Management of Ischemic Heart Disease (IHD)

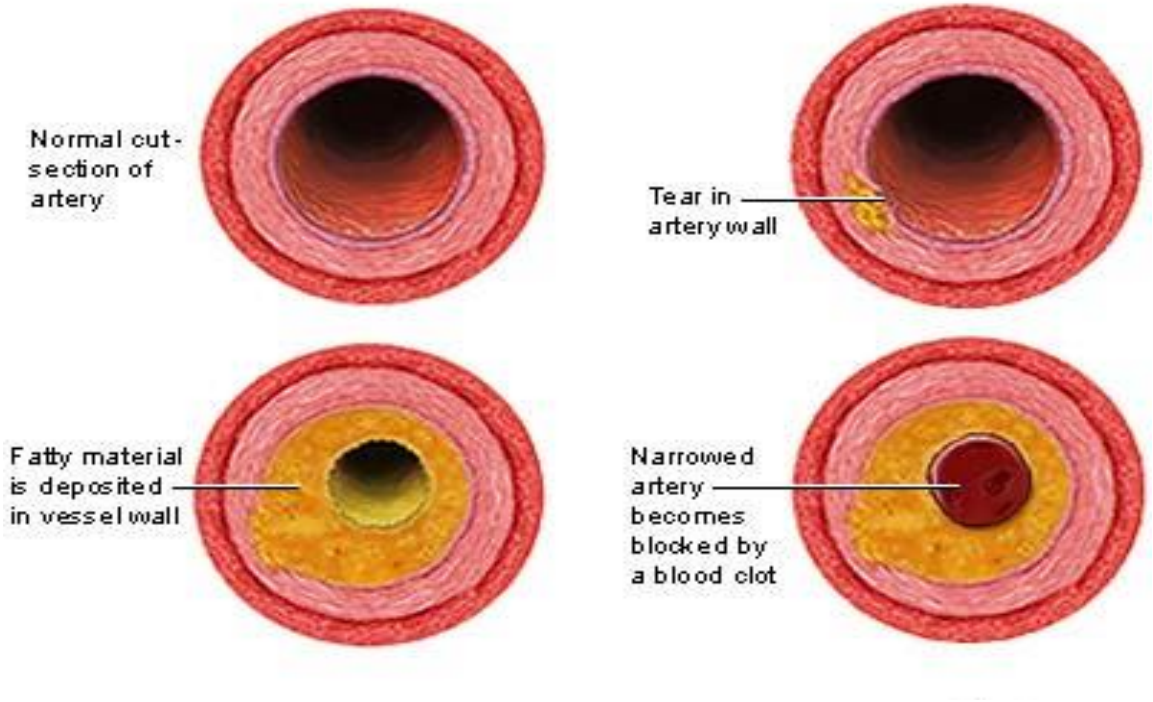
Dr Tarek Elhussein MD, FRCP, FASE
Director, Adult Cardiology Fellowship Program
Assisstant professor of Medicine & Cardiac Sicences
Consultant cardiology & echocardiography
King Fahad Cardiac Centre – KSU

Introduction

- Coronary heart disease (CHD) is the most common form of heart disease
- An estimated 330 000 people have a myocardial infarct each year
- Approximately 1.3 million people have angina each year

Introduction

- Disease of the coronary arteries is almost always due to atheroma and its complications
  - particularly thrombosis

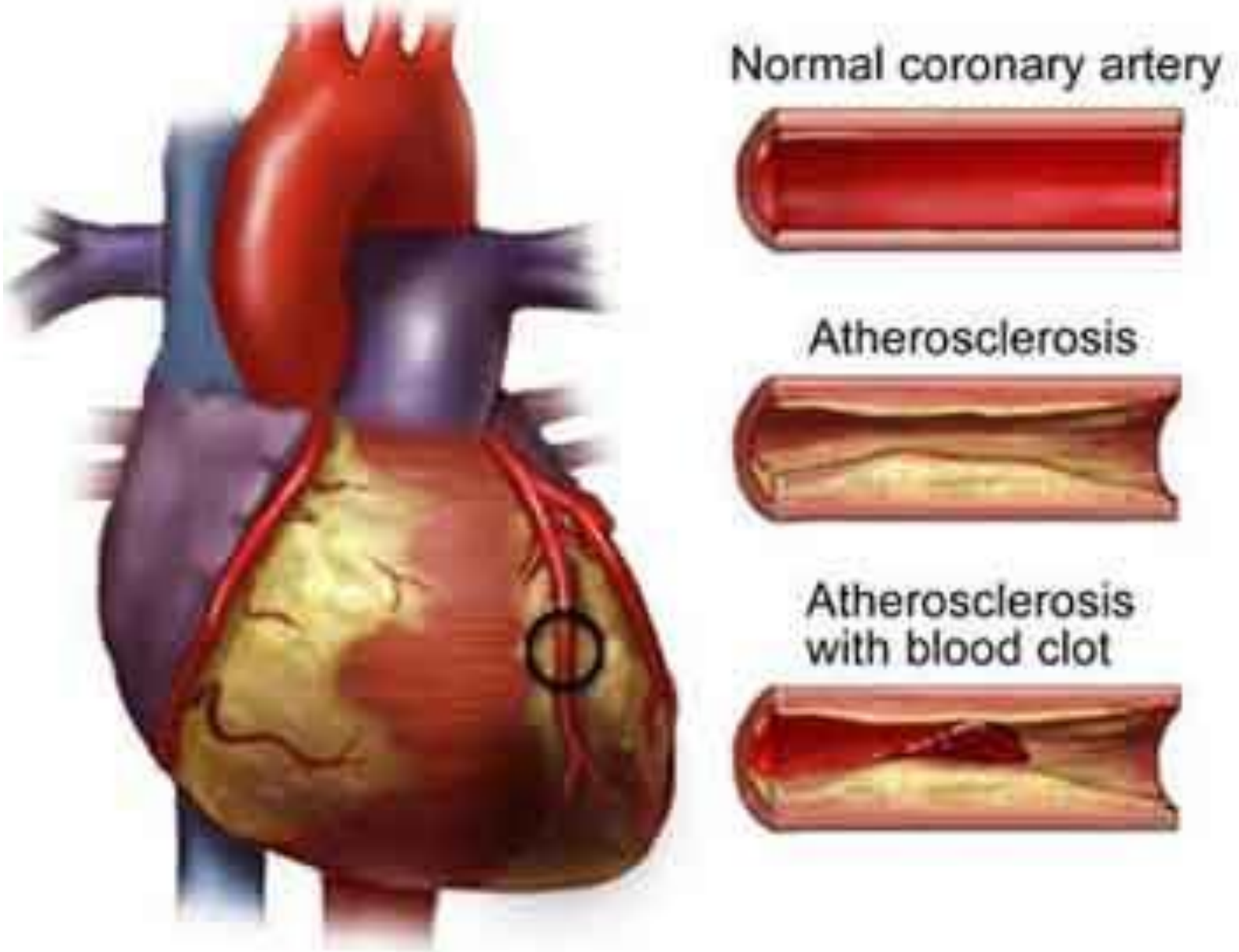


Myocardial Ischemia

- Results when there is an imbalance between myocardial oxygen supply and demand
- Most occurs because of atherosclerotic plaque with in one or more coronary arteries
- Limits normal rise in coronary blood flow in response to increase in myocardial oxygen demand

Oxygen Carrying Capacity

- The oxygen carrying capacity relates to the content of hemoglobin and systemic oxygenation
- When atherosclerotic disease is present, the artery lumen is narrowed and vasoconstriction is impaired
- Coronary blood flow cannot increase in the face of increased demands and ischemia may result


