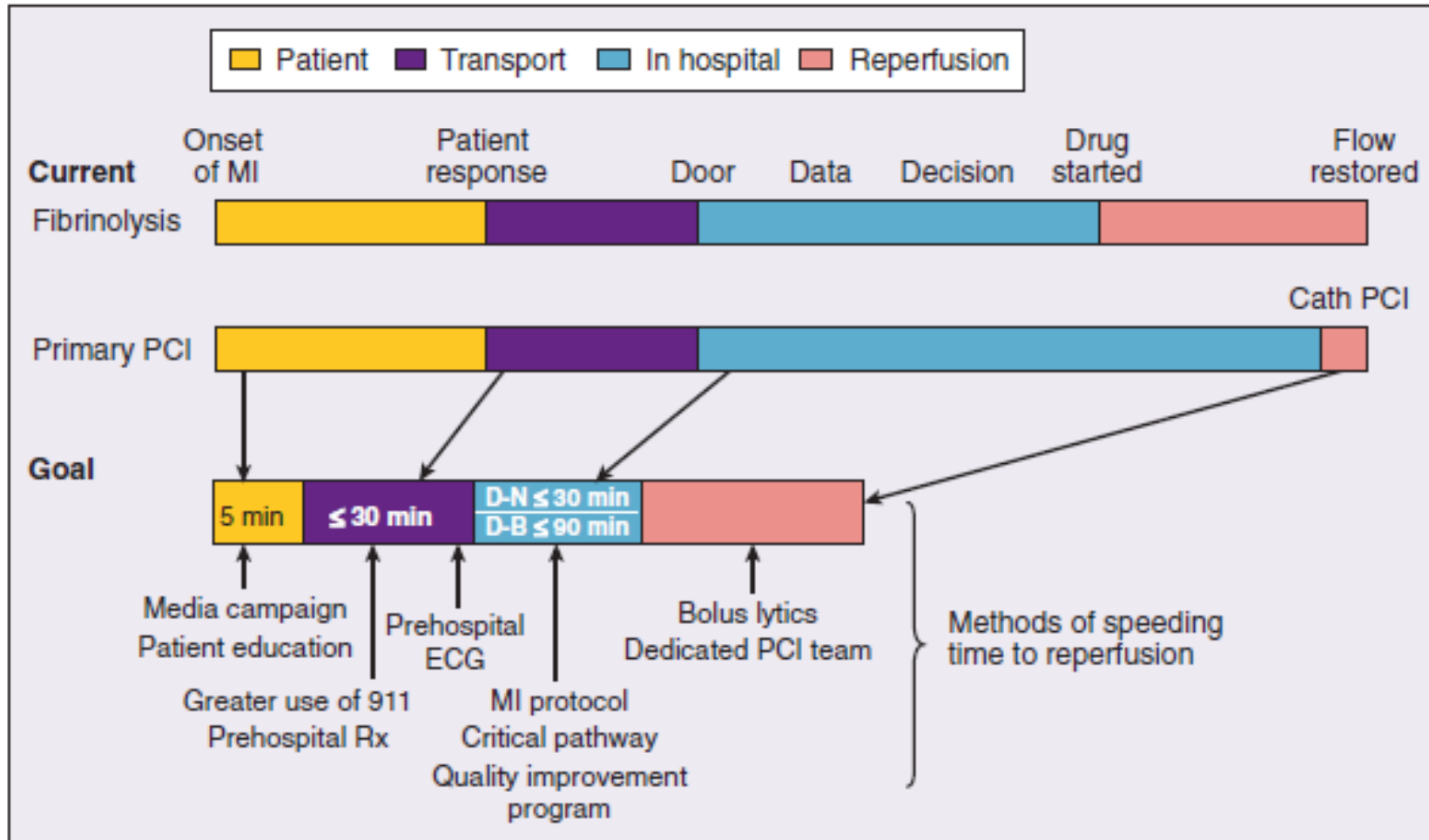


An invasive strategy is generally preferred if:

- Skilled PCI laboratory is available with surgical backup
 - Skilled PCI laboratory is available, defined by^{†‡}
 - Medical contact-to-balloon or door-to-balloon less than 90 min
 - (Door-to-balloon)–(door-to-needle) less than 1 hr[‡]
- High risk from STEMI
 - Cardiogenic shock
 - Killip class ≥ 3
- Contraindications to fibrinolysis, including increased risk of bleeding and ICH
- Late presentation
 - Symptom onset was more than 3 hr ago
- Diagnosis of STEMI is in doubt

Each 30-minute delay from symptom onset increases relative risk of 1 year-mortality by 8%

STEMI- System of Care



Contraindications to Fibrinolysis

Absolute

- Any prior ICH
- Known structural cerebral vascular lesion (eg, arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 mo EXCEPT acute ischemic stroke within 4.5 h
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head or facial trauma within 3 mo
- Intracranial or intraspinal surgery within 2 mo
- Severe uncontrolled hypertension (unresponsive to emergency therapy)
- For streptokinase, prior treatment within the previous 6 mo

Relative

- History of chronic, severe, poorly controlled hypertension
- Significant hypertension on presentation (SBP 180 mm Hg or DBP 110 mm Hg)
- History of prior ischemic stroke 3 mo
- Dementia
- Known intracranial pathology not covered in absolute contraindications
- Traumatic or prolonged (10 min) CPR
- Major surgery (3 wk)
- Recent (within 2 to 4 wk) internal bleeding
- Noncompressible vascular punctures
- Pregnancy
- Active peptic ulcer
- Oral anticoagulant therapy

Reperfusion Strategies

TABLE 55-6 Comparison of Approved Fibrinolytic Agents*

PARAMETER	STREPTOKINASE	ALTEPLASE	RETEPLASE	TNK T-PA
Dose	1.5 MU in 30-60 min	Up to 100 mg in 90 min (based on weight)	10 U × 2 (30 min apart) each over 2 min	30-50 mg based on weight
Bolus administration	No	No	Yes	Yes
Antigenic				
Allergic reactions (hypotension most common)	Yes	No	No	No
Systemic fibrinogen depletion	Marked	Mild	Moderate	Minimal
90-min patency rates (%)	≈50	≈75	≈75	≈75 [†]
TIMI grade 3 flow (%)	32	54	60	63

TABLE 56G-7 ACC/AHA Appropriateness Ratings for Type of Revascularization

	CABG			PCI		
	NO DIABETES AND NORMAL LVEF	DIABETES	DEPRESSED LVEF	NO DIABETES AND NORMAL LVEF	DIABETES	DEPRESSED LVEF
Two-vessel coronary artery disease with proximal LAD stenosis	A	A	A	A	A	A
Three-vessel coronary artery disease	A	A	A	U	U	U
Isolated left main stenosis	A	A	A	I	I	I
Left main stenosis and additional coronary artery disease	A	A	A	I	I	I

A = appropriate; I = inappropriate; U = uncertain; CABG = coronary artery bypass graft; LAD = left anterior descending coronary artery; LVEF = left ventricular ejection fraction; PCI = percutaneous coronary intervention.

