Case-based Approach: Ischemic Heart Disease

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History

- A 68-year-old man presents to his new primary care provider for an initial patient visit. His sole active complaint is mild retrosternal chest pressure that he experiences only after significant exertion that resolves within minutes of stopping. The pattern and severity of his symptoms have not changed significantly over the last 3 years. He denies any episodes of chest pain at rest, dizziness, syncope, dyspnea, palpitations, or lower extremity edema.

History

PMH:

- CAD – s/p PCI with BMS of the RCA ~5 years ago
- Hyperlipidemia
- HTN

Meds:

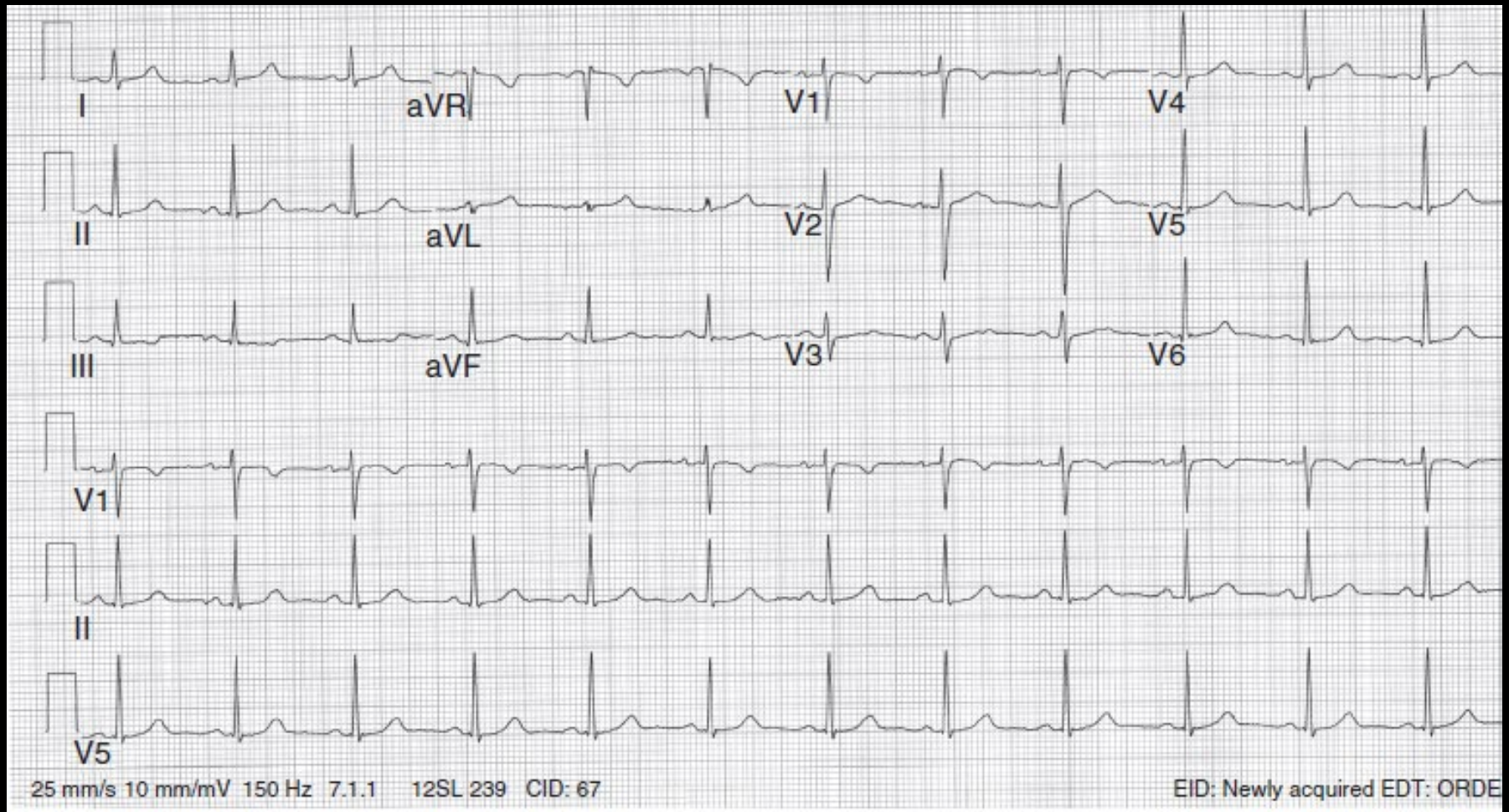
- Aspirin 81 mg QD
- Metoprolol succinate 25 mg QD
- HCTZ 12.5 mg QD
- Simvastatin 10 mg QD

He is a lifelong nonsmoker and does not consume alcohol or use illicit drugs.

Px Exam

- Vitals – BP 155/85 HR 90
- Gen'l – NAD
- Neck – Normal JVP. 2+ carotids without murmur or bruit.
- Lungs – CTA B
- Heart – RRR. Normal S1. Physiologically split S2. No murmurs, rubs or gallops.
- Ext – No LE edema. 2+ pedal pulses bilaterally.

- ECG



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Questions

- Differential diagnosis?
- Diagnostic work-up?
- Management?

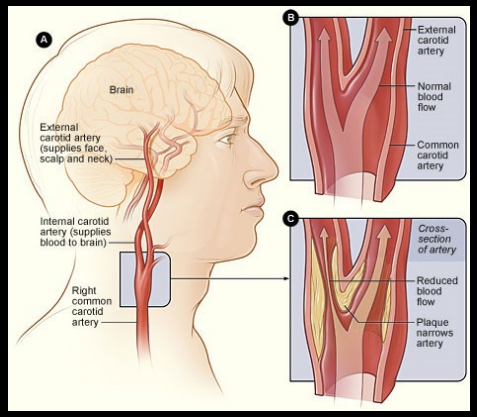
DDx

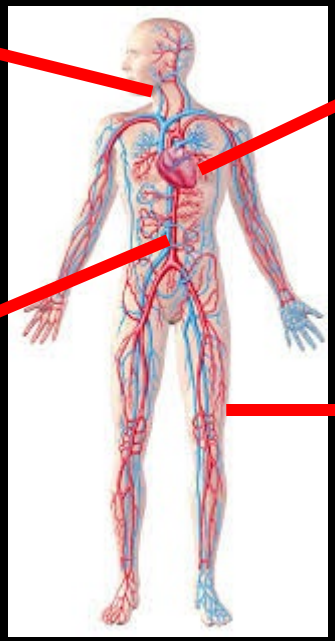
Cardiovascular	Pulmonary	GI	MSK	Miscellaneous
Pericardium Pericarditis Myocardium Myocarditis Heart failure exacerbation Hypertrophic cardiomyopathy Takotsubo cardiomyopathy Valves Aortic stenosis Conduction system Tachyarrhythmias Vessels Acute coronary syndrome Aortic dissection Hypertensive emergency	Pleura Pleuritis (a.k.a. pleurisy) Pneumothorax Airways Asthma exacerbation Alveoli Pneumonia Vessels Pulmonary embolism Pulmonary hypertension Lung cancer	Esophagus GERD Esophagitis Esophageal spasm Stomach Gastritis Peptic ulcer disease	Rib fractures Costochondritis	Severe anemia Herpes zoster (a.k.a. shingles) Acute intoxication with cocaine or amphetamines Acute chest syndrome in sickle cell anemia Psychiatric Panic attack Somatization