Normal coronary artery
Atherosclerosis
Atherosclerosis
with blood clot

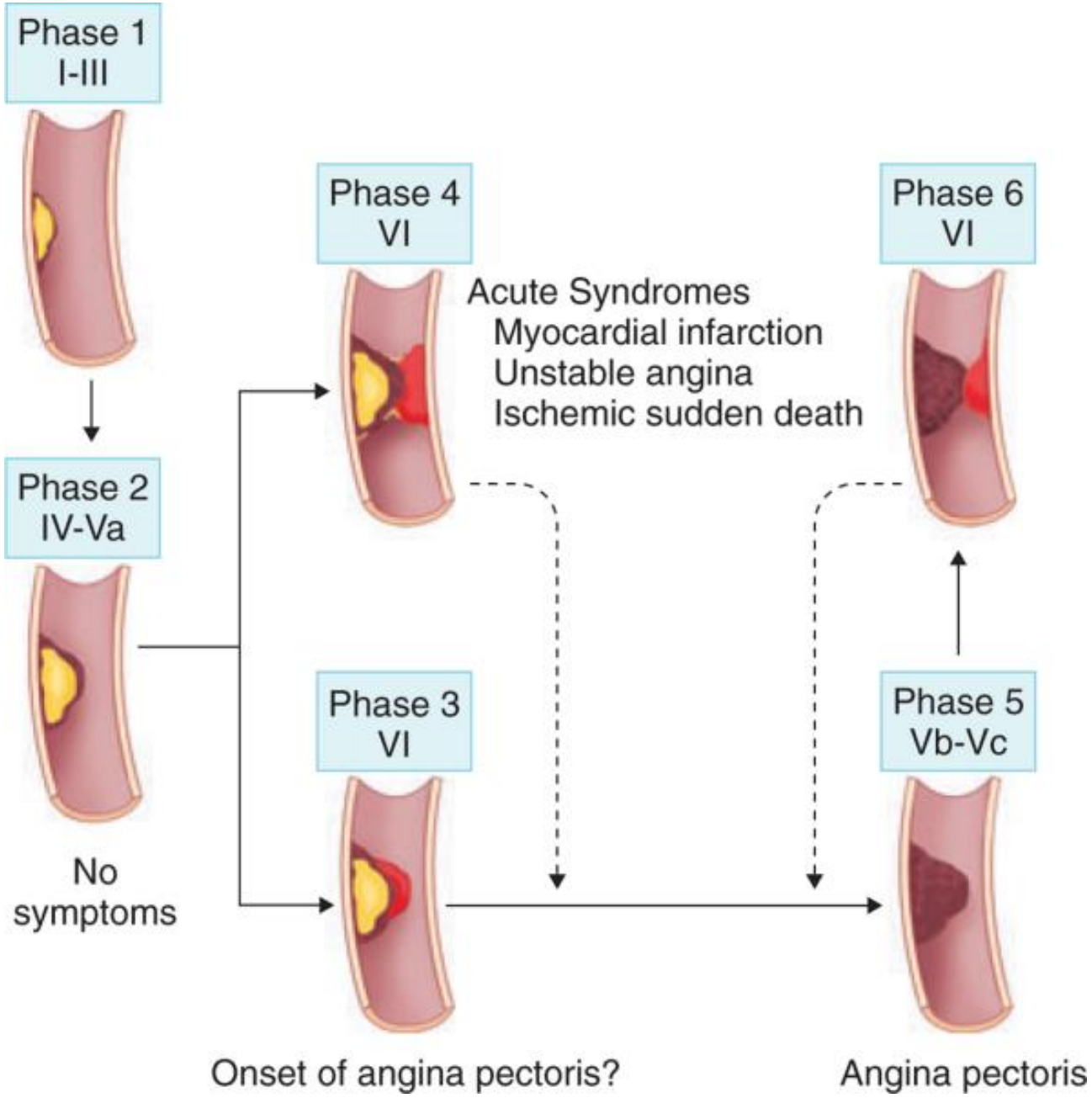
Ischaemic Heart Disease

- **Angina**
  - **Stable**
  - **Unstable**
  - **Prinzmetal's**

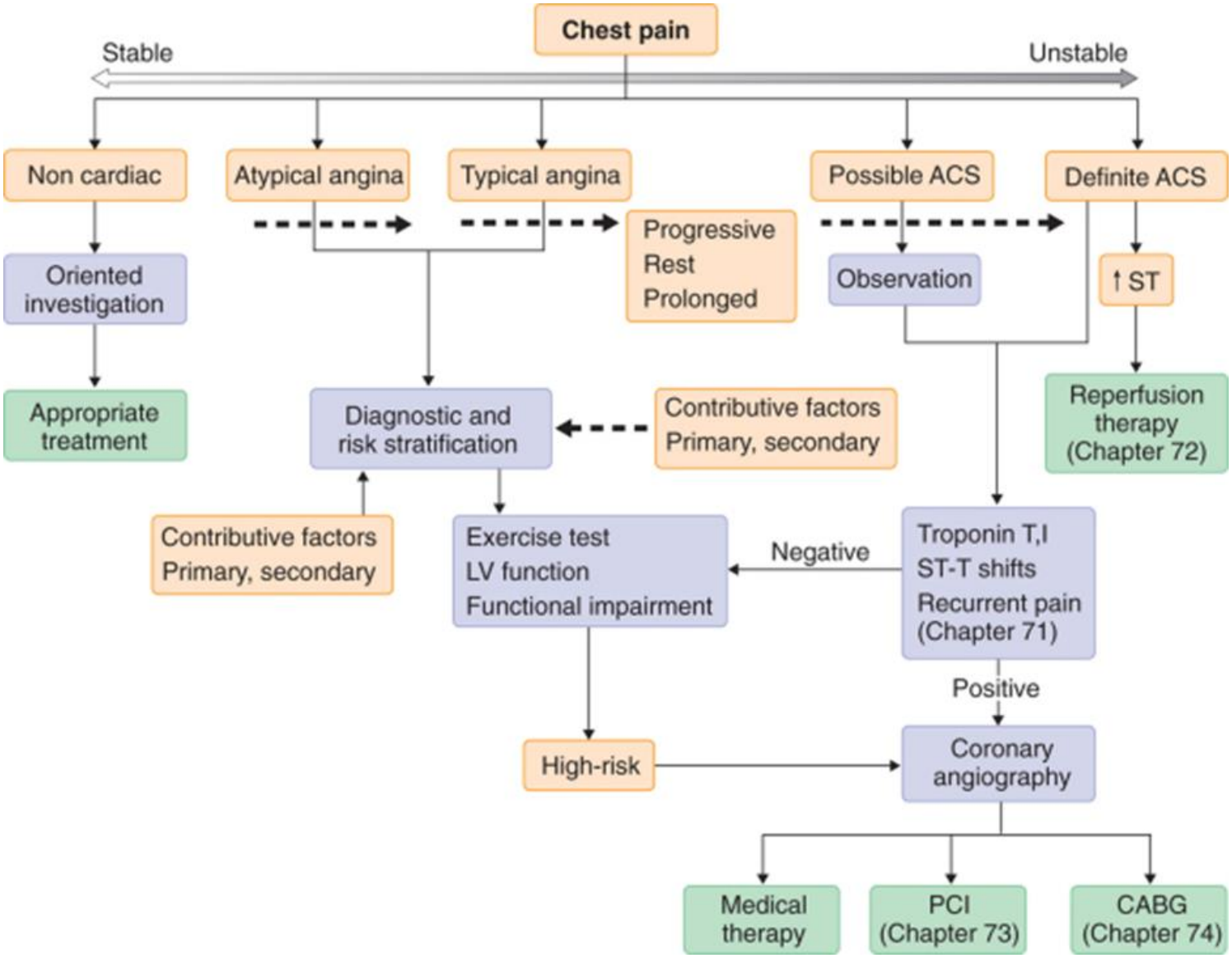
- **Myocardial Infarction**
  - **NSTEMI**
  - **STEMI**

Clinic pathological correlation

Clinical Problem	Pathology
Stable angina	Ischaemia due to fixed atheromatous stenosis of one or more coronary arteries
Unstable angina	Ischaemia caused by dynamic obstruction of a coronary artery due to plaque rupture with superimposed thrombosis and spasm
Myocardial infarction	Myocardial necrosis caused by acute occlusion of a coronary artery due to plaque rupture and thrombosis
Heart failure	Myocardial dysfunction due to infarction or ischaemia
Arrhythmia	Altered conduction due to ischaemia or infarction
Sudden death	Ventricular arrhythmia, asystole or massive myocardial infarction


Phase 1
I-III
Phase 2
IV-Va
No
symptoms
Phase 4
VI
Phase 3
VI
Acute Syndromes
Myocardial infarction
Unstable angina
Ischemic sudden death
Phase 6
VI
Phase 5
Vb-Vc
Onset of angina pectoris?
Angina pectoris

Evaluation of Chest pain



Acute Coronary Syndromes

	Stable Angina	Unstable Angina	STEMI	NSTEMI
Character of pain	Exertional pain	Rest pain	Rest pain	Rest pain
Relievers	Responds to GTN	No GTN effect	No GTN effect	No GTN effect
Enzymes	Normal	Normal	Elevated	Elevated
ECG	Often normal	Often ST depression	ST segment elevation	No ST segment elevation

Angina

- When ischemia results it is frequently accompanied by chest discomfort: Angina Pectoris

- In the majority of patients with angina, development of myocardial ischemia results from a combination of fixed and vasospastic stenosis

Chronic Stable Angina

- May develop sudden increase in frequency and duration of ischemic episodes occurring at lower workloads than previously or even at rest

- Known as unstable angina: up to 70% patients sustain MI over the ensuing 3 months

Angina: cont

- Patients with mild obstruction coronary lesions can also experience unstable angina

- >90% of Acute MI result from an acute thrombus obstructing a coronary artery with resultant prolonged ischemia and tissue necrosis

Treatment of Angina

- Treatment of Chronic Angina is directed at minimizing myocardial oxygen demand and increasing coronary flow

- Where as in the acute syndromes of unstable angina or MI primary therapy is also directed against platelet aggregation and thrombosis

Epidemiology

- Modifiable Factors: hyperlipidemia- ^ LDL (<130 normal) or low HDL (>60 normal), Hypertension, cigarette smoking and diabetes, obesity, BMI of >30
- Non-Modifiable Factors: advanced age, male sex, family medical history: male <55 y/o, female <65 y/o
- Other: sedentary lifestyle and stressful emotional stress

Quality

- Tightness, squeezing, heaviness, pressure, burning, indigestion or aching sensation
- It is rarely “PAIN”
- Never: sharp, stabbing, prickly, spasmodic, or pleuritic
- Lasts a few seconds < 10 minutes
- Relieved by NTG s/l
- Levine Sign: clench fist to sternum

Signs & Symptoms accompany Angina

- Dyspnea, nausea, diaphoresis resolve quickly after cessation of angina

- Angina is a diffuse sensation rather than discrete

Ischemic Heart Disease

- Imbalance between Myocardial oxygen supply and demand = Myocardial hypoxia and accumulation of waste metabolites due to atherosclerotic disease of coronary arteries

Stable Angina

- Stable Angina: chronic pattern of transient angina pectoris precipitated by physical activity or emotional upset, relieved by rest with in few minutes

- Temporary depression of ST segment with no permanent myocardial damage

Angina Pectoris

- Angina Pectoris: uncomfortable sensation in the chest or neighboring anatomic structures produced by myocardial ischemia

Variant Angina

- Typical anginal discomfort usually at rest
- Develops due to coronary artery spasm rather than increase myocardial oxygen demand
- Transient shifts of ST segment – ST elevation

Unstable Angina

- Increased frequency and duration of Angina episodes, produced by less exertion or at rest = high frequency of myocardial infarction if not treated

Silent Ischemia

- Asymptomatic episodes of myocardial ischemia

- Detected by electrocardiogram and laboratory studies

Myocardial Infarction

- Region of myocardial necrosis due to prolonged cessation of blood supply
- Results from acute thrombus at side of coronary atherosclerotic stenosis
- May be first clinical manifestation of ischemic heart disease or history of Angina Pectoris

Precipitants

- Exertion: walking, climbing stairs, vigorous work using arms, sexual activity
- Vasoconstriction: extremities, increased systemic vascular resistance, increased in myocardial wall tension and oxygen requirements
- Myocardial Ischemia displays a circadian rhythm threshold for Angina it is lower in morning hours.

Physical Examination

- Arcus senilis, xanthomas, funduscopic exam: AV nicking, exudates
- Signs and symptoms: hyperthyroidism with increased myocardial oxygen demand, hypertension, palpitations
- Auscultate carotid and peripheral arteries and abdomen: aortic aneurysm
- Cardiac: S4 common in CAD, increased heart rate, increased blood pressure

Ischemia

- Myocardial ischemia may result in papillary muscle regurgitation
- Ischemic induced left ventricular wall motion abnormalities may be detected as an abnormal precordial bulge on chest palpation
- A transient S3 gallop and pulmonary rales = ischemic induced left ventricular dysfunction

Diagnostic Tests

- Blood tests include serum lipids, fasting blood sugar, Hematocrit, thyroid (anemias and hyperthyroidism can exacerbate myocardial ischemia
- Resting Electrocardiogram: CAD patients have normal baseline ECGs
  - pathologic Q waves = previous infarction
  - minor ST and T waves abnormalities not specific for CAD

Electrocardiogram

- Electrocardiogram: is useful in diagnosis during cc: chest pain
- When ischemia results in transient horizontal or downsloping ST segments or T wave inversions which normalize after pain resolution
- ST elevation suggest severe transmural ischemia or coronary artery spasm which is less often

Exercise Stress Test

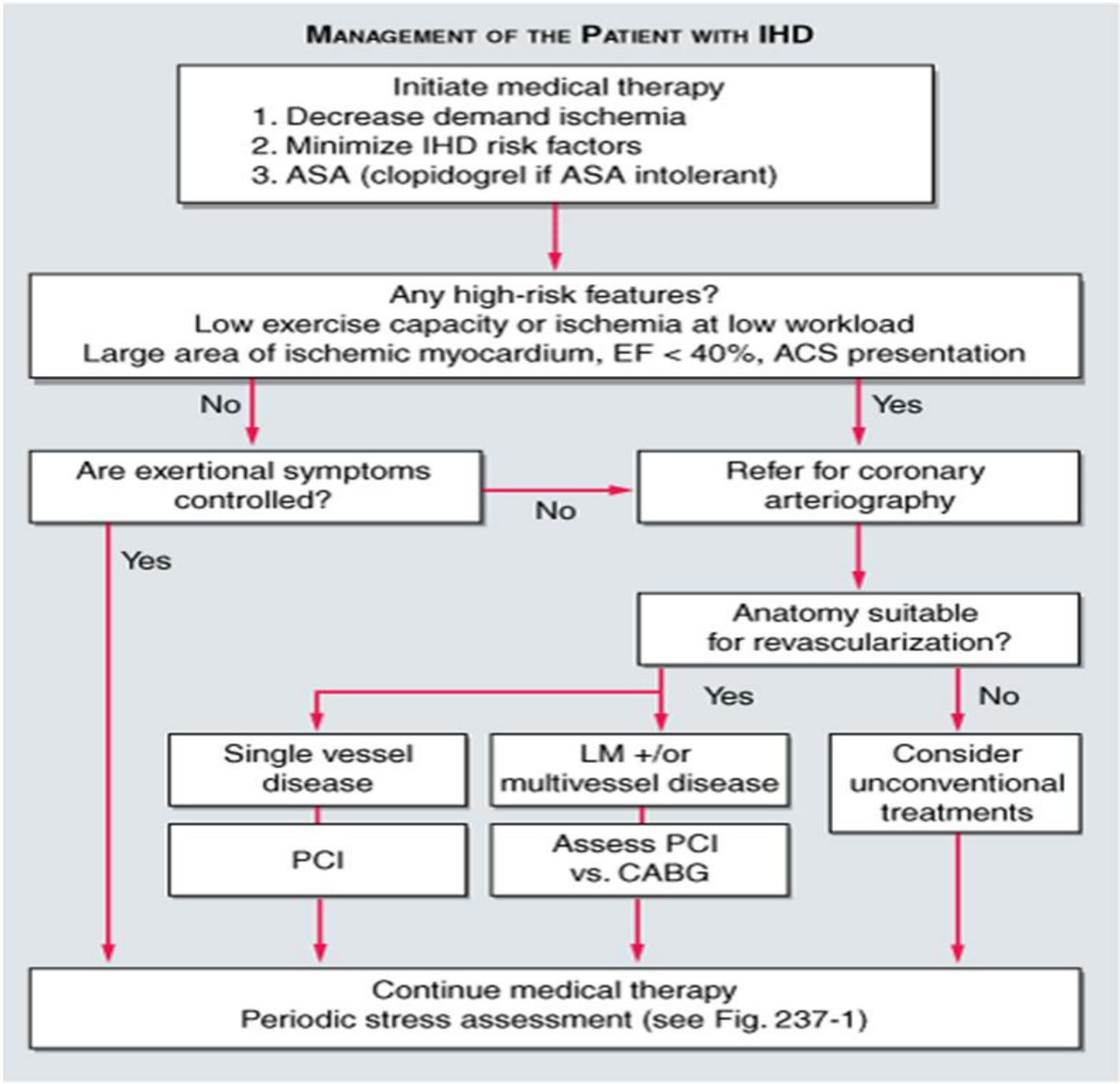
- Used to confirm diagnosis of angina
- Terminate if hypotension, high grade ventricular disrrhythmias, 3 mm ST segment depression develop
- (+): reproduction of chest pain, ST depression
- Severe: chest pain, ST changes in 1st 3 minutes, >3 mm ST depression, persistent > 5 minutes after exercise stopped
- Low systolic BP, multifocal ventricular ectopy or V- tach, ST changes, poor duration of exercise (<2 minutes) due to cardiopulmonary limitations