Table 3. Adjunctive Antithrombotic Therapy to Support Reperfusion With Primary PCI

	COR	LOE
Antiplatelet therapy		
Aspirin		
• 162- to 325-mg load before procedure	I	B
• 81- to 325-mg daily maintenance dose (indefinite)*	I	A
• 81 mg daily is the preferred maintenance dose*	IIa	B
P2Y₁₂ inhibitors		
Loading doses		
• Clopidogrel: 600 mg as early as possible or at time of PCI	I	B
• Prasugrel: 60 mg as early as possible or at time of PCI	I	B
• Ticagrelor: 180 mg as early as possible or at time of PCI	I	B
Maintenance doses and duration of therapy		
<i>DES placed: Continue therapy for 1 y with:</i>		
• Clopidogrel: 75 mg daily	I	B
• Prasugrel: 10 mg daily	I	B
• Ticagrelor: 90 mg twice a day*	I	B
<i>BMS† placed: Continue therapy for 1 y with:</i>		
• Clopidogrel: 75 mg daily	I	B
• Prasugrel: 10 mg daily	I	B
• Ticagrelor: 90 mg twice a day*	I	B
<i>DES placed:</i>		
• Clopidogrel, prasugrel, or ticagrelor* continued beyond 1 y	IIb	C
• Patients with STEMI with prior stroke or TIA: prasugrel	III: Harm	B
IV GP IIb/IIIa receptor antagonists in conjunction with UFH or bivalirudin in selected patients		
• Abciximab: 0.25-mg/kg IV bolus, then 0.125 mcg/kg/min (maximum 10 mcg/min)	IIa	A
• Tirofiban: (high-bolus dose): 25-mcg/kg IV bolus, then 0.15 mcg/kg/min	IIa	B
• In patients with CrCl <30 mL/min, reduce infusion by 50%		
• Eptifibatide: (double bolus): 180-mcg/kg IV bolus, then 2 mcg/kg/min; a second 180-mcg/kg bolus is administered 10 min after the first bolus	IIa	B
• In patients with CrCl <50 mL/min, reduce infusion by 50%		
• Avoid in patients on hemodialysis		
• Pre-catheterization laboratory administration of IV GP IIb/IIIa receptor antagonist	IIb	B
• Intracoronary abciximab 0.25-mg/kg bolus	IIb	B
Anticoagulant therapy		
• UFH:	I	C
• With GP IIb/IIIa receptor antagonist planned: 50- to 70-U/kg IV bolus to achieve therapeutic ACT‡		
• With no GP IIb/IIIa receptor antagonist planned: 70- to 100-U/kg bolus to achieve therapeutic ACT§	I	C
• Bivalirudin: 0.75-mg/kg IV bolus, then 1.75–mg/kg/h infusion with or without prior treatment with UFH. An additional bolus of 0.3 mg/kg may be given if needed.	I	B
• Reduce infusion to 1 mg/kg/h with estimated CrCl <30 mL/min		
• Preferred over UFH with GP IIb/IIIa receptor antagonist in patients at high risk of bleeding	IIa	B
• Fondaparinux: not recommended as sole anticoagulant for primary PCI	III: Harm	B

Table 7. Adjunctive Antithrombotic Therapy to Support Reperfusion With Fibrinolytic Therapy

	COR	LOE
Antiplatelet therapy		
<i>Aspirin</i>		
• 162- to 325-mg loading dose	I	A
• 81- to 325-mg daily maintenance dose (indefinite)	I	A
• 81 mg daily is the preferred maintenance dose	IIa	B
<i>P2Y₁₂ receptor inhibitors</i>		
• Clopidogrel:	I	A
• Age ≤ 75 y: 300-mg loading dose		
• Followed by 75 mg daily for at least 14 d and up to 1 y in absence of bleeding	I	A (14 d) C (up to 1 y)
• Age > 75 y: no loading dose, give 75 mg	I	A
• Followed by 75 mg daily for at least 14 d and up to 1 y in absence of bleeding	I	A (14 d) C (up to 1 y)
Anticoagulant therapy		
• UFH:	I	C
• Weight-based IV bolus and infusion adjusted to obtain aPTT of 1.5 to 2.0 times control for 48 h or until revascularization. IV bolus of 60 U/kg (maximum 4000 U) followed by an infusion of 12 U/kg/h (maximum 1000 U) initially, adjusted to maintain aPTT at 1.5 to 2.0 times control (approximately 50 to 70 s) for 48 h or until revascularization		
• Enoxaparin:	I	A
• If age < 75 y: 30-mg IV bolus, followed in 15 min by 1 mg/kg subcutaneously every 12 h (maximum 100 mg for the first 2 doses)		
• If age ≥ 75 y: no bolus, 0.75 mg/kg subcutaneously every 12 h (maximum 75 mg for the first 2 doses)		
• Regardless of age, if CrCl < 30 mL/min: 1 mg/kg subcutaneously every 24 h		
• Duration: For the index hospitalization, up to 8 d or until revascularization		
• Fondaparinux:	I	B
• Initial dose 2.5 mg IV, then 2.5 mg subcutaneously daily starting the following day, for the index hospitalization up to 8 d or until revascularization		
• Contraindicated if CrCl < 30 mL/min		

Pharmacoinvasive therapy

Table 9. Indications for Coronary Angiography in Patients Who Were Managed With Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

	COR	LOE	References
Cardiogenic shock or acute severe HF that develops after initial presentation	I	B	(215,354, 372,373)
Intermediate- or high-risk findings on predischarge noninvasive ischemia testing	I	B	(232,233)
Spontaneous or easily provoked myocardial ischemia	I	C	N/A
Failed reperfusion or reocclusion after fibrinolytic therapy	IIa	B	(346,355–357)
Stable* patients after successful fibrinolysis, before discharge and ideally between 3 and 24 h	IIa	B	(358–363,374)

*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

Table 10. Indications for PCI of an Infarct Artery in Patients Who Were Managed With Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