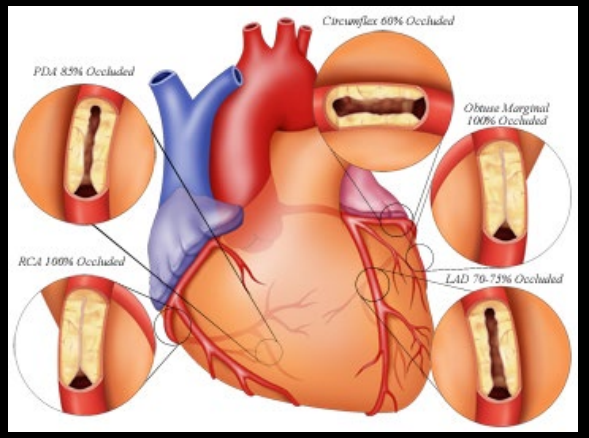
Atherosclerosis: Inflammatory Hypothesis

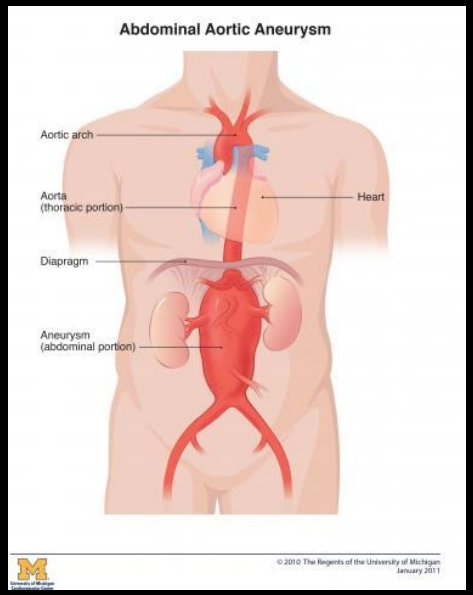
- *Response to injury hypothesis*

- **Atherosclerosis is considered to be a chronic inflammatory response of the arterial wall initiated by the injury to the endothelium**


A
Brain
External carotid artery (supplies face, scalp and neck)
Internal carotid artery (supplies blood to brain)
Right common carotid artery
B
External carotid artery
Normal blood flow
Common carotid artery
C
Cross-section of artery
Reduced blood flow
Plaque narrows artery




Circumflex 60% Occluded
PDA 85% Occluded
Obtuse Marginal 100% Occluded
RCA 100% Occluded
LAD 70-75% Occluded


Abdominal Aortic Aneurysm
Aortic arch
Aorta (thoracic portion)
Heart
Diapragm
Aneurysm (abdominal portion)
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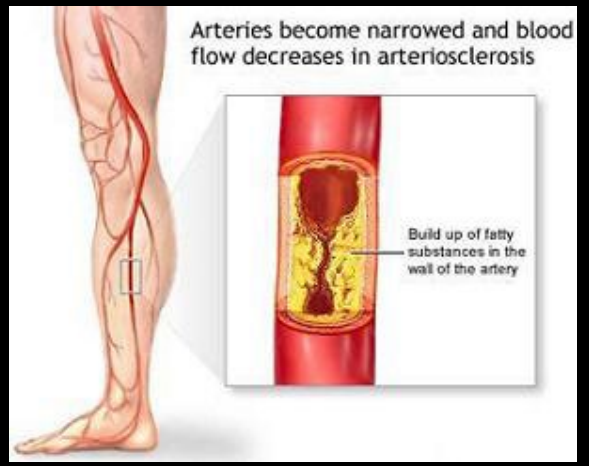
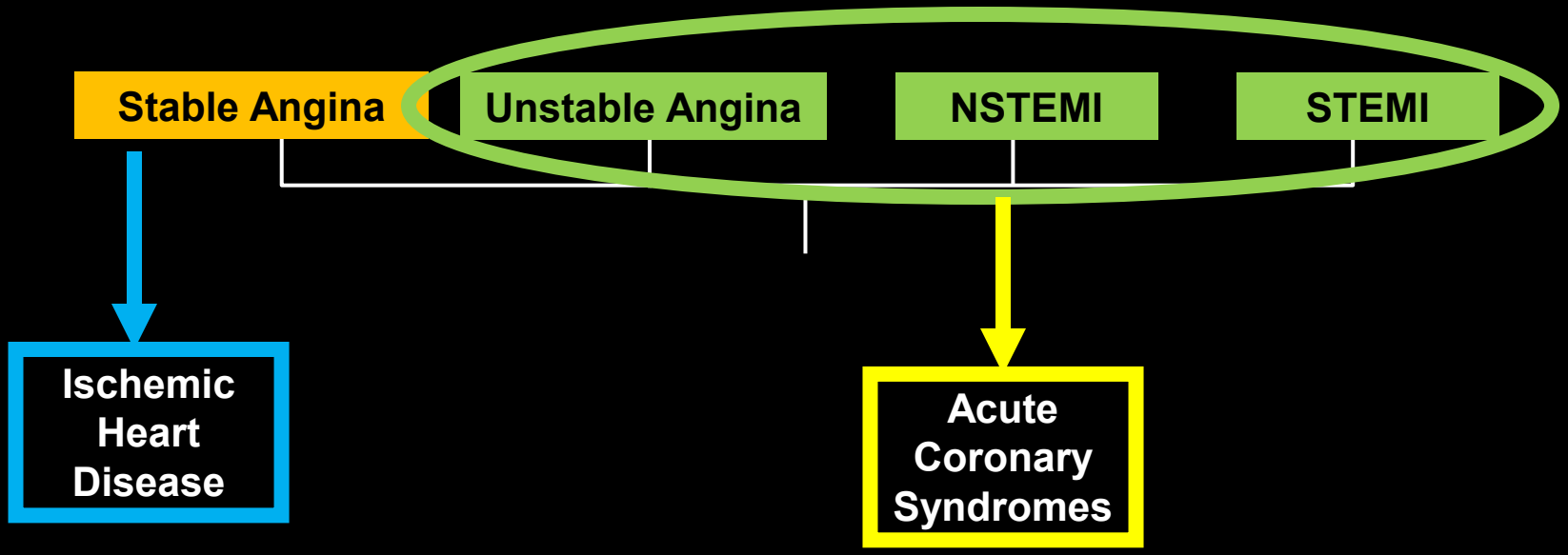
January 2011