Other Diagnostic Tests

- Radionuclide studies
- Myocardial perfusion scintigraphy
- Exercise radionuclide ventriculography
- Echocardiography
- Ambulatory ECG monitoring
- Coronary arteriography

Management Goals to reduce Anginal Symptoms

- Prevent complications – myocardial infarction, and to prolong life
- No smoking, lower weight, control hypertension and diabetes
- Patients with CAD – LDL cholesterol should achieve lower levels (<100)
- HMG-COA reductase inhibitors are effective


MANAGEMENT OF THE PATIENT WITH IHD
Initiate medical therapy
1. Decrease demand ischemia
2. Minimize IHD risk factors
3. ASA (clopidogrel if ASA intolerant)
Any high-risk features?
Low exercise capacity or ischemia at low workload
Large area of ischemic myocardium, EF < 40%, ACS presentation
No
Yes
Are exertional symptoms controlled?
No
Refer for coronary arteriography
Yes
Anatomy suitable for revascularization?
Yes
No
Single vessel disease
LM +/or multivessel disease
Consider unconventional treatments
PCI
Assess PCI vs. CABG
Continue medical therapy
Periodic stress assessment (see Fig. 237-1)

Pharmacologic Therapy

- Therapy is aimed in restoring balance between myocardial oxygen supply and demand

- Useful Agents: nitrates, beta-blockers and calcium channel blockers

Nitrates

- Reduce myocardial oxygen demand
- Relax vascular smooth muscle
- Reduces venous return to heart
- Arteriolar dilators decrease resistance against-which left ventricle contracts and reduces wall tension and oxygen demand

Nitrates: cont

- Dilate coronary arteries with augmentation of coronary blood flow
- Side effects: generalized warmth, transient throbbing headache, or lightheadedness, hypotension
- ER if no relief after X2 nitro's: unstable angina or MI

Problems with Nitrates

- Drug tolerance
- Continued administration of drug will decrease effectiveness
- Prevented by allowing 8 – 10 hours nitrate free interval each day.
- Elderly/inactive patients: long acting nitrates for chronic antianginal therapy is recommended
- Physical active patients: additional drugs are required

Beta Blockers

- Prevent effort induced angina
- Decrease mortality after myocardial infarction
- Reduce Myocardial oxygen demand by slowing heart rate, force of ventricular contraction and decrease blood pressure

Beta -1

- Block myocardial receptors with less effect on bronchial and vascular smooth muscle- patients with asthma, intermittent claudication

Beta-Agonist blockers

- With partial B-agonist activity:
- Intrinsic sympathomimetic activity (ISA) have mild direct stimulation of the beta receptor while blocking receptor against circulating catecholamines
- Agents with ISA are less desirable in patients with angina because higher heart rates during their use may exacerbate angina
- not reduce mortality after AMI

Beta-blockers

- Duration of beta-blockers depends on lipid solubility
- Accounts for different dosage schedules

Contraindications

- Symptomatic CHF, history of bronchospasm, bradycardia or AV block, peripheral vascular disease with s/s of claudication

Side Effects

- Bronchospasm (RAD), CHF, depression, sexual dysfunction, AV block, exacerbation of claudication, potential masking of hypoglycemia in IDDM patients

Calcium Channel Blockers

- Anti-anginal agents prevent angina
- Helpful: episodes of coronary vasospasm
- Decreases myocardial oxygen requirements and increase myocardial oxygen supply
- Potent arterial vasodilators: decrease systemic vascular resistance, blood pressure, left ventricular wall stress with decrease myocardial oxygen consumption

Calcium Channel Blockers

- Secondary agents in management of stable angina
- Are prescribed only after beta blockers and nitrate therapy has been considered
- Potential to adversely decrease left ventricular contractility
- Used cautiously in patients with left ventricular dysfunction

Drug Selection

- Chronic Stable Angina: beta blocker and long acting nitrate or calcium channel blocker (not verapamil: bradycardia) or both.
- If contraindication to BB a CCB is recommended (bronchospasm, IDDM, or claudication) any of CCB approved for angina are appropriate.

Drugs

- Verapamil and Cardizem is preferred because of effect on slowing heart rate
- Patients with resting bradycardia or AV block, a dihydropyridine calcium blocker is better choice
- Patients with CHF: nitrates preferred amlodipine should be added if additional therapy is needed

Drugs

- Primary coronary vasospasm: no treatment with beta blockers, it could increase coronary constriction
- Nitrates and CCB are preferred
- Concomitant hypertension: BB or CCB are useful in treatment
- Ischemic Heart Disease & Atrial Fibrillation: treatment with BB, verapamil or Cardizem can slow ventricular rate

Combination Therapy

- If patients do not respond to initial antianginal therapy – a drug dosage increase is recommended unless side effects occur.

- Combination therapy: successful use of lower dosages of each agent while minimizing individual drug side effects

Combination Therapy Include:

- Nitrate and beta blocker
- Nitrate and verapamil or cardizem for similar reasons
- Long acting dihydropyridine calcium channel blocker and beta blocker
- A dihydropyridine and nitrate is often not tolerated without concomitant beta blockade because of marked vasodilatation with resultant head ache and increased heart rate

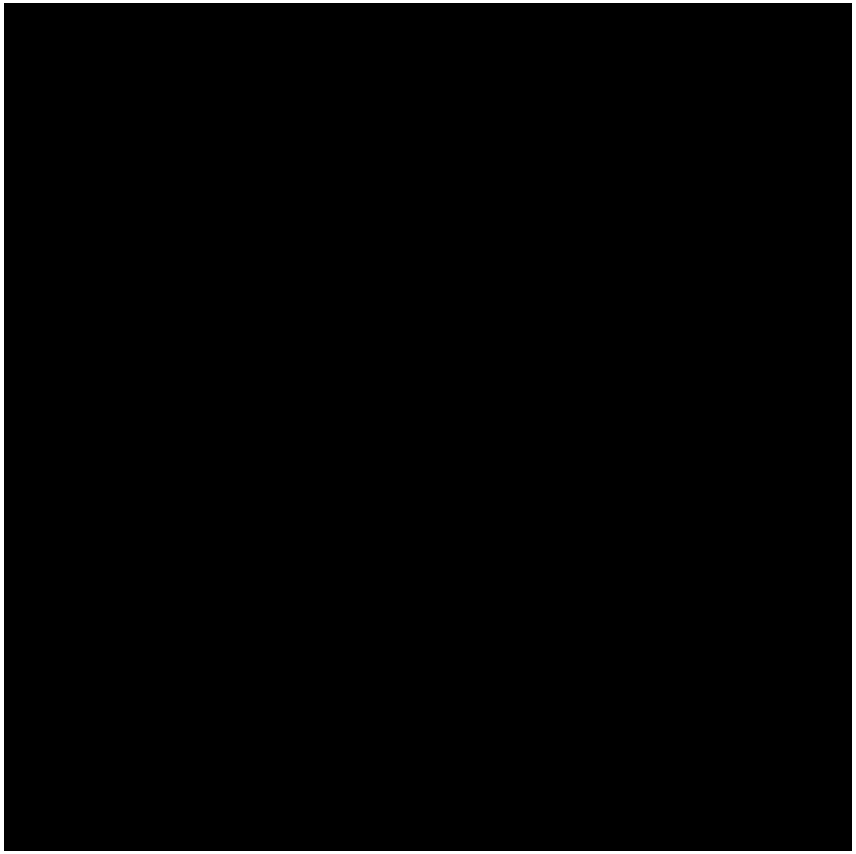
Combinations

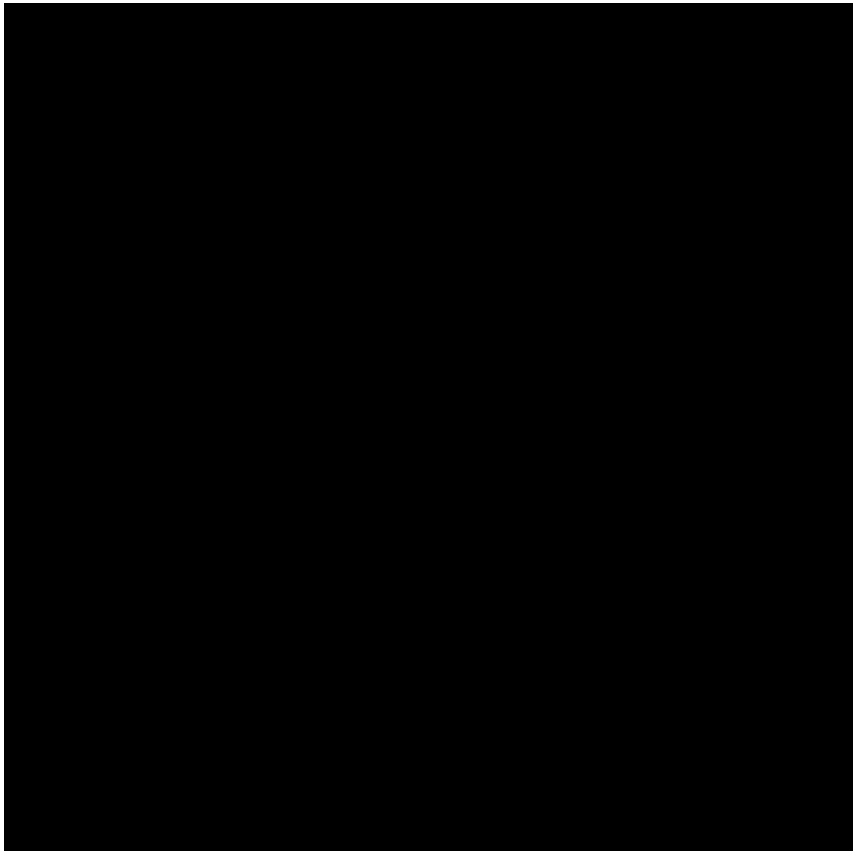
- Beta blockers should be combined only very cautiously with verapamil or cardizem because of potential of excessive bradycardia or CHF in patients with left ventricular dysfunction