	COR	LOE	References
Cardiogenic shock or acute severe HF	I	B	(354)
Intermediate- or high-risk findings on predischarge noninvasive ischemia testing	I	C	(232,233)
Spontaneous or easily provoked myocardial ischemia	I	C	N/A
Patients with evidence of failed reperfusion or reocclusion after fibrinolytic therapy (as soon as possible)	IIa	B	(344–347)
Stable* patients after successful fibrinolysis, ideally between 3 and 24 h	IIa	B	(358–363)
Stable* patients >24 h after successful fibrinolysis	IIb	B	(213,232,233,366,374–378)
Delayed PCI of a totally occluded infarct artery >24 h after STEMI in stable patients	III: No Benefit	B	(213,376)

*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

Table 11. Adjunctive Antithrombotic Therapy to Support PCI After Fibrinolytic Therapy

	COR	LOE	References
Antiplatelet therapy			
Aspirin			
• 162- to 325-mg loading dose given with fibrinolytic agent (before PCI). (Section 5.1.4.1 and Table 7)	I	A	(308,330,331)
• 81- to 325-mg daily maintenance dose after PCI (indefinite)	I	A	(253,254,257,259,330,331)
• 81 mg daily is the preferred daily maintenance dose	IIa	B	(253,259,263,264)
P2Y₁₂ receptor inhibitors			
Loading doses			
<i>For patients who received a loading dose of clopidogrel with fibrinolytic therapy:</i>			
• Continue clopidogrel 75 mg daily without an additional loading dose	I	C	(260,262,330,331)
<i>For patients who have not received a loading dose of clopidogrel:</i>			
• If PCI is performed ≤24 h after fibrinolytic therapy: clopidogrel 300-mg loading dose before or at the time of PCI	I	C	N/A
• If PCI is performed >24 h after fibrinolytic therapy: clopidogrel 600-mg loading dose before or at the time of PCI	I	C	N/A
• If PCI is performed >24 h after treatment with a fibrin-specific agent or >48 h after a non-fibrin-specific agent: prasugrel 60 mg at the time of PCI	IIa	B	(260,262)
<i>For patients with prior stroke/TIA: prasugrel</i>	III: Harm	B	(260)
Maintenance doses and duration of therapy			
<i>DES placed: Continue therapy for at least 1 y with:</i>			
• Clopidogrel: 75 mg daily	I	C	(260,262,330,331)
• Prasugrel: 10 mg daily	IIa	B	(260,262)
<i>BMS* placed: Continue therapy for at least 30 d and up to 1 y with:</i>			
• Clopidogrel: 75 mg daily	I	C	(330,331)
• Prasugrel: 10 mg daily	IIa	B	(260,262)
Anticoagulant therapy			
• Continue UFH through PCI, administering additional IV boluses as needed to maintain therapeutic ACT depending on use of GP IIb/IIIa receptor antagonist†	I	C	N/A
• Continue enoxaparin through PCI:	I	B	(332,390)
• No additional drug if last dose was within previous 8 h			
• 0.3-mg/kg IV bolus if last dose was 8 to 12 h earlier			
• Fondaparinux:	III: Harm	C	(304)
• As sole anticoagulant for PCI			

*Balloon angioplasty without stent placement may be used in selected patients. It might be reasonable to provide P2Y₁₂ inhibitor therapy to patients with STEMI undergoing balloon angioplasty after fibrinolysis alone according to the recommendations listed for BMS. (Level of Evidence: C)

ACCF/AHA Recommendations for Initial Management of UA/NSTEMI

Diagnosis of UA/NSTEMI is Likely or Definite

ASA (Class I)

Select Management Strategy

Initial Conservative Strategy or Unknown

Initiate Anticoagulant Therapy (Class I)

Acceptable options include:

- Enoxaparin or UFH (class I)
- Fondaparinux (class I)
- Enoxaparin or fondaparinux preferred over UFH (class I)

Initiate Clopidogrel or Ticagrelor (Class I)

Invasive Strategy

Initiate Anticoagulant Therapy (Class I)

Acceptable options include:

- Enoxaparin or UFH (class I)
- Bivalirudin (class I)
- Fondaparinux (class I)

Precatheterization: Add Second Antiplatelet Agent (Class I)

- Clopidogrel or ticagrelor (class I)
- GP IIb/IIIa inhibitor (class I)
(IV eptifibatide or tirofiban preferred)

Next Step Per Triage Decision at Angiography

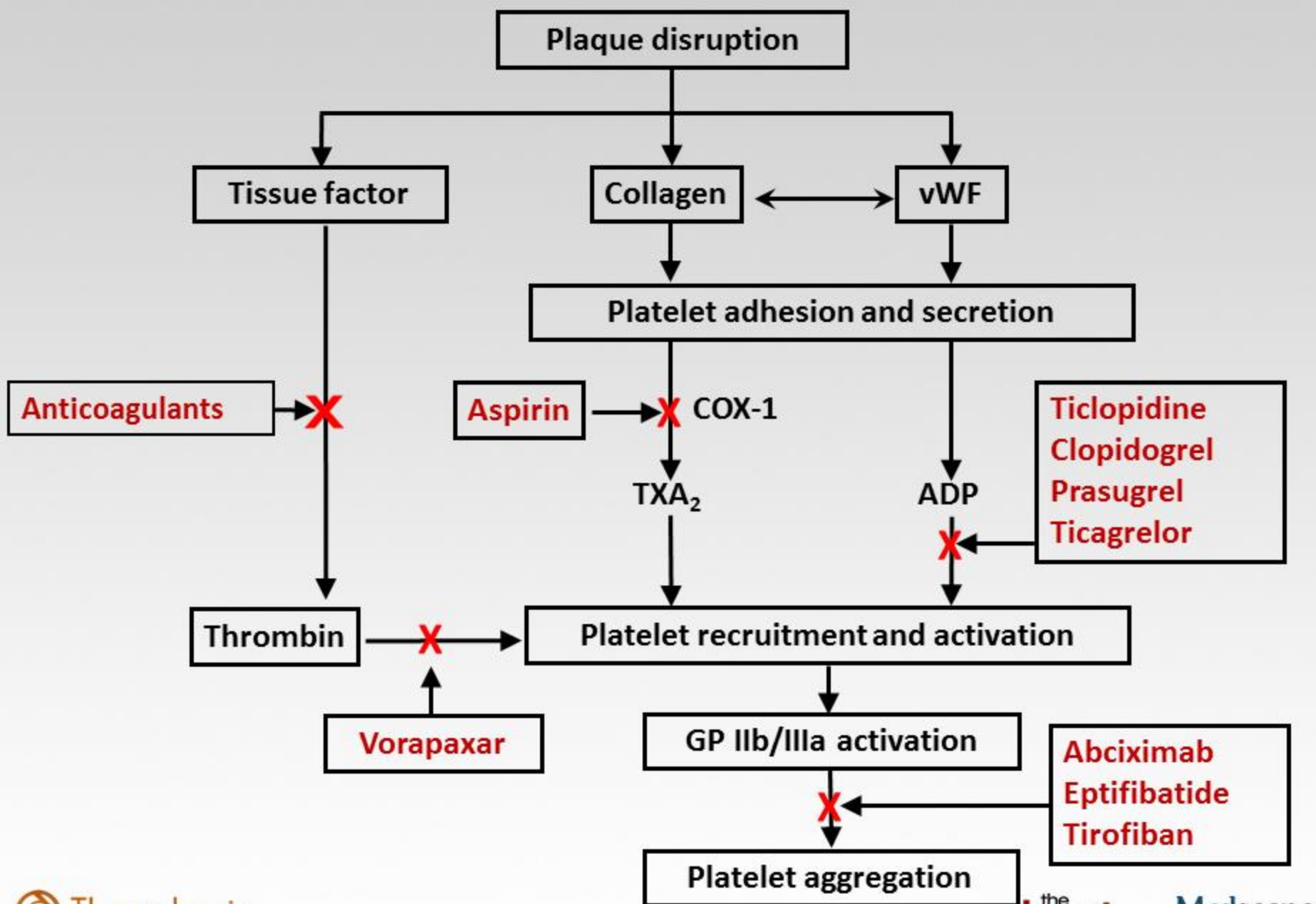
CABG
Maintenance ASA
(Class I)