Arteries become narrowed and blood flow decreases in arteriosclerosis
Build up of fatty substances in the wall of the artery

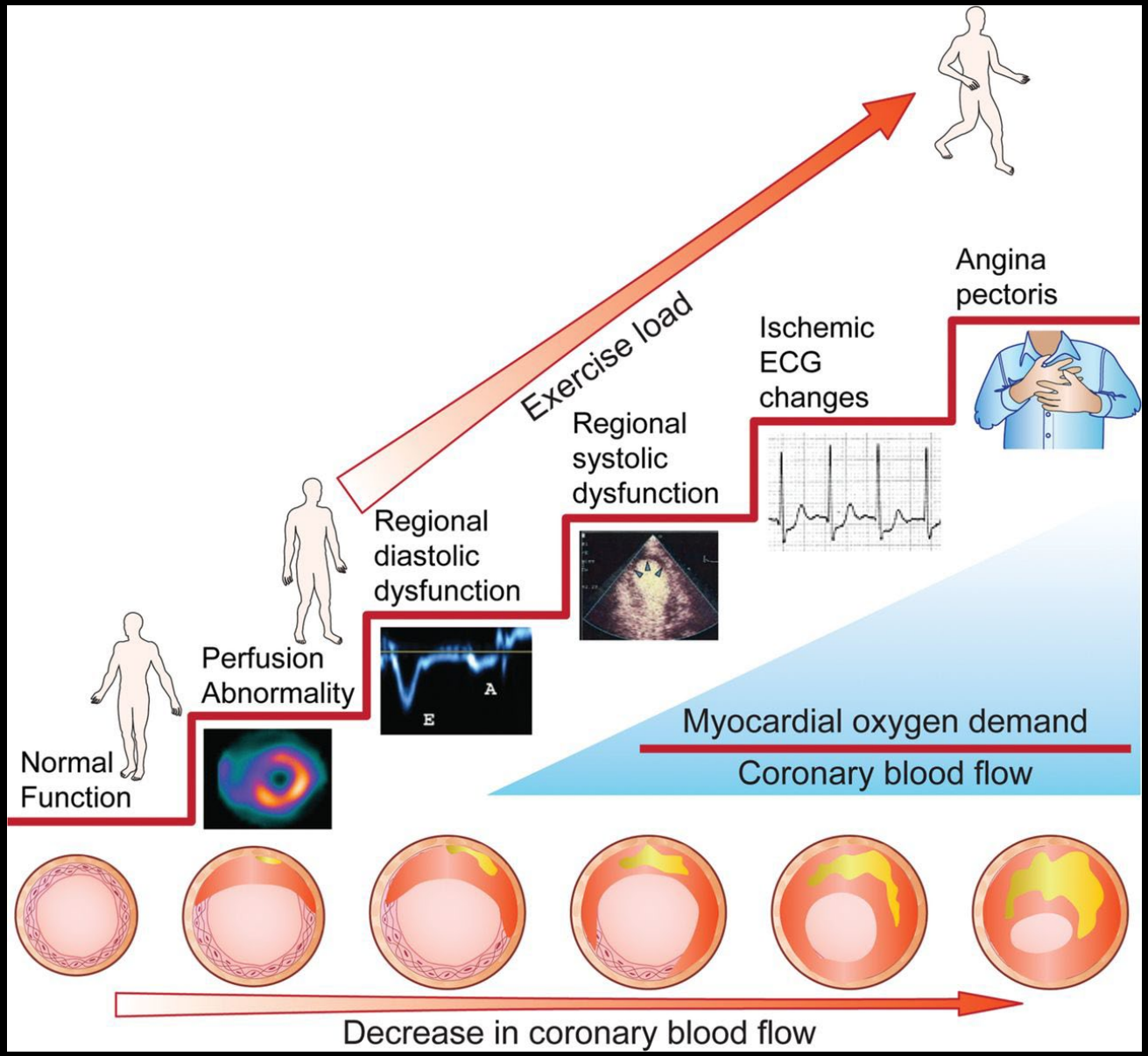
Spectrum of Ischemic Heart Disease

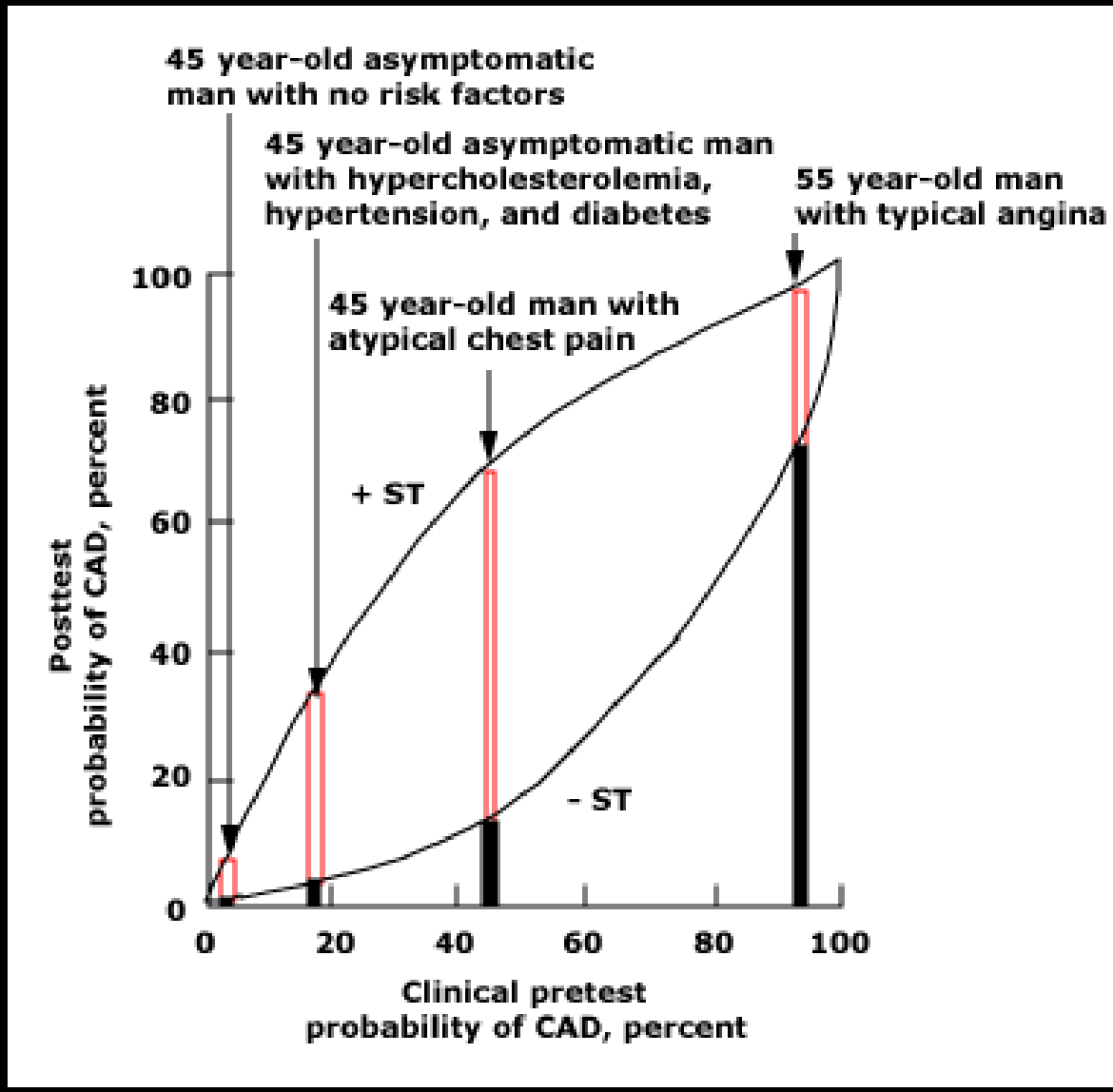


Stress Testing

- **Type of Stress**
  - A. Exercise – Treadmill
  - B. Pharmacologic – Vasodilator or Dobutamine

- **Imaging Modality to detect ischemia**


Exercise load
Normal Function
Perfusion Abnormality
Regional diastolic dysfunction
Regional systolic dysfunction
Ischemic ECG changes
Angina pectoris
A
E
Myocardial oxygen demand
Coronary blood flow
Decrease in coronary blood flow


45 year-old asymptomatic man with no risk factors
45 year-old asymptomatic man with hypercholesterolemia, hypertension, and diabetes
55 year-old man with typical angina
45 year-old man with atypical chest pain
+ ST
- ST
Posttest probability of CAD, percent
Clinical pretest probability of CAD, percent
0
20
40
60
80
100

- **The result of the exercise stress test is *NOT* dichotomous, i.e., positive or negative.**