Other methods

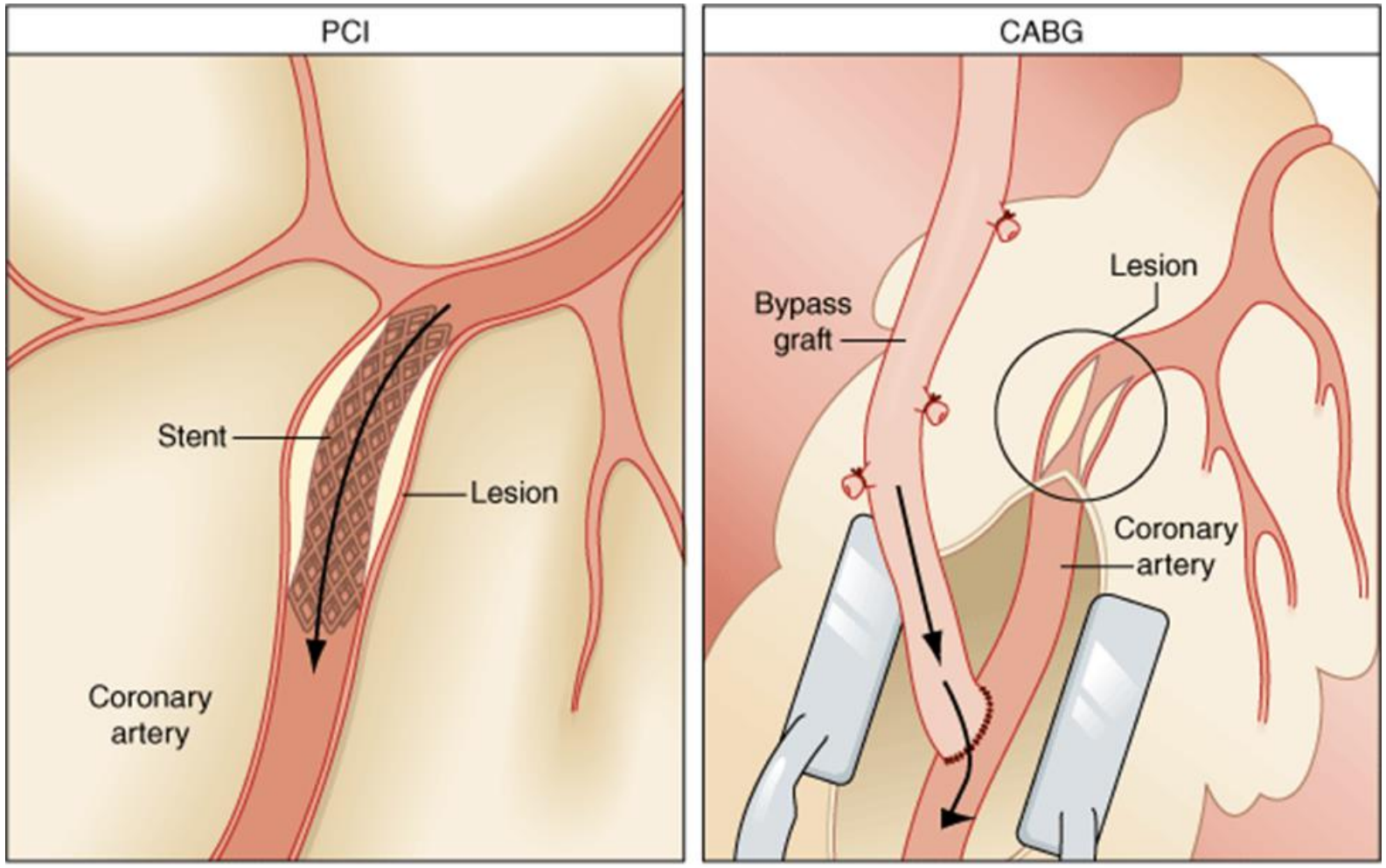
- Patients with 1 – 2 vessel disease with normal left ventricular function are referred for catheter based procedures
- Patients with 2 and 3 vessel disease with widespread ischemia, left ventricular dysfunction or DM and those with lesions are not amendable to catherization based procedures and are referred for CABG

Cardiac Cath





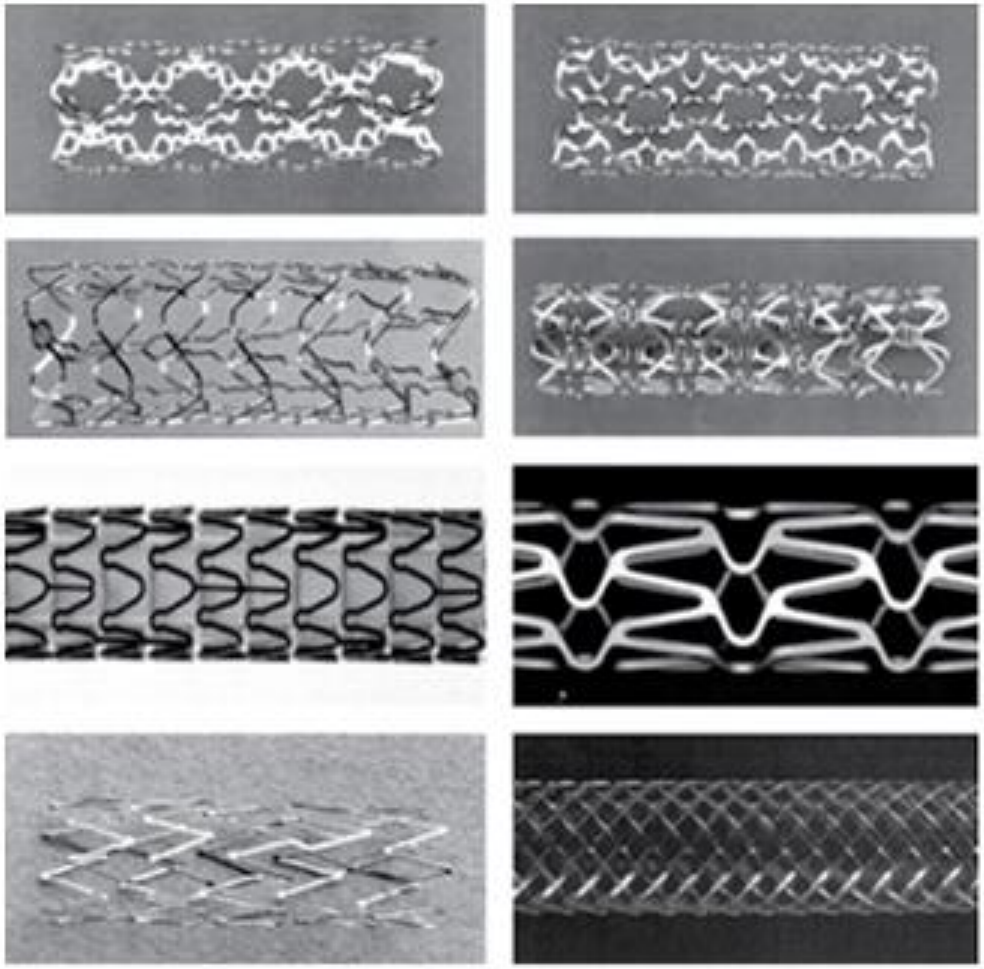
PCI vs CABG



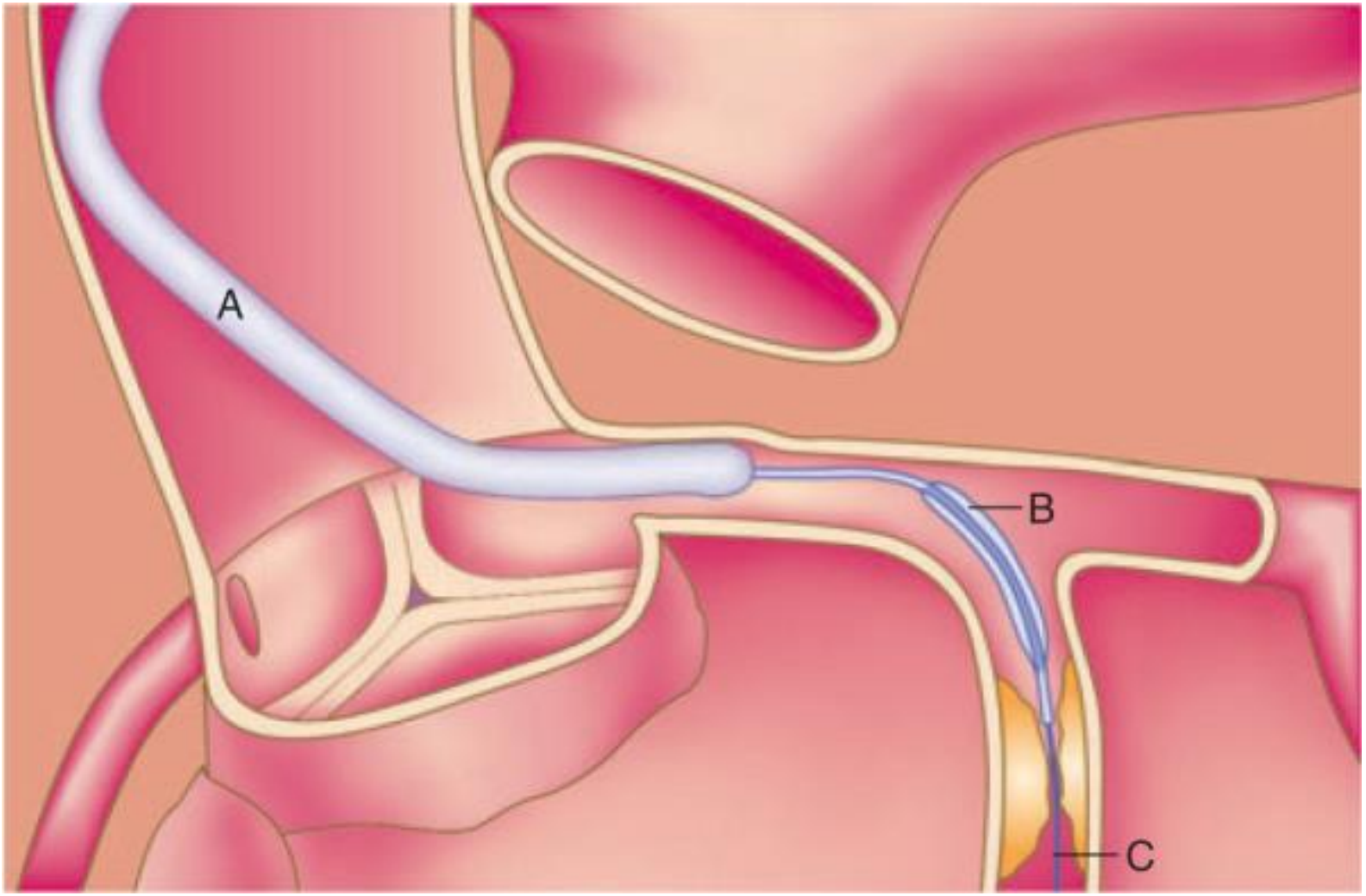
Stent addresses the existing lesion but not future lesions.

Bypass grafting addresses the existing lesion and also future culprit lesions.