PCI: Class I:

- Clopidogrel or ticagrelor (if not begun precatheterization) or
- Prasugrel or
- Selectively, a GP IIb/IIIa inhibitor (if not begun precatheterization)

Medical Therapy: D/C GP IIb/IIIa inhibitors if begun and give clopidogrel or ticagrelor per conservative strategy

Pathophysiology of Atherothrombosis and Its Treatment



Antiplatelets

Indicated for all ACS Patients

- **Aspirin**
 - 160-325mg chewed and swallowed/soluble
 - Irreversible blockage of platelet aggregation
 - Plaque stabilization and arrest of thrombus formation
 - Indicated for full spectrum of ACS'
 - 23% reduction in mortality for patients with STEMI



Anti-platelets

	Clopidogrel	Prasugrel	Ticagrelor	Cangrelor
Chemical class	Thienopyridine	Thienopyridine	Cyclopentyl-triazolopyrimidine	Stabilized ATP analogue
Administration	Oral	Oral	Oral	Intravenous
Dose	300–600 mg orally then 75 mg a day	60 mg orally then 10 mg a day	180 mg orally then 90 mg twice a day	30 µg/kg bolus and 4 µg/kg/min infusion
Dosing in CKD				
• Stage 3 (eGFR 30–59 mL/min/1.73m ²)	No dose adjustment	No dose adjustment	No dose adjustment	No dose adjustment
• Stage 4 (eGFR 15–29 mL/min/1.73m ²)	No dose adjustment	No dose adjustment	No dose adjustment	No dose adjustment
• Stage 5 (eGFR <15 mL/min/1.73m ²)	Use only for selected indications (e.g. stent thrombosis prevention)	Not recommended	Not recommended	No dose adjustment
Binding reversibility	Irreversible	Irreversible	Reversible	Reversible
Activation	Prodrug, with variable liver metabolism	Prodrug, with predictable liver metabolism	Active drug, with additional active metabolite	Active drug
Onset of loading dose effect^a	2–6 hours ^b	30 min ^b	30 min ^b	2 min
Duration of effect	3–10 days	7–10 days	3–5 days	1–2 hours
Withdrawal before surgery	5 days ^c	7 days ^c	5 days ^c	1 hour
Plasma half-life of active P2Y₁₂ inhibitor^d	30–60 min	30–60 min ^e	6–12 hours	5–10 min
Inhibition of adenosine reuptake	No	No	Yes	Yes ('inactive' metabolite only)

Anticoagulants

Table 11 Dosing of anticoagulants in patients with normal and impaired renal function

Drug	Recommendations		
	Normal renal function or stage 1–3 CKD (eGFR ≥ 30 mL/min/1.73m ²)	Stage 4 CKD (eGFR 15–29 mL/min/1.73m ²)	Stage 5 CKD (eGFR < 15 mL/min/1.73m ²)
Unfractionated heparin	• Prior to coronary angiography: 60–70 IU/kg i.v. (max 5000 IU) and infusion (12–15 IU/kg/h) (max 1000 IU/h), target aPTT 1.5–2.5x control • During PCI: 70–100 IU/kg i.v. (50–70 IU/kg if concomitant with GPIIb/IIIa inhibitors)	No dose adjustment	No dose adjustment
Enoxaparin	1 mg/kg s.c. twice a day	1 mg/kg s.c. once a day	Not recommended
Fondaparinux	2.5 mg s.c. once a day	Not recommended if eGFR < 20 mL/min/1.73m ²	Not recommended
Bivalirudin	Bolus 0.75 mg/kg i.v., infusion 1.75 mg/kg/h	No adjustment of bolus, reduce infusion rate to 1 mg/kg/h	On dialysis, no adjustment of bolus, reduce infusion rate to 0.25 mg/kg/h

aPTT = activation partial thromboplastin time; CKD = chronic kidney disease; eGFR = estimated glomerular filtration rate; IU = international units; i.v. = intravenous; kg = kilograms bodyweight; s.c. = subcutaneous.