- **Rather, the test provides useful diagnostic and prognostic information.**

  Extent of exercise **<4 METs vs. ≥10 METs**

  BP and HR response

  Chest discomfort

  Duke treadmill score

  Arrhythmias

  O2 saturation

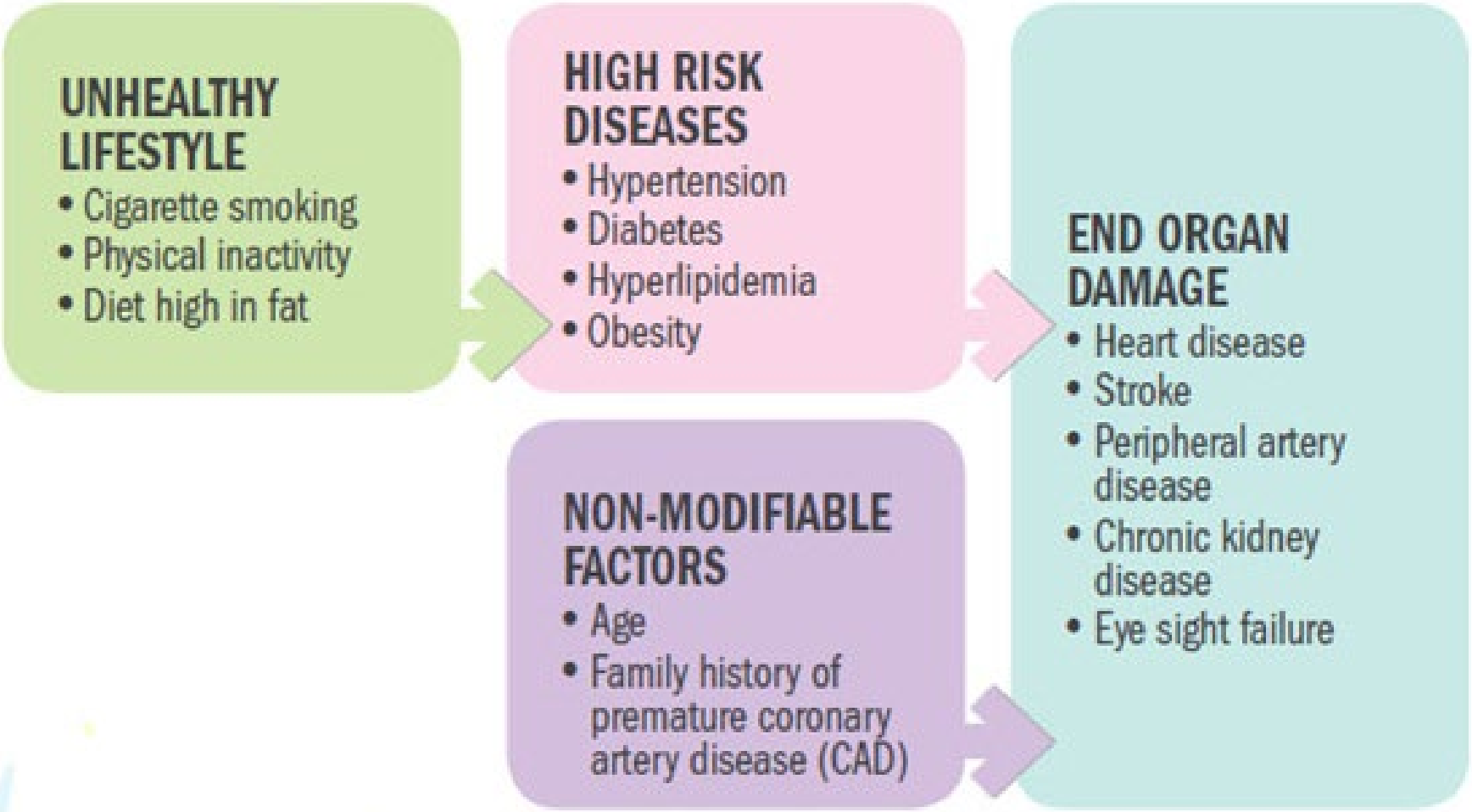
  Pulmonary findings – wheezes

- **Does not predict the risk for an MI**

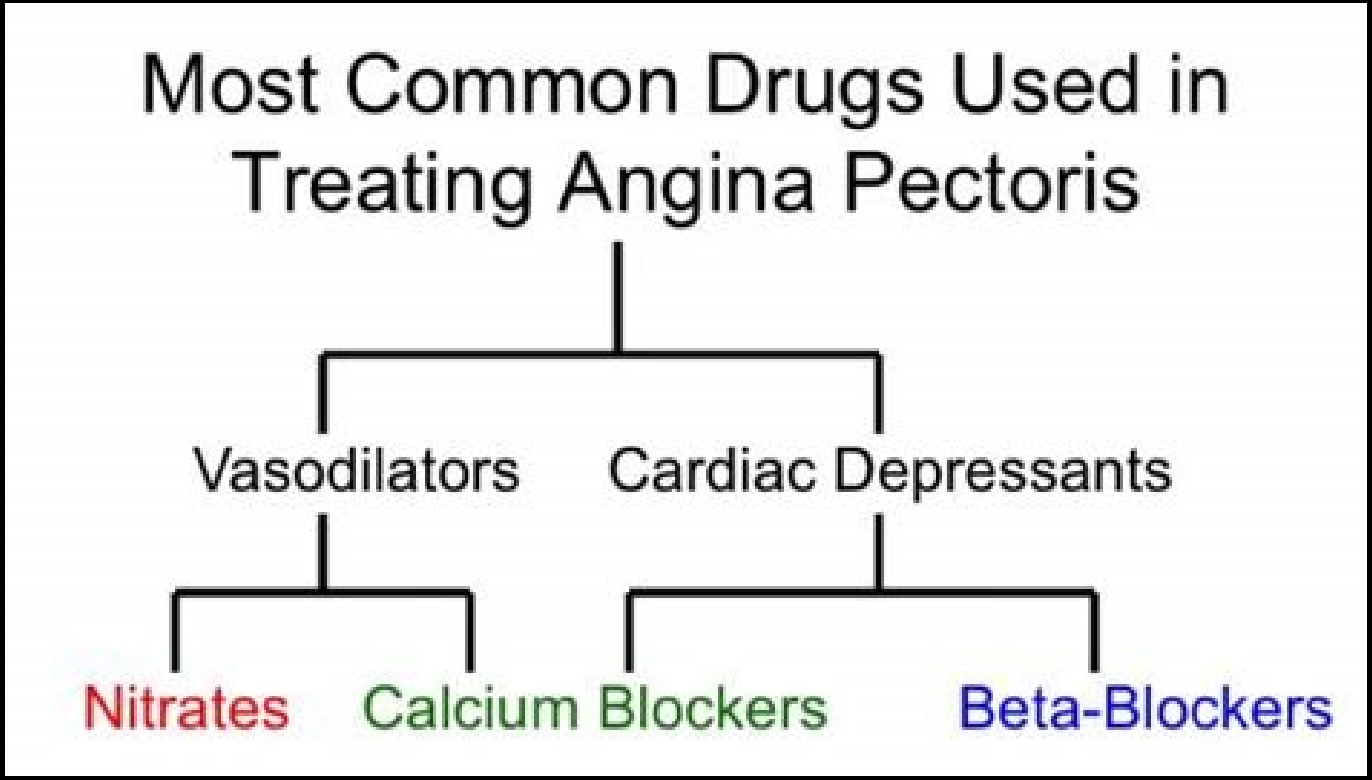
Management

- Lifestyle modification
- Medications
  - Anti-anginal
  - Disease modifying – Aspirin & Statin therapy
- Percutaneous revascularization
- Surgical revascularization