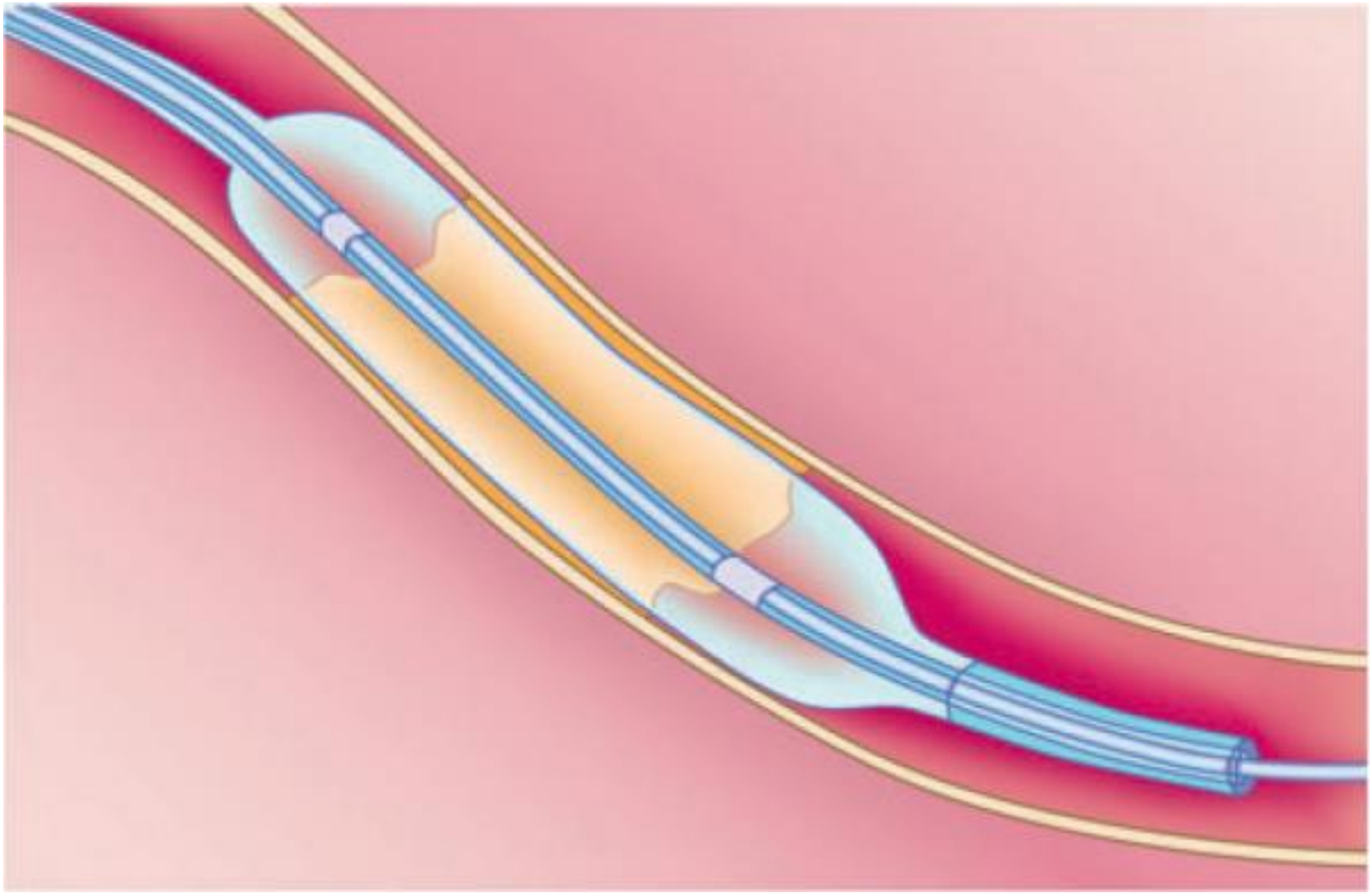
Metal stents



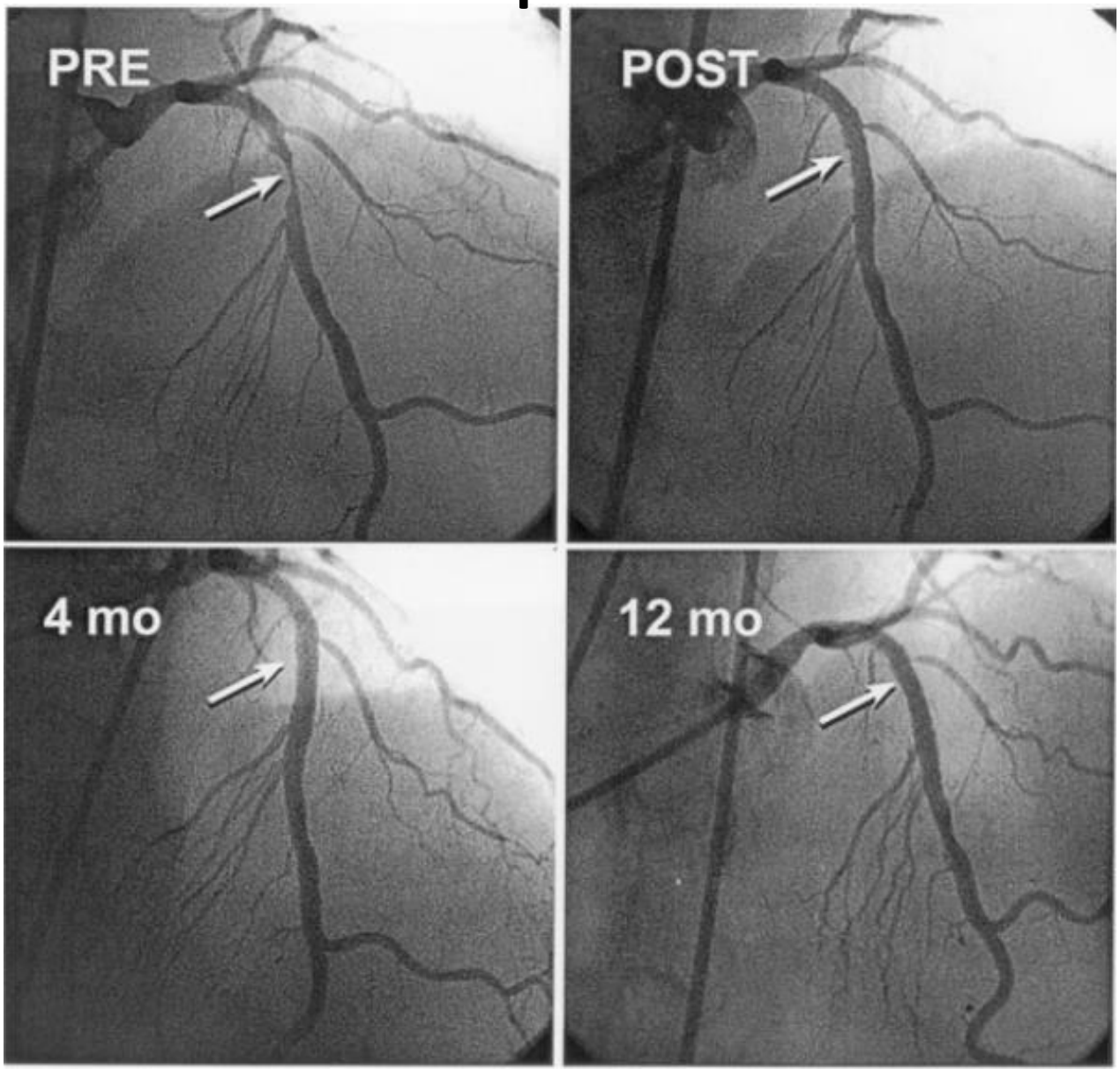
PCI



PCI



Pre & post PCI



Acute Coronary Syndromes

- Unstable Angina
- Non-ST-Segment Elevation MI (NSTEMI)
- ST-Segment Elevation MI (STEMI)

Similar pathophysiology

Similar presentation and early management rules

STEMI requires evaluation for acute reperfusion intervention

Diagnosis of Acute MI
STEMI / NSTEMI

- At least 2 of the following
  - Ischemic symptoms
  - Diagnostic ECG changes
  - Serum cardiac marker elevations

Diagnosis of Angina

- Typical angina—All three of the following
  - Substernal chest discomfort
  - Onset with exertion or emotional stress
  - Relief with rest or nitroglycerin
- Atypical angina
  - 2 of the above criteria
- Noncardiac chest pain
  - 1 of the above

Diagnosis of Unstable Angina

- Patients with typical angina - An episode of angina
  - Increased in severity or duration
  - Has onset at rest or at a low level of exertion
  - Unrelieved by the amount of nitroglycerin or rest that had previously relieved the pain

- Patients not known to have typical angina
  - First episode with usual activity or at rest within the previous two weeks
  - Prolonged pain at rest

Unstable Angina

Non occlusive thrombus

Non specific ECG

Normal cardiac enzymes

NSTEMI

Occluding thrombus sufficient to cause tissue damage & mild myocardial necrosis

ST depression +/- T wave inversion on ECG

Elevated cardiac enzymes

STEMI

Complete thrombus occlusion

ST elevations on ECG or new LBBB

Elevated cardiac enzymes

More severe symptoms

Acute Management

- Initial evaluation & stabilization
- Efficient risk stratification
- Focused cardiac care

Evaluation

- Efficient & direct history
- Initiate stabilization interventions

Occurs simultaneously

Plan for moving rapidly to indicated cardiac care

Chest pain suggestive of ischemia

Immediate assessment within 10 Minutes

Initial labs and tests

- 12 lead ECG
- Obtain initial cardiac enzymes
- electrolytes, cbc lipids, bun/cr, glucose, coags
- CXR

Emergent care

- **IV access**
- **Cardiac monitoring**
- **Oxygen**
- **Aspirin**
- **Nitrates**

History & Physical

- **Establish diagnosis**
- **Read ECG**
- **Identify complications**
- **Assess for reperfusion**

Focused History

- Aid in diagnosis and rule out other causes
  - Palliative/Provocative factors
  - Quality of discomfort
  - Radiation
  - Symptoms associated with discomfort
  - Cardiac risk factors
  - Past medical history - especially cardiac
- Reperfusion questions
  - Timing of presentation
  - ECG c/w STEMI
  - Contraindication to fibrinolysis
  - Degree of STEMI risk

Targeted Physical Examination

- Examination
  - Vitals
  - Cardiovascular system
  - Respiratory system
  - Abdomen
  - Neurological status
- Recognize factors that increase risk
  - Hypotension
  - Tachycardia
  - Pulmonary rales, JVD, pulmonary edema,
  - New murmurs/heart sounds
  - Diminished peripheral pulses
  - Signs of stroke