Glycoprotein IIb/IIIa Inhibitors

- Inhibit platelet aggregation
- Greatest benefit, in conjunction with Aspirin and Heparin, for those with planned intervention
- May also be beneficial with other high risk features w/out planned intervention

	ABCIXIMAB	EPTIFIBATIDE	TIROFIBAN
Type	Antibody fragment	Peptide	Non-Peptide
Size (daltons)	≈50,000	≈800	≈500
Platelet-bound half-life	Long (h)	Short (sec)	Short (sec)
Plasma half-life	Short (min)	Extended (2 h)	Extended (2 h)
Immunogenic	Yes*	No	No
Return of platelet function [†] (h)	12	≈4	≈4
Reversible with platelet transfusion	Yes	No	No

*Induction of antibody response seen in 5%–6% of patients tested; [†]50% reduction in antiplatelet effect without transfusion

Table 10 Dosing of glycoprotein IIb/IIIa inhibitors in patients with normal and impaired renal function

Drug	Recommendations			
	Normal renal function or stage 1–2 CKD (eGFR ≥60 mL/min/1.73m ²)	Stage 3 CKD (eGFR 30–59 mL/min/1.73m ²)	Stage 4 CKD (eGFR 15–29 mL/min/1.73m ²)	Stage 5 CKD (eGFR <15 mL/min/1.73m ²)
Eptifibatide	Bolus 180 µg/kg i.v., Infusion 2 µg/kg/min	No adjustment of bolus, reduce infusion rate to 1 µg/kg/min if eGFR <50 mL/min/1.73m ²	Not recommended	Not recommended
Tirofiban	Bolus 25 µg/kg or 10 µg/kg i.v., Infusion 0.15 µg/kg/min	No dose adjustment	No adjustment of bolus, reduce infusion to 0.05 µg/kg/min	Not recommended
Abciximab	Bolus 0.25 mg/kg i.v., Infusion 0.125 µg/kg/min (max. 10 µg/min)	No specific recommendations for the use of abciximab, or for dose adjustment in the case of renal failure. Careful evaluation of haemorrhagic risk is needed.		

CKD = chronic kidney disease; eGFR = estimated glomerular filtration rate; i.v. = intravenous; kg = kilograms bodyweight.

Table 12. Selected Routine Medical Therapies

Therapy	Indications	Dose/Administration	Avoid/Caution
Beta-Receptor Antagonists	- Oral: All patients without contraindication - IV: Patients with refractory hypertension or ongoing ischemia without contraindication	Individualize: - Metoprolol tartrate 25 to 50 mg every 6 to 12 h orally, then transition over next 2 to 3 d to twice-daily dosing of metoprolol tartrate or to daily metoprolol succinate; titrate to daily dose of 200 mg as tolerated - Carvedilol 6.25 mg twice daily, titrate to 25 mg twice daily as tolerated - Metoprolol tartrate IV 5 mg every 5 min as tolerated up to 3 doses; titrate to heart rate and BP	- Signs of HF - Low output state - Increased risk of cardiogenic shock - Prolonged first-degree or high-grade AV block - Reactive airways disease
ACE Inhibitors	- For patients with anterior infarction, post-MI LV systolic dysfunction (EF $\leq$ 0.40) or HF - May be given routinely to all patients without contraindication	Individualize: - Lisinopril 2.5 to 5 mg/d to start; titrate to 10 mg/d or higher as tolerated - Captopril 6.25 to 12.5 mg 3 times/d to start; titrate to 25 to 50 mg 3 times/d as tolerated - Ramipril 2.5 mg twice daily to start; titrate to 5 mg twice daily as tolerated - Trandolapril test dose 0.5 mg; titrate up to 4 mg daily as tolerated	- Hypotension - Renal failure - Hyperkalemia
ARB	For patients intolerant of ACE inhibitors	Valsartan 20 mg twice daily to start; titrate to 160 mg twice daily as tolerated	- Hypotension - Renal failure - Hyperkalemia
Statins	All patients without contraindications	High-dose atorvastatin 80 mg daily	- Caution with drugs metabolized via CYP3A4, fibrates - Monitor for myopathy, hepatic toxicity - Combine with diet and lifestyle therapies - Adjust dose as dictated by targets for LDL cholesterol and non-HDL cholesterol reduction
Nitroglycerin	- Ongoing chest pain - Hypertension and HF	0.4 mg sublingual every 5 min up to 3 doses as BP allows - IV dosing to begin at 10 mcg/min; titrate to desired BP effect	- Avoid in suspected RV infarction - Avoid with SBP <90 mm Hg or if SBP >30 mm Hg below baseline - Avoid if recent (24 to 48 h) use of 5'-phosphodiesterase inhibitors
Oxygen	- Clinically significant hypoxemia (oxygen saturation <90%) - HF - Dyspnea	2 to 4 L/min via nasal cannula - Increase rate or change to face mask as needed	Caution with chronic obstructive pulmonary disease and CO₂ retention
Morphine	- Pain - Anxiety - Pulmonary edema	4 to 8 mg IV initially, with lower doses in elderly 2 to 8 mg IV every 5 to 15 min if needed	- Lethargic or moribund patient - Hypotension - Bradycardia - Known hypersensitivity

ACE indicates angiotensin-converting enzyme; ARB, angiotensin receptor blocker; AV, atrioventricular; BP, blood pressure; CO₂, carbon dioxide; EF, ejection fraction; HDL, high-density lipoprotein; HF, heart failure; IV, intravenous; LDL, low-density lipoprotein; LV, left ventricular; MI, myocardial infarction; RV, right ventricular; and SBP, systolic blood pressure.

Critical Care in ACS

- Hemodynamic Assessment
- Left Ventricular Failure
- Cardiogenic Shock
- Right Ventricular Infarction
- Mechanical Complications
- Electrical Complications
- Others

Hemodynamic Assessment

TABLE 55-9 Indications for Hemodynamic Monitoring in Patients with STEMI

Management of complicated acute myocardial infarction
 Hypovolemia versus cardiogenic shock
 Ventricular septal rupture versus acute mitral regurgitation
 Severe left ventricular failure
 Right ventricular failure
 Refractory ventricular tachycardia
 Differentiating severe pulmonary disease from left ventricular failure
 Assessment of cardiac tamponade
 Assessment of therapy in *selected* individuals
 Afterload reduction in patients with severe left ventricular failure
 Inotropic agent therapy
 Beta blocker therapy
 Temporary pacing (ventricular versus atrioventricular)
 Intra-aortic balloon counterpulsation
 Mechanical ventilation

From Gore JM, Zwernert PL: Hemodynamic monitoring of acute myocardial infarction. In Francis GS, Alpert JS (eds): Modern Coronary Care. Boston, Little, Brown, 1990, p 138.