Major risk factors for cardiovascular disease

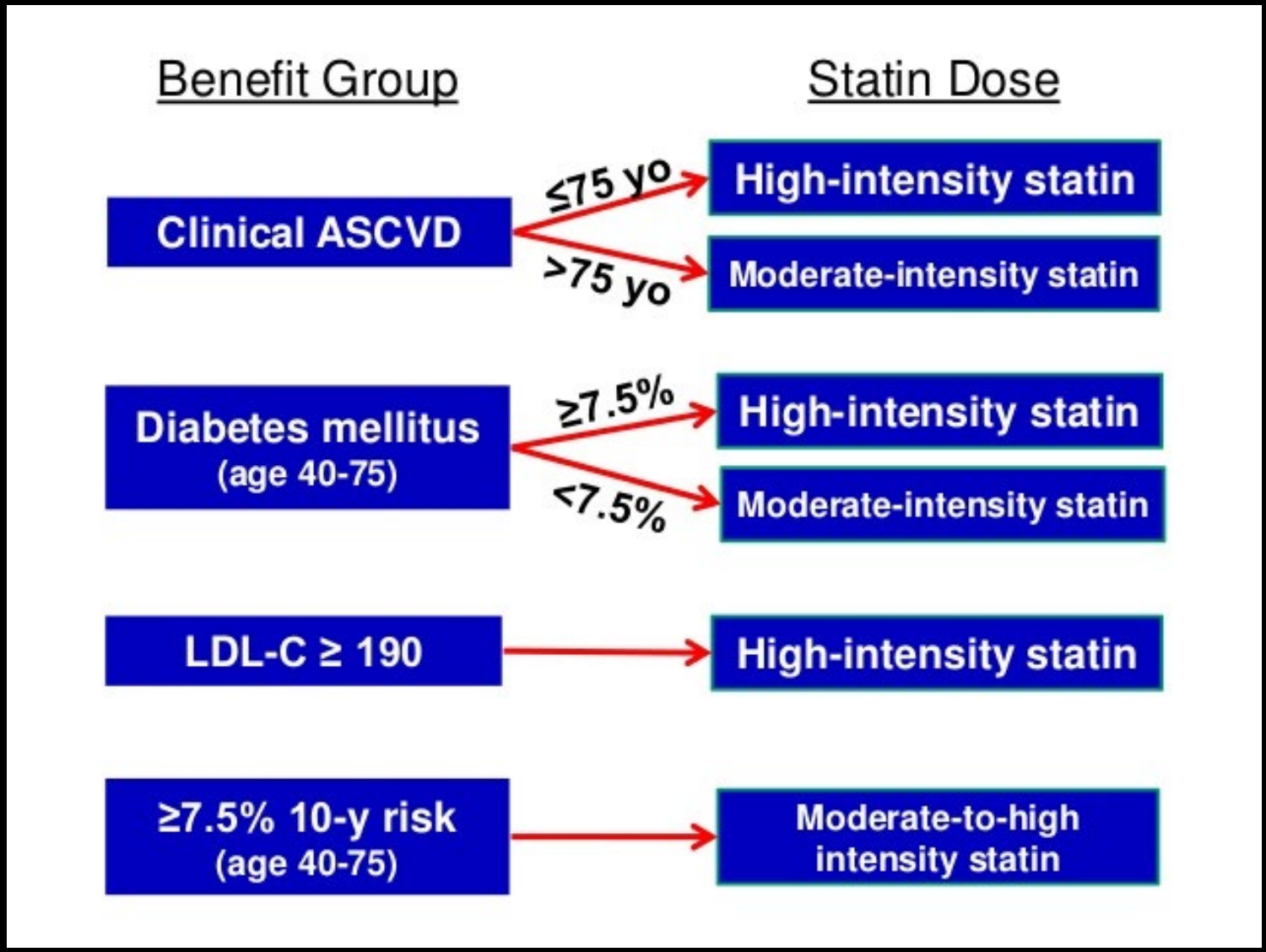


Anti-anginal Therapy



Disease Modifying (Event Prevention)

- Aspirin
- Statin
- Consider ACE-I



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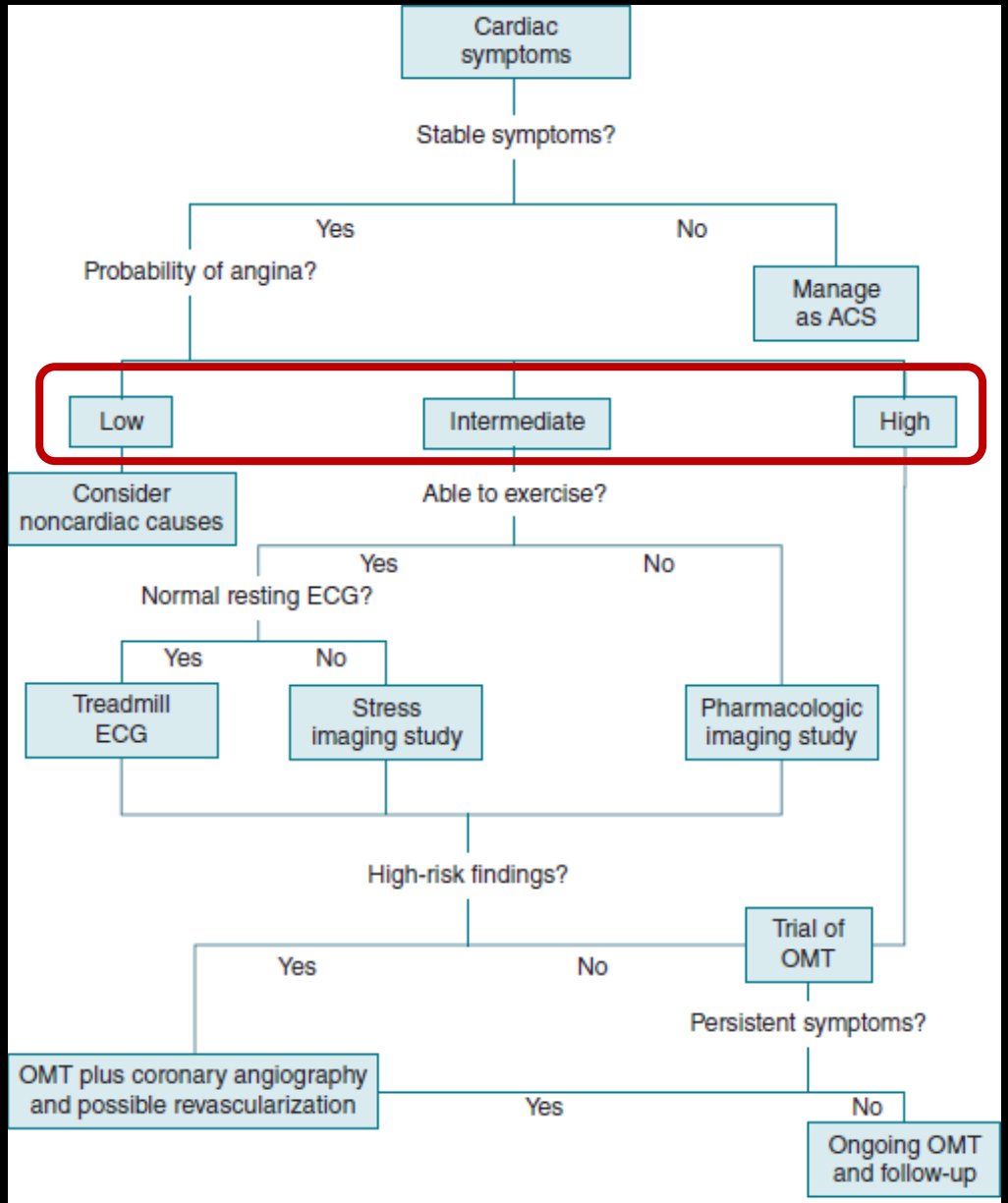
Optimal Medical Therapy vs. Revascularization

- Based on several randomized clinical trials, optimal medical therapy is as efficacious as PCI plus optimal medical therapy in reducing the risk of nonfatal myocardial infarction, other cardiovascular events, and death in patients with stable ischemic heart disease.



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Clinical Pearls

- Exercise stress testing is the preferred testing modality if the resting ECG is interpretable and the patient is able to exercise.
- Evaluate the patient with stable angina for signs and symptoms of left ventricular dysfunction, as this may suggest the presence of a high-risk epicardial coronary artery obstruction.
- Optimal medical therapy is an accepted initial management strategy in chronic stable angina without high-risk features on noninvasive testing.
- Titrate beta-blockers, nitrates, and calcium channel antagonists to the maximally tolerated dosages to alleviate anginal symptoms during exertion.

History

73 year old woman presents to the ER with episodic, burning precordial chest pain occurring at rest. The pain began several hours ago while she was taking a shower and has been intermittent since. She initially attributed it to indigestion and took antacids without relief.