ECG assessment

ST Elevation or new LBBB

STEMI

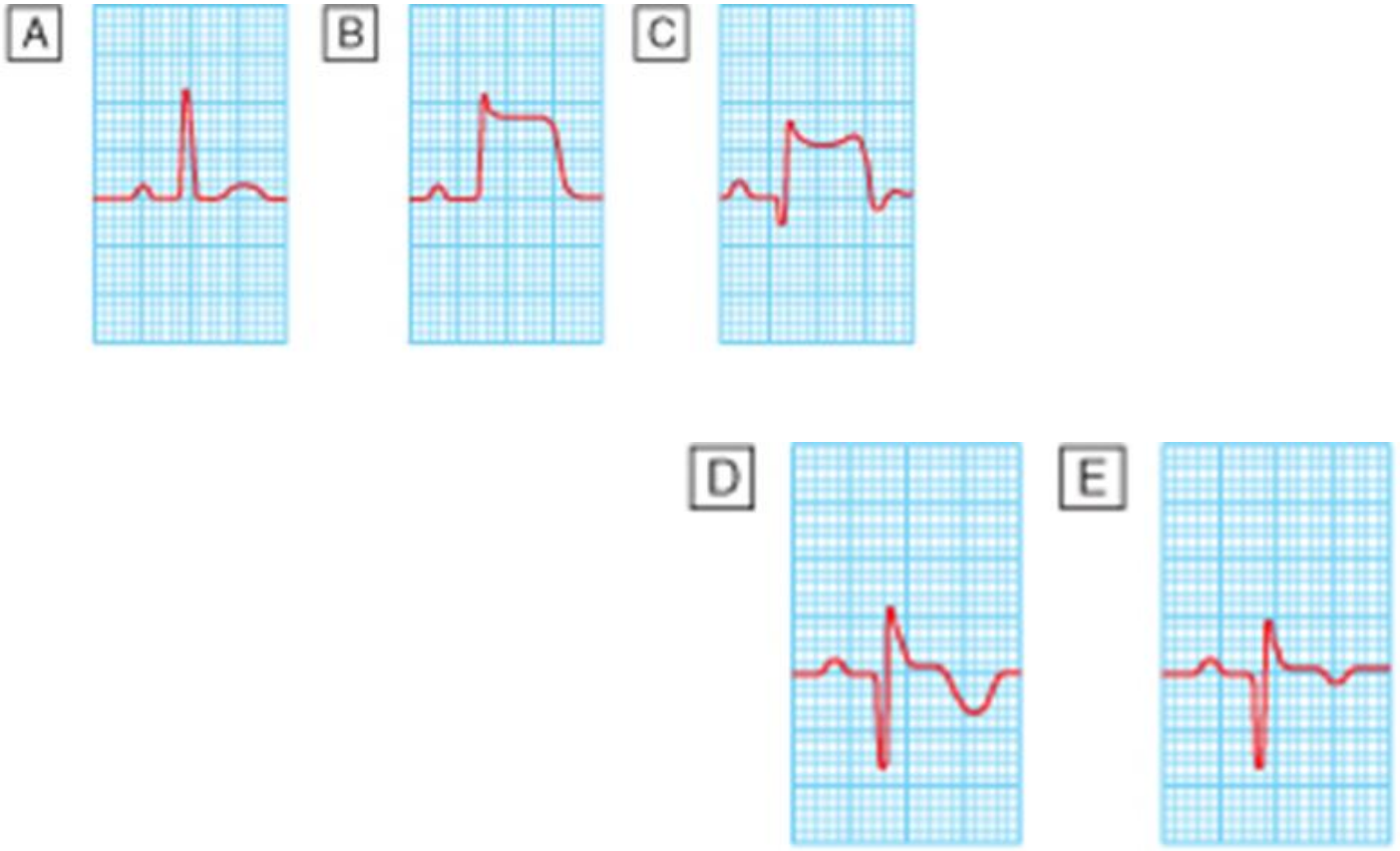
ST Depression or dynamic
T wave inversions