TABLE 55-10 Hemodynamic Classifications of Patients with Acute Myocardial Infarction

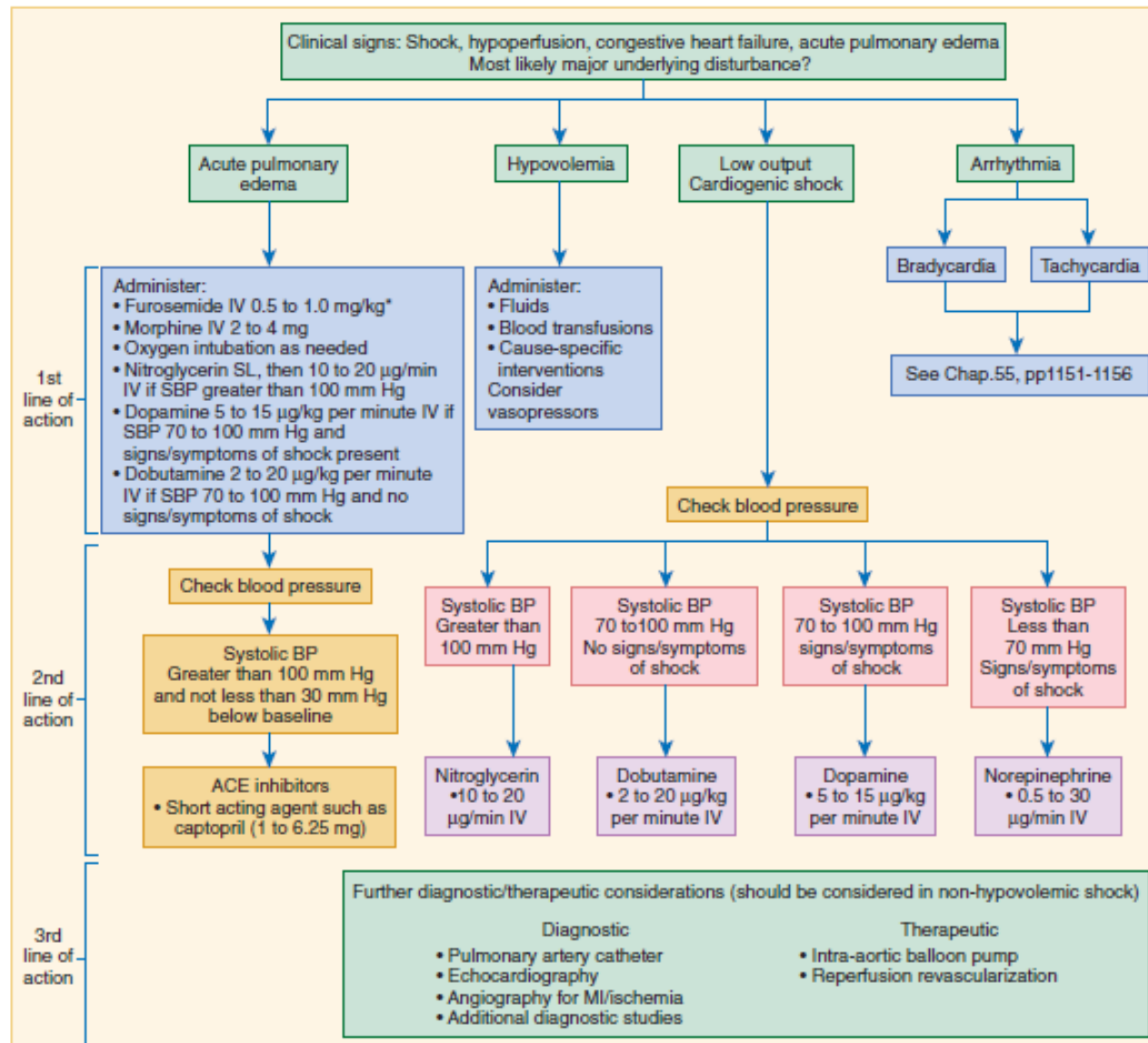
Based on Clinical Examination*		Based on Invasive Monitoring†	
CLASS	DEFINITION	SUBSET	DEFINITION
I	Rales and S ₃ absent	I	Normal hemodynamics; PCWP < 18 mm Hg, CI > 2.2
II	Crackles, S ₃ gallop, elevated jugular venous pressure	II	Pulmonary congestion; PCWP > 18 mm Hg, CI > 2.2
III	Frank pulmonary edema	III	Peripheral hypoperfusion; PCWP < 18 mm Hg, CI < 2.2
IV	Shock	IV	Pulmonary congestion and peripheral hypoperfusion; PCWP > 18 mm Hg, CI < 2.2

CI = cardiac index; PCWP = pulmonary capillary wedge pressure.

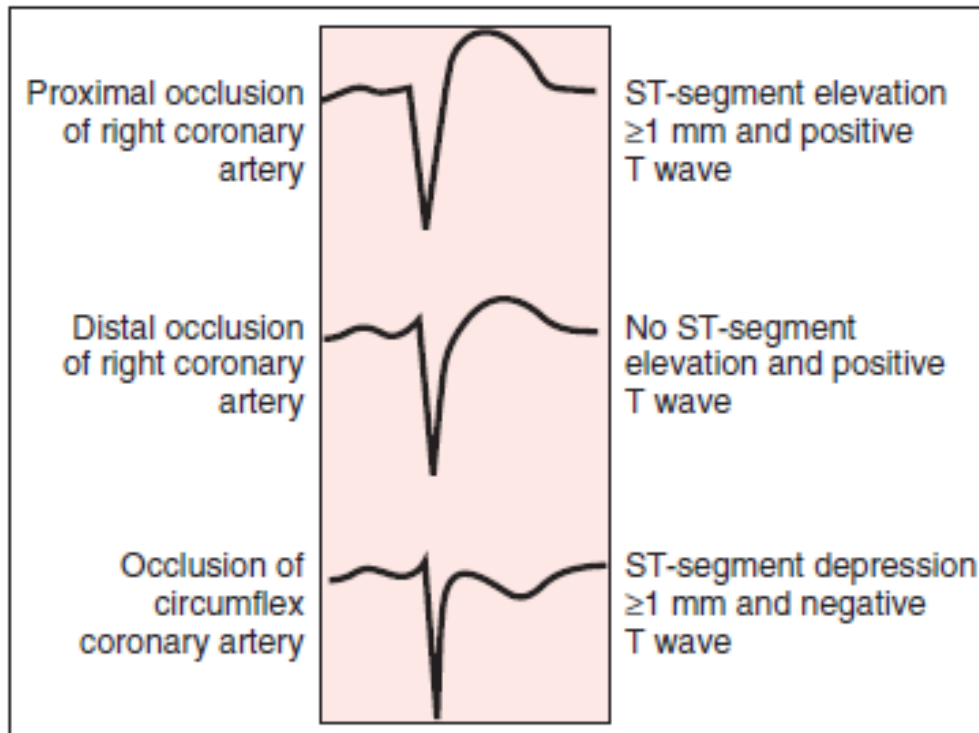
*Modified from Killip T, Kimball J: Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. Am J Cardiol 20:457, 1967.