PMH:

- CAD (s/p PCI with DES – approx. 3 years ago)
- HTN
- Hyperlipidemia

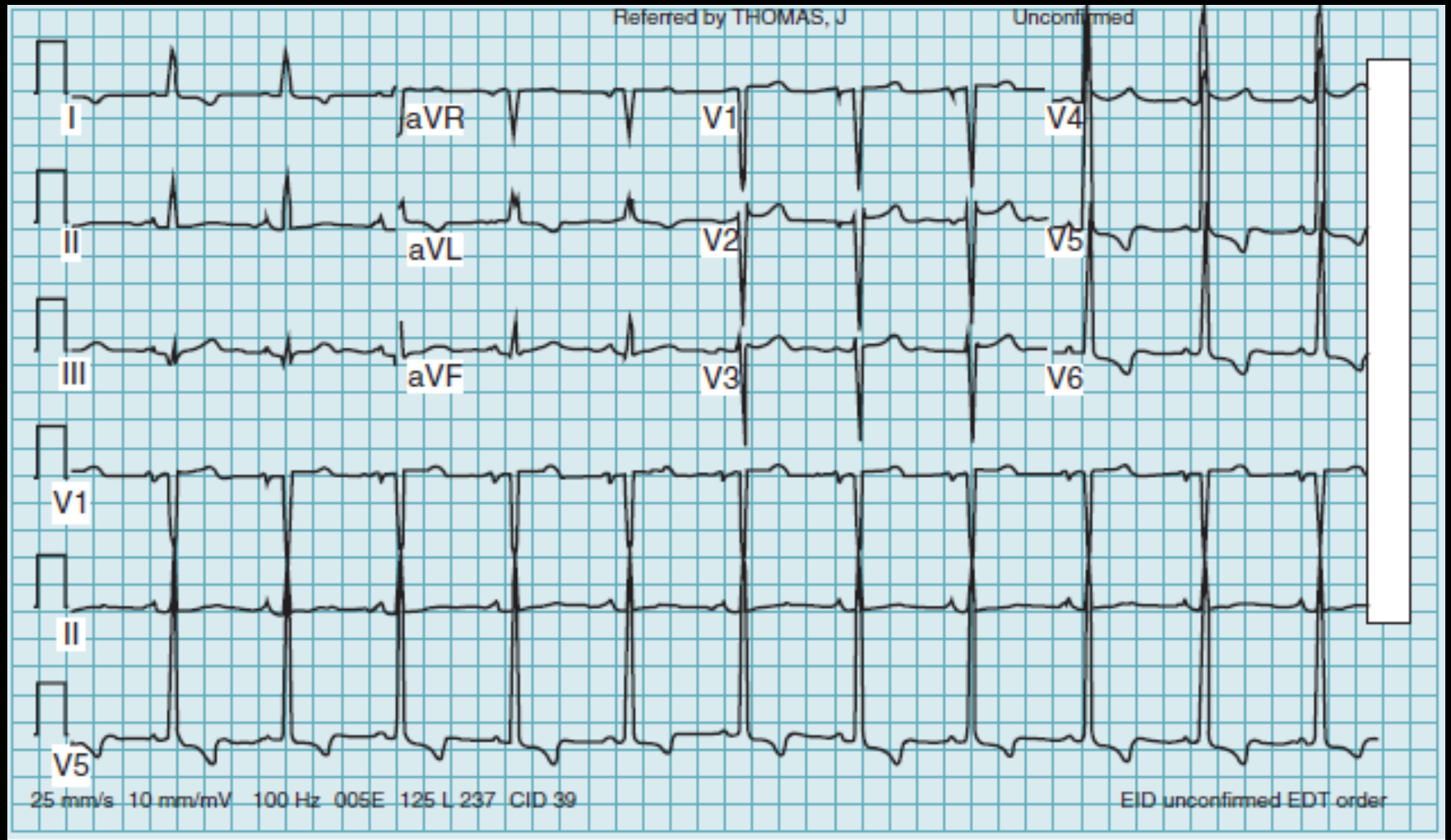
Meds:

- Aspirin 81 mg QD
- Lisinopril 10 mg QD
- Atorvastatin 40 mg QD

Px Exam

- Vitals – BP 168/94 HR 95 RR 20 RA ox sat 99%
- Gen'l – NAD
- Neck – Normal JVP. 2+ carotids without murmur or bruit.
- Lungs – CTA B
- Heart – RRR. Normal S1. Physiologically split S2. Soft systolic murmur at the RUSB. No gallops
- Ext – No LE edema. 2+ pedal pulses bilaterally.

- ECG
- Troponin +



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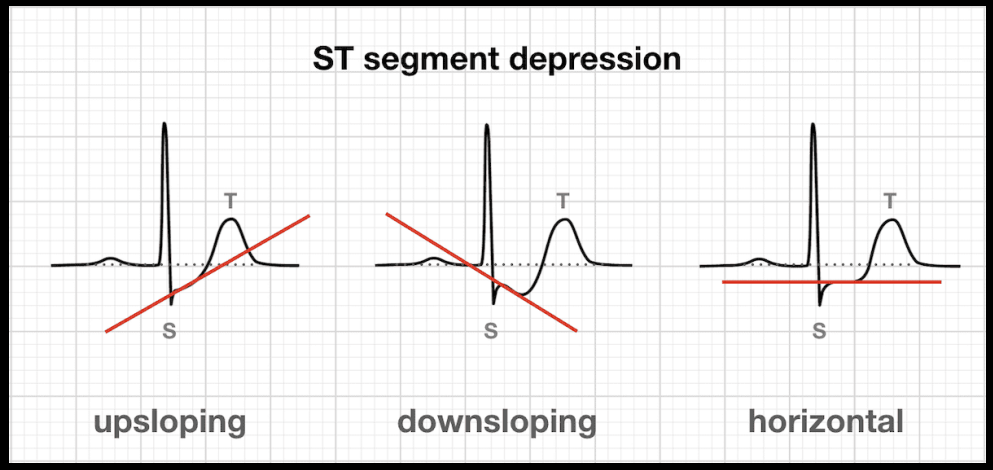
Differential of ST Depression