NSTEMI

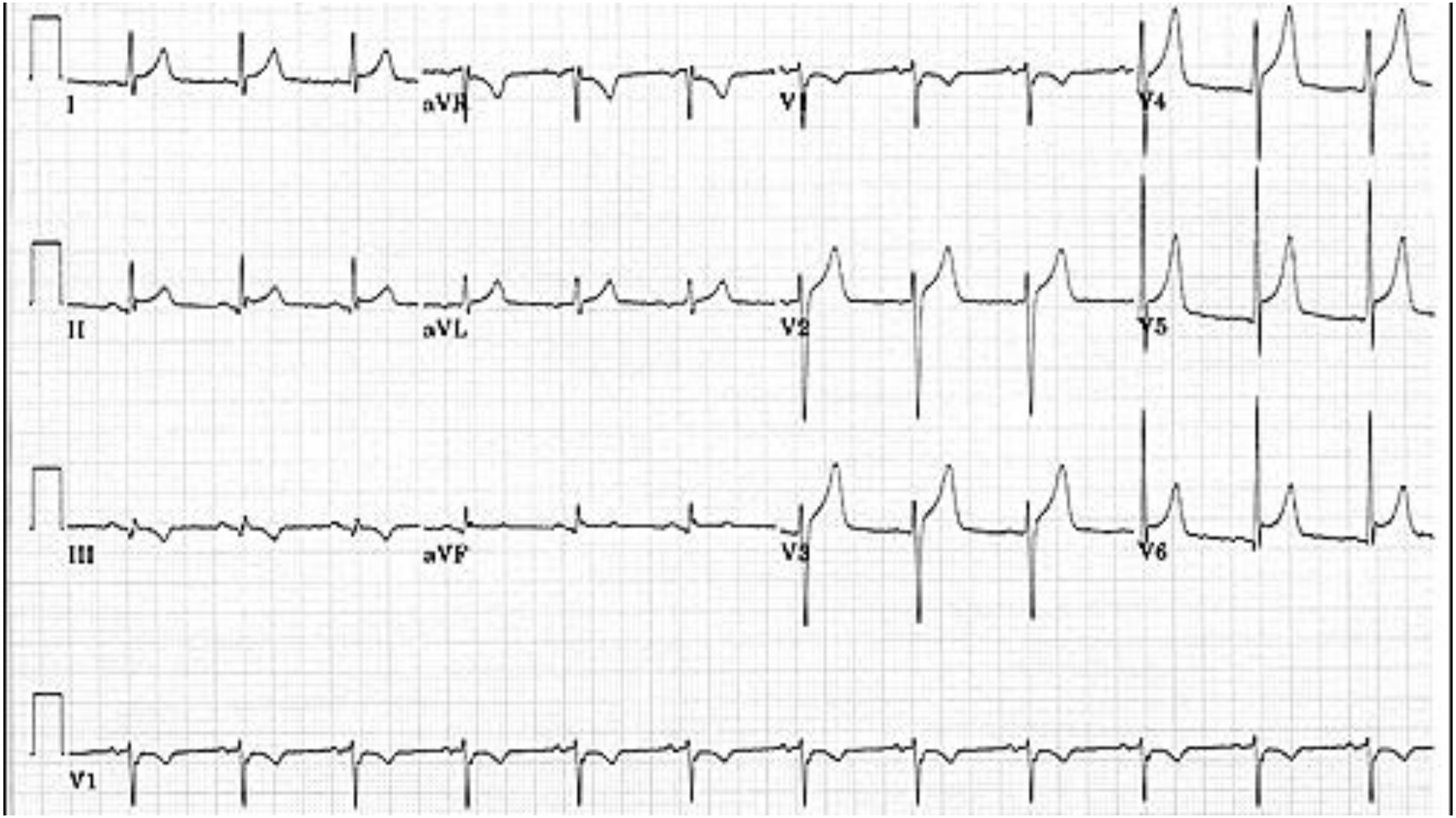
Non-specific ECG

Unstable Angina

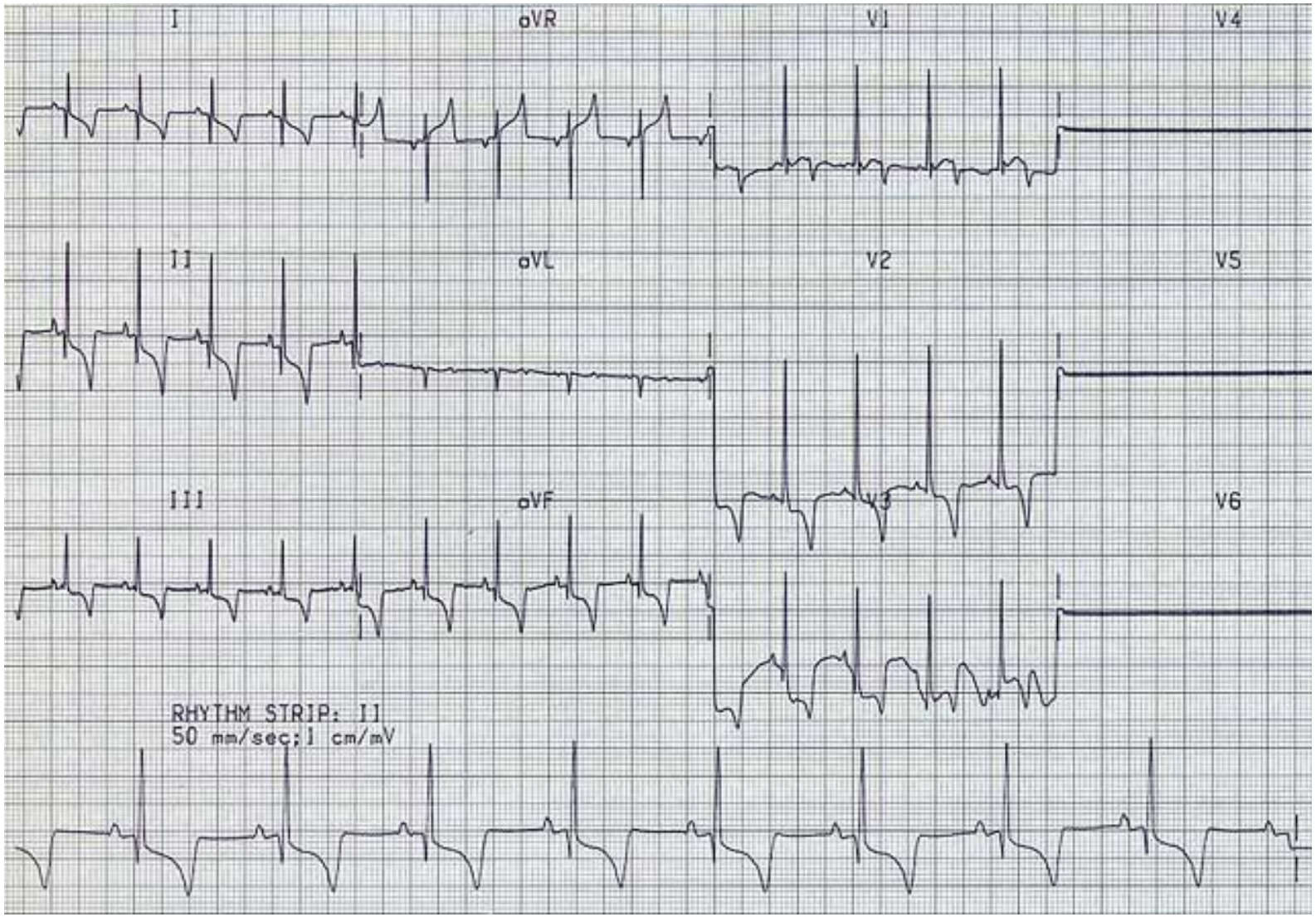
ECG changes with MI



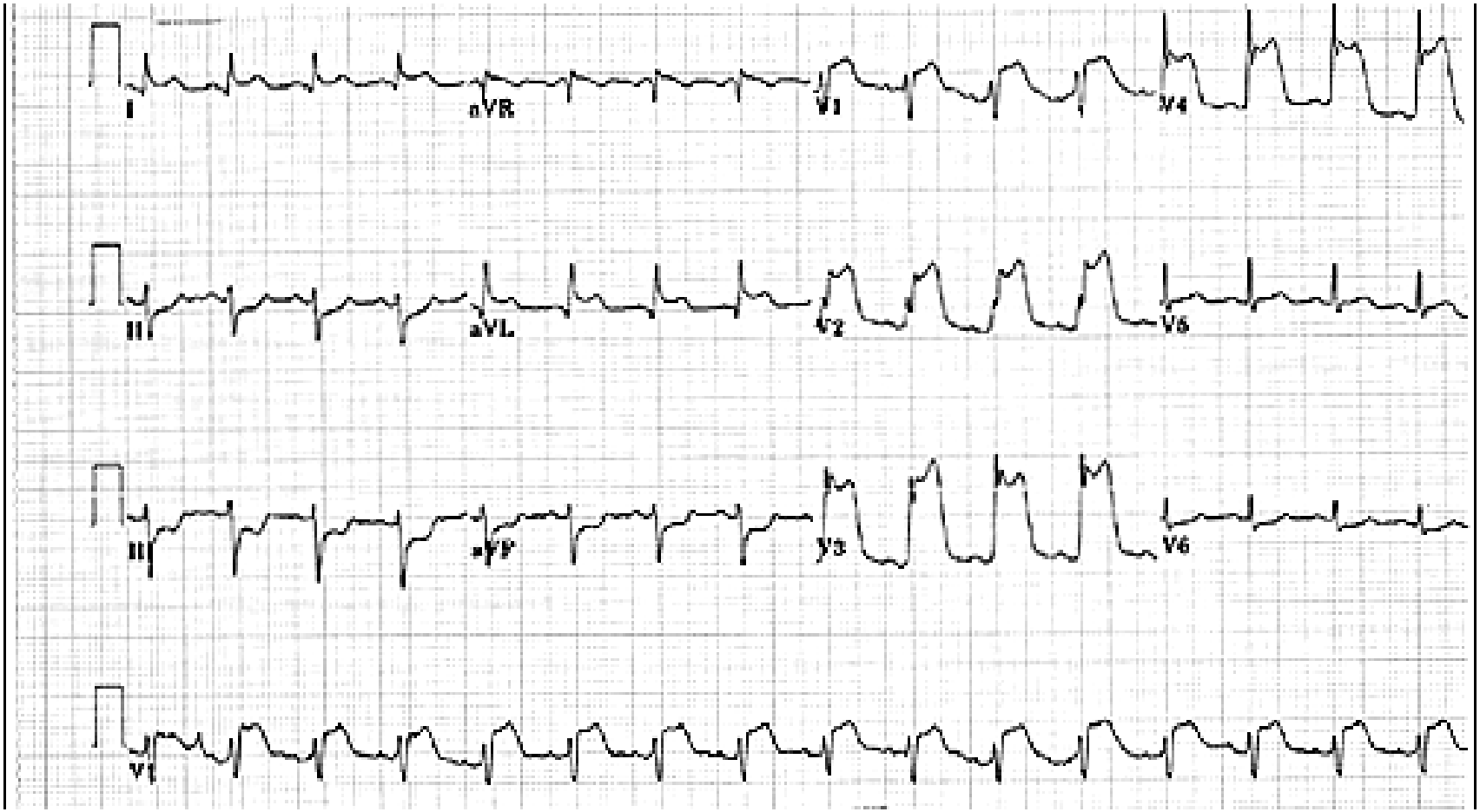
Normal or non-diagnostic ECG



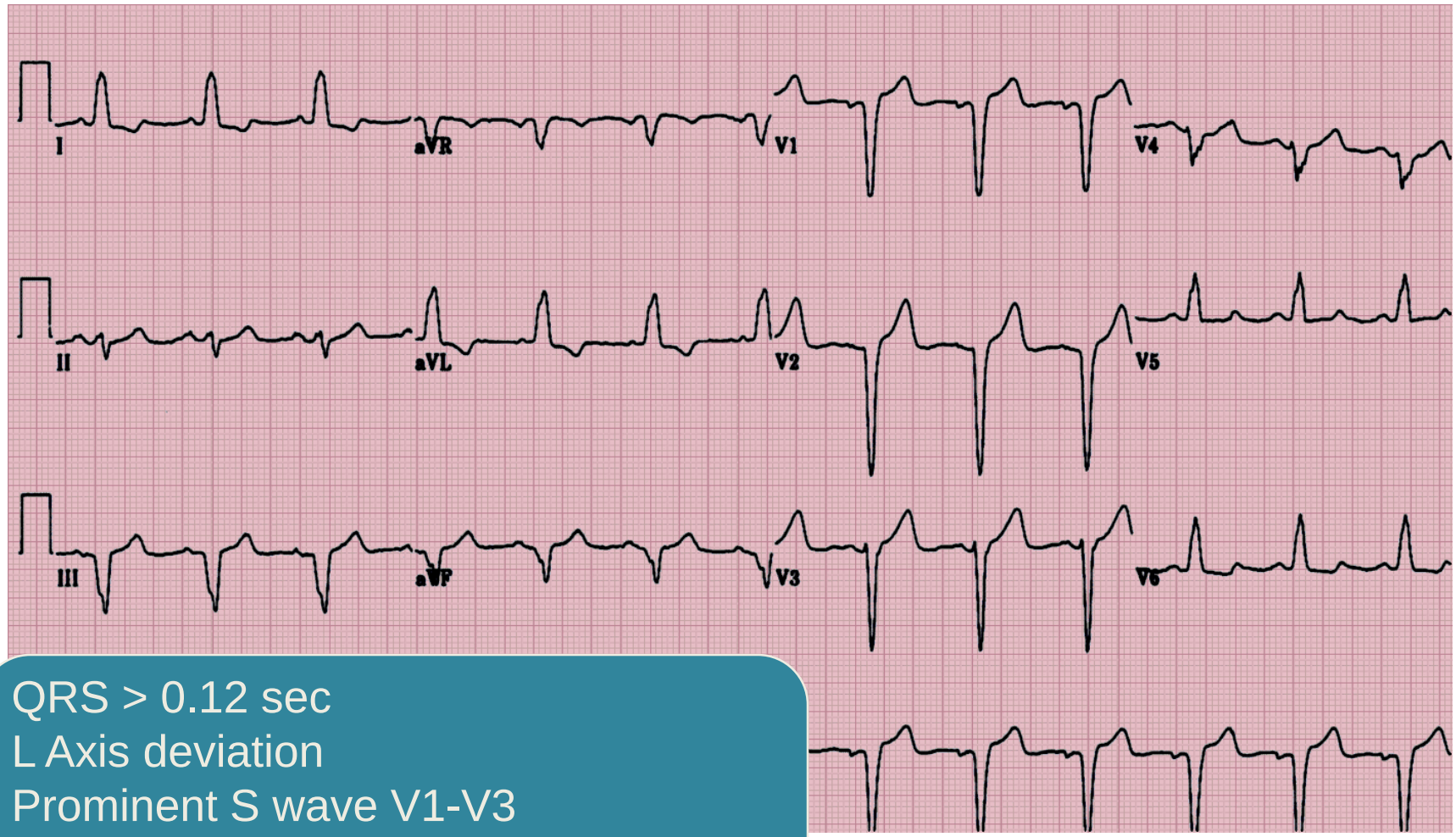
ST Depression or Dynamic T wave Inversions



ST-Segment Elevation MI



New LBBB

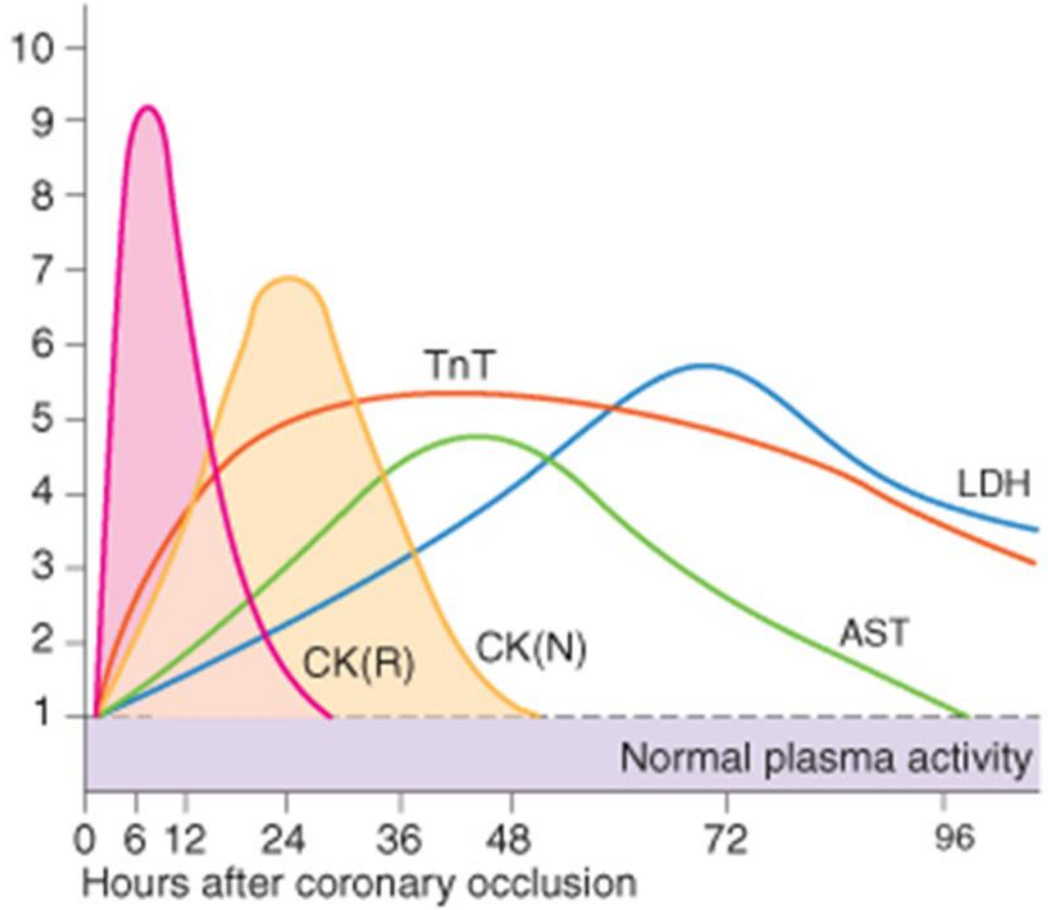


QRS > 0.12 sec
L Axis deviation
Prominent S wave V1-V3
Prominent R wave 1, aVL, V5-V6
with t-wave inversion

Cardiac markers

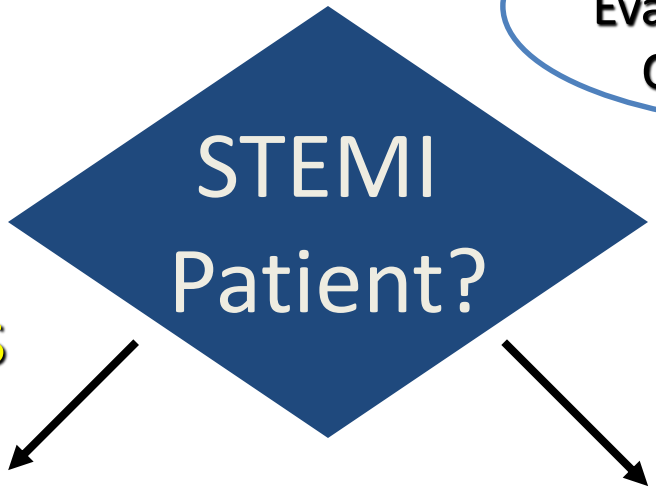
- Troponin (T, I)
  - Very specific and more sensitive than CK
  - Rises 4-8 hours after injury
  - May remain elevated for up to two weeks
  - Can provide prognostic information
  - Troponin T may be elevated with renal dz, poly/dermatomyositis
- CK-MB isoenzyme
  - Rises 4-6 hours after injury and peaks at 24 hours
  - Remains elevated 36-48 hours
  - Positive if CK/MB > 5% of total CK and 2 times normal
  - Elevation can be predictive of mortality
  - False positives with exercise, trauma, muscle dz, DM, PE

Cardiac markers



Risk Stratification

Based on initial Evaluation, ECG, and Cardiac markers



- **Assess for reperfusion**
- **Select & implement reperfusion therapy**
- **Directed medical therapy**

UA or NSTEMI

- Evaluate for Invasive vs. conservative treatment
- Directed medical therapy

Cardiac Care Goals

- Decrease amount of myocardial necrosis
- Preserve LV function
- Prevent major adverse cardiac events
- Treat life threatening complications