†Modified from Forrester J, Diamond G, Chatterjee K, et al: Medical therapy of acute myocardial infarction by the application of hemodynamic subsets. N Engl J Med 295:1356, 1976.

Pump Failure/ Cardiogenic Shock



Right Ventricular Infarction



Clinical findings:

Shock with clear lungs, elevated JVP
Kussmaul sign

Hemodynamics:

Increased RA pressure (γ descent)
Square root sign in RV tracing

ECG:

ST elevation in right-sided leads

Echo:

Depressed RV function

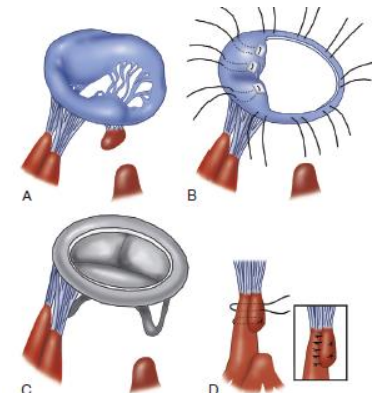
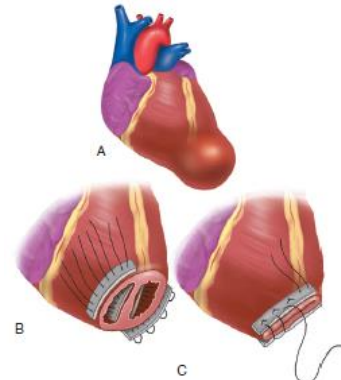
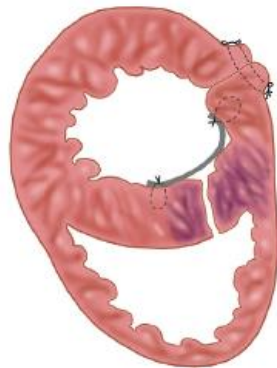
Management:

Maintain RV preload
Lower RV afterload (PA---PCW)
Restore AV synchrony
Inotropic support
Reperfusion

Mechanical Complications

TABLE 55-12 Features of Ventricular Septal Rupture (VSR), Rupture of Ventricular Free Wall, and Papillary Muscle Rupture

FEATURE	VSR	RUPTURE OF VENTRICULAR FREE WALL	PAPILLARY MUSCLE RUPTURE
Incidence	1%-3% without reperfusion therapy, 0.2%-0.34% with fibrinolytic therapy, 3.9% among patients with cardiogenic shock	0.8%-6.2%; fibrinolytic therapy does not reduce risk; primary PTCA seems to reduce risk	About 1% (posteromedial more frequent than anterolateral papillary muscle)
Time course	Bimodal peak; within 24 hr and 3-5 days; range, 1-14 days	Bimodal peak; within 24 hr and 3-5 days; range, 1-14 days	Bimodal peak; within 24 hr and 3-5 days; range, 1-14 days
Clinical manifestations	Chest pain, shortness of breath, hypotension	Anginal, pleuritic, or pericardial chest pain, syncope, hypotension, arrhythmia, nausea, restlessness, sudden death	Abrupt onset of shortness of breath and pulmonary edema; hypotension
Physical findings	Harsh holosystolic murmur, thrill (+), S ₃ , accentuated second heart sound, pulmonary edema, RV and LV failure, cardiogenic shock	Jugular venous distention (29% of patients), pulsus paradoxus (47%), electromechanical dissociation, cardiogenic shock	A soft murmur in some cases, no thrill, variable signs of RV overload, severe pulmonary edema, cardiogenic shock
Echocardiographic findings	VSR, left-to-right shunt on color flow Doppler echocardiography through the ventricular septum, pattern of RV overload	>5 mm pericardial effusion not visualized in all cases; layered, high-acoustic echoes within the pericardium (blood clot); direct visualization of tear; signs of tamponade	Hypercontractile LV, torn papillary muscle or chordae tendineae, flail leaflet, severe MR on color flow Doppler echocardiography
Right-heart catheterization	Increase in oxygen saturation from the RA to RV, large v waves	Ventriculography insensitive, classic signs of tamponade not always present (equalization of diastolic pressures among the cardiac chambers)	No increase in oxygen saturation from the RA to RV, large v waves,* very high pulmonary-capillary wedge pressures