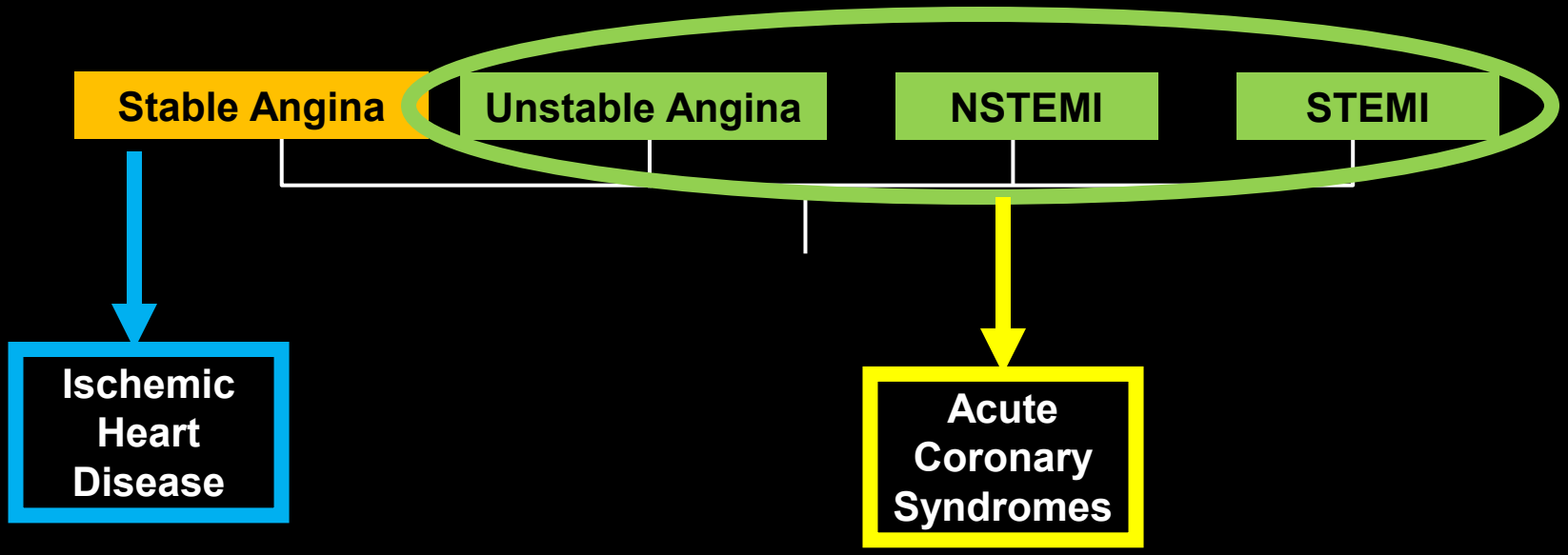


- Subendocardial ischemia
- LVH or enlargement
- Hypokalemia
- Bundle branch blocks
- Reciprocal changes in setting of MI
- Rate-related
- Neurologic
- Non-specific

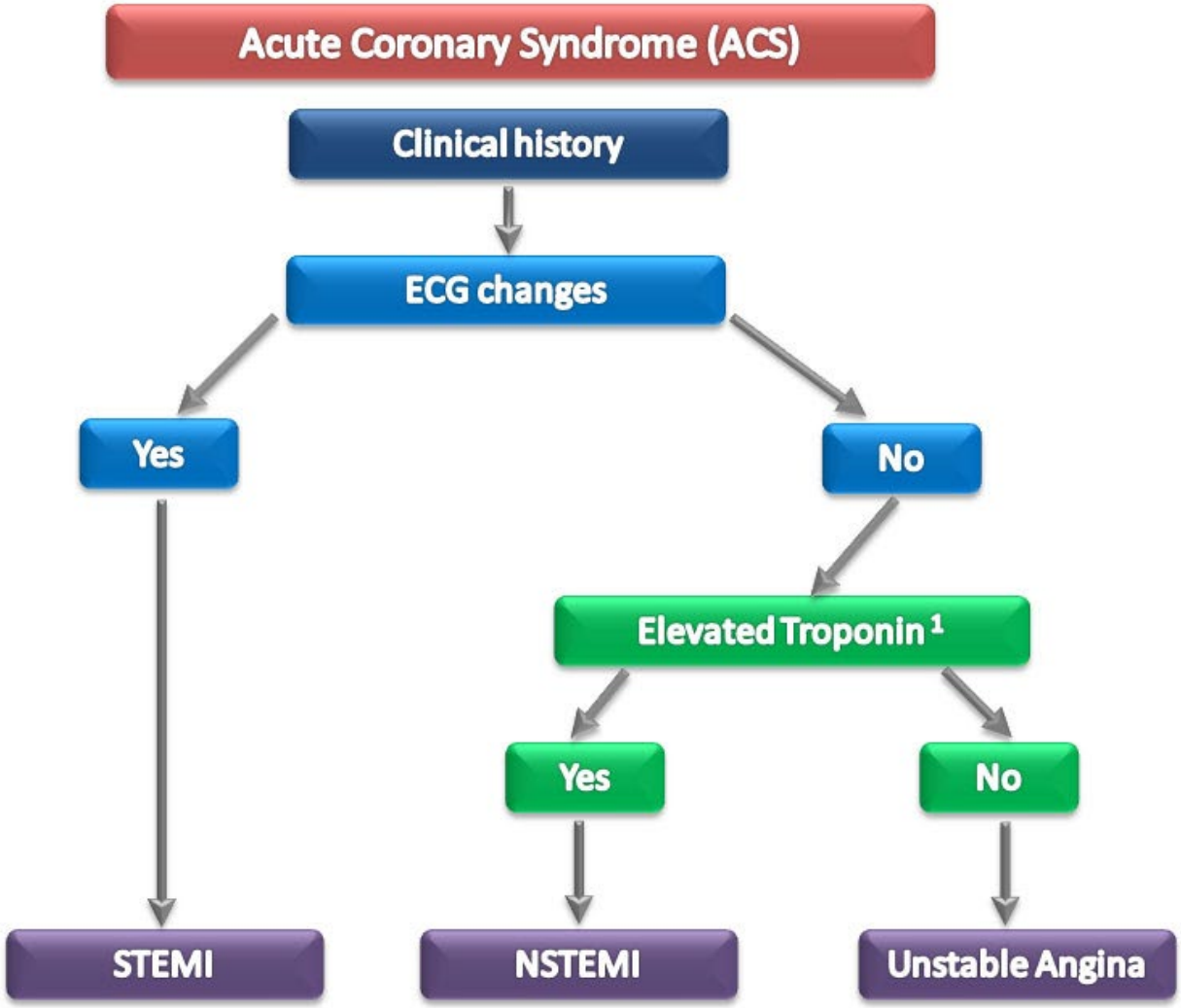
Questions

- Differential diagnosis?
- Diagnostic work-up?
- Management?

Spectrum of Ischemic Heart Disease



Pathophysiology



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TYPE 1 MYOCARDIAL INFARCTION

Spontaneous myocardial infarction related to ischaemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring or dissection

TYPE 2 MYOCARDIAL INFARCTION

Myocardial infarction secondary to ischaemia due to either increased oxygen demand or decreased supply

TYPE 3 MYOCARDIAL INFARCTION

Sudden unexpected cardiac death often with symptoms suggestive of myocardial ischaemia

TYPE 4 MYOCARDIAL INFARCTION

Myocardial infarction associated with percutaneous coronary intervention (4a) or stent thrombosis (4b)

TYPE 5 MYOCARDIAL INFARCTION

Myocardial infarction associated with cardiac surgery

MYOCARDIAL INJURY

Multifactorial aetiology; acute or chronic based on change in cardiac troponin concentrations with serial testing