STEMI cardiac care

- **STEP 1**: Assessment
  - Time since onset of symptoms
    - 90 min for PCI / 12 hours for fibrinolysis
  - Is this high risk STEMI?
    - KILLIP classification
    - If higher risk may manage with more invasive rx
  - Determine if fibrinolysis candidate
    - Meets criteria with no contraindications
  - Determine if PCI candidate
    - Based on availability and time to balloon rx

Fibrinolysis indications

- ST segment elevation >1mm in two contiguous leads
- New LBBB
- Symptoms consistent with ischemia
- Symptom onset less than 12 hrs prior to presentation

Absolute contraindications for fibrinolysis therapy in patients with acute STEMI

- Any prior ICH
- Known structural cerebral vascular lesion (e.g., AVM)
- 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

STEMI cardiac care

- **STEP 2**: Determine preferred reperfusion strategy

Fibrinolysis preferred if:

- ≤3 hours from onset
- PCI not available/delayed
  - door to balloon > 90min
  - door to balloon minus door to needle > 1hr
- Door to needle goal <30min
- No contraindications

PCI preferred if:

- PCI available
- Door to balloon < 90min
- Door to balloon minus door to needle < 1hr
- Fibrinolysis contraindications
- Late Presentation > 3 hr
- High risk STEMI
  - Killup 3 or higher
- STEMI dx in doubt

Medical Therapy
MONA + BAH

- **Morphine** (class I, level C)
  - Analgesia
  - Reduce pain/anxiety—decrease sympathetic tone, systemic vascular resistance and oxygen demand
  - Careful with hypotension, hypovolemia, respiratory depression
- **Oxygen** (2-4 liters/minute) (class I, level C)
  - Up to 70% of ACS patient demonstrate hypoxemia
  - May limit ischemic myocardial damage by increasing oxygen delivery/reduce ST elevation

- **Nitroglycerin** (class I, level B)
  - Analgesia—titrate infusion to keep patient pain free
  - Dilates coronary vessels—increase blood flow
  - Reduces systemic vascular resistance and preload
  - Careful with recent ED meds, hypotension, bradycardia, tachycardia, RV infarction
- **Aspirin** (160-325mg chewed & swallowed) (class I, level A)
  - Irreversible inhibition of platelet aggregation
  - Stabilize plaque and arrest thrombus
  - Reduce mortality in patients with STEMI
  - Careful with active PUD, hypersensitivity, bleeding disorders

- **Beta-Blockers** (class I, level A)
  - 14% reduction in mortality risk at 7 days at 23% long term mortality reduction in STEMI
  - Approximate 13% reduction in risk of progression to MI in patients with threatening or evolving MI symptoms
  - Be aware of contraindications (CHF, Heart block, Hypotension)
  - Reassess for therapy as contraindications resolve
- **ACE-Inhibitors / ARB** (class I, level A)
  - Start in patients with anterior MI, pulmonary congestion, LVEF < 40% in absence of contraindication/hypotension
  - Start in first 24 hours
  - ARB as substitute for patients unable to use ACE-I

- **Heparin** (class I, level C to class IIa, level C)
  - LMWH or UFH (max 4000u bolus, 1000u/hr)
    - Indirect inhibitor of thrombin
    - less supporting evidence of benefit in era of reperfusion
    - Adjunct to surgical revascularization and thrombolytic / PCI reperfusion
    - 24-48 hours of treatment
    - Coordinate with PCI team (UFH preferred)
    - Used in combo with aspirin and/or other platelet inhibitors
    - Changing from one to the other not recommended

Additional medication therapy

- **Clopidodrel** (class I, level B)
  - Irreversible inhibition of platelet aggregation
  - Used in support of cath / PCI intervention or if unable to take aspirin
  - 3 to 12 month duration depending on scenario
- **Glycoprotein IIb/IIIa inhibitors**
  (class IIa, level B)
  - Inhibition of platelet aggregation at final common pathway
  - In support of PCI intervention as early as possible prior to PCI

Additional medication therapy

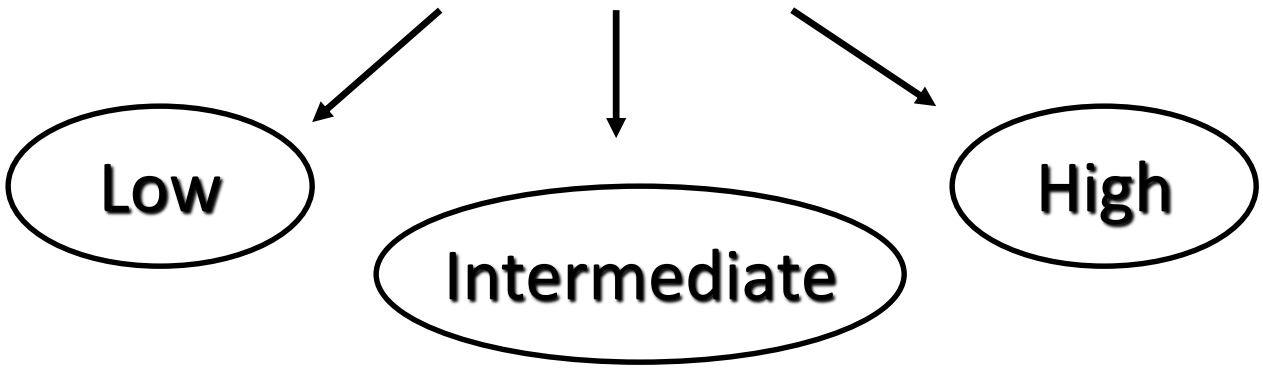
- **Aldosterone blockers** (class I, level A)
  - Post-STEMI patients
    - no significant renal failure (cr < 2.5 men or 2.0 for women)
    - No hyperkalemis > 5.0
    - LVEF < 40%
    - Symptomatic CHF or DM

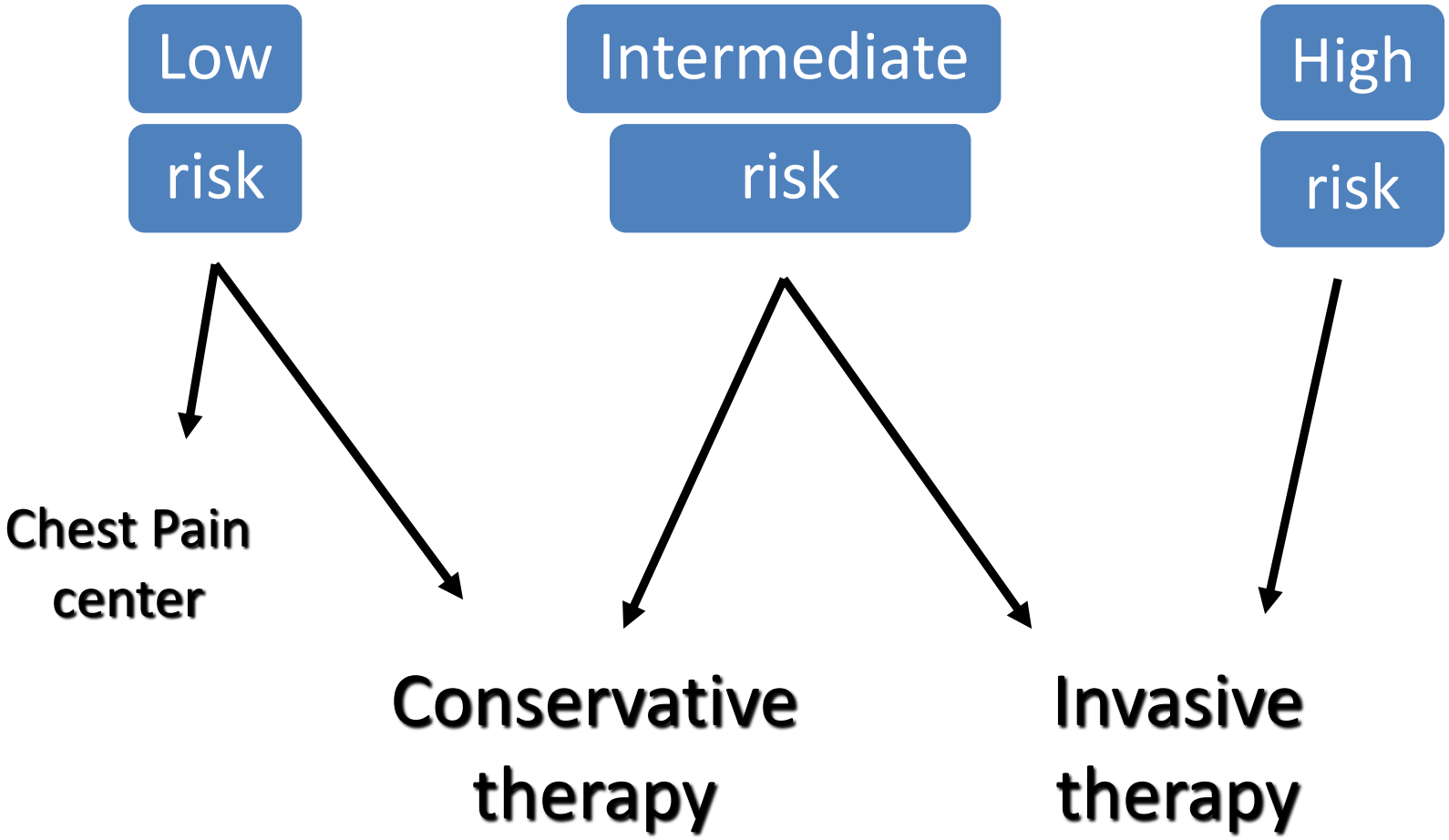
STEMI care CCU

- Monitor for complications:
  - recurrent ischemia, cardiogenic shock, ICH, arrhythmias
- Review guidelines for specific management of complications & other specific clinical scenarios
  - PCI after fibrinolysis, emergent CABG, etc…
- Decision making for risk stratification at hospital discharge and/or need for CABG

Unstable angina/NSTEMI cardiac care

- Evaluate for conservative vs. invasive therapy based upon:
  - Risk of actual ACS
  - TIMI risk score
  - ACS risk categories per AHA guidelines




Low
risk
Intermediate
risk
High
risk
Chest Pain
center
Conservative
therapy
Invasive
therapy

Invasive therapy option
UA/NSTEMI

- Coronary angiography and revascularization within 12 to 48 hours after presentation to ED
- For high risk ACS (class I, level A)
- MONA + BAH *(UFH)*
- Clopidogrel
  - 20% reduction death/MI/Stroke – CURE trial
  - 1 month minimum duration and possibly up to 9 months
- Glycoprotein IIb/IIIa inhibitors

Conservative Therapy for UA/NSTEMI

- Early revascularization or PCI not planned
- MONA + BAH *(LMW or UFH)*
- Clopidogrel
- Glycoprotein IIb/IIIa inhibitors
  - Only in certain circumstances (planning PCI, elevated TnI/T)
- Surveillence in hospital
  - Serial ECGs
  - Serial Markers

Secondary Prevention

- Disease
  - HTN, DM, HLP
- Behavioral
  - smoking, diet, physical activity, weight
- Cognitive
  - Education, cardiac rehab program

Secondary Prevention disease management

- Blood Pressure
  - Goals < 140/90 or <130/80 in DM /CKD
  - Maximize use of beta-blockers & ACE-I
- Lipids
  - LDL < 100 (70) ; TG < 200
  - Maximize use of statins; consider fibrates/niacin first line for TG>500; consider omega-3 fatty acids
- Diabetes
  - A1c < 7%

Secondary prevention behavioral intervention

- Smoking cessation
  - Cessation-class, meds, counseling
- Physical Activity
  - Goal 30 - 60 minutes daily
  - Risk assessment prior to initiation
- Diet
  - DASH diet, fiber, omega-3 fatty acids
  - <7% total calories from saturated fats

Thank You