Surgical Options

1. VSD Repair
2. Aneurysmal Repair
3. MVR

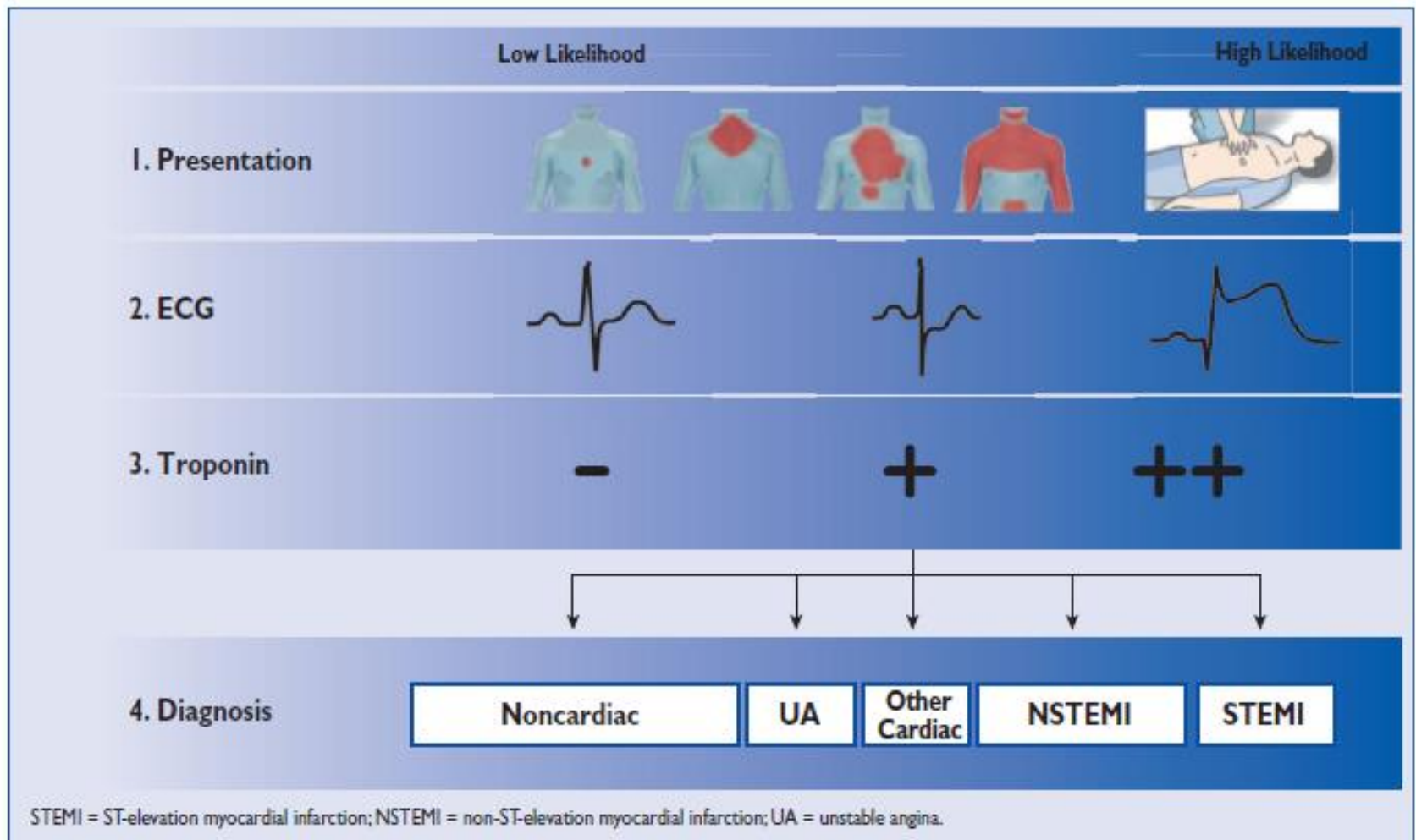
Electrical Complications

TABLE 55-13 Management of Cardiac Arrhythmias During Acute Myocardial Infarction			
CATEGORY	ARRHYTHMIA	OBJECTIVE OF TREATMENT	THERAPEUTIC OPTIONS
Electrical instability	Ventricular premature beats	Correction of electrolyte deficits and increased sympathetic tone	Potassium and magnesium solutions, beta blocker
	Ventricular tachycardia	Prophylaxis against ventricular fibrillation, restoration of hemodynamic stability	Antiarrhythmic agents; cardioversion/defibrillation
	Ventricular fibrillation	Urgent reversion to sinus rhythm	Defibrillation; bretylium tosylate
	Accelerated idioventricular rhythm	Observation unless hemodynamic function is compromised	Increase sinus rate (atropine, atrial pacing); antiarrhythmic agents
	Nonparoxysmal atrioventricular junctional tachycardia	Search for precipitating causes (e.g., digitalis intoxication); suppress arrhythmia only if hemodynamic function is compromised	Atrial overdrive pacing; antiarrhythmic agents; cardioversion relatively contraindicated if digitalis intoxication present
Pump failure, excessive sympathetic stimulation	Sinus tachycardia	Reduce heart rate to diminish myocardial oxygen demands	Antipyretics; analgesics; consider beta blocker unless congestive heart failure present; treat latter if present with anticongestive measures (diuretics, afterload reduction)
	Atrial fibrillation and/or atrial flutter	Reduce ventricular rate; restore sinus rhythm	Verapamil, digitalis glycosides; anticongestive measures (diuretics, afterload reduction); cardioversion; rapid atrial pacing (for atrial flutter)
	Paroxysmal supraventricular tachycardia	Reduce ventricular rate; restore sinus rhythm	Vagal maneuvers; verapamil, cardiac glycosides, beta-adrenergic blockers; cardioversion; rapid atrial pacing
Bradycardias and conduction disturbances	Sinus bradycardia	Acceleration of heart rate only if hemodynamic function is compromised	Atropine; atrial pacing
	Junctional escape rhythm	Acceleration of sinus rate only if loss of atrial "kick" causes hemodynamic compromise	Atropine; atrial pacing
	Atrioventricular block and intraventricular block		Insertion of pacemaker

Others

- Pericarditis
including Dressler's Syndrome
- Recurrent Ischemia
- Left Ventricular Thrombus and Arterial Embolization
- Acute Kidney Injury
- Hyperglycemia
- Misc

Summary



Summary

TABLE 56G-3 ACC/AHA Guideline Recommendations for Selection of Initial Treatment Strategy: Invasive Versus Conservative Strategy	
PREFERRED STRATEGY	PATIENT CHARACTERISTICS
Invasive	Recurrent angina or ischemia at rest or with low-level activities, despite intensive medical therapy Elevated cardiac biomarkers (TnT or TnI) New or presumably new ST-segment depression Signs or symptoms of HF or new or worsening mitral regurgitation High-risk findings from noninvasive testing Hemodynamic instability Sustained ventricular tachycardia PCI within 6 months Prior CABG High-risk score (e.g., TIMI, GRACE) Reduced left ventricular function (LVEF <40%)
Conservative	Low-risk score (e.g., TIMI, GRACE) Patient or physician preference in the absence of high-risk features

TABLE 56G-4 ACC/AHA Class I and III Recommendations for Anti-Ischemic Therapy

Class I

1. Bed/chair rest with continuous ECG monitoring
2. NTG 0.4 mg sublingually every 5 min for a total of 3 doses; afterward, assess need for IV NTG
3. NTG IV for first 48 hr after UA/NSTEMI for treatment of persistent ischemia, HF, or hypertension
4. Decision to administer NTG IV and dose should not preclude therapy with other mortality-reducing interventions such as beta blockers or ACE inhibitors
5. Beta blockers (via oral route) within 24 hr without a contraindication (e.g., HF) irrespective of concomitant performance of PCI
6. When beta blockers are contraindicated, a nondihydropyridine calcium antagonist (e.g., verapamil or diltiazem) should be given as initial therapy in the absence of severe LV dysfunction or other contraindications
7. ACE inhibitor (via oral route) within first 24 hr with pulmonary congestion, or LVEF ≤ 0.40 , in the absence of hypotension (systolic blood pressure <100 mm Hg or <30 mm Hg below baseline) or known contraindications to that class of medications
8. ARB should be administered to UA/NSTEMI patients who are intolerant of ACE inhibitors and have either clinical or radiologic signs of heart failure or LVEF ≤ 0.40 . Valsartan and candesartan have demonstrated efficacy for this indication.

Thank You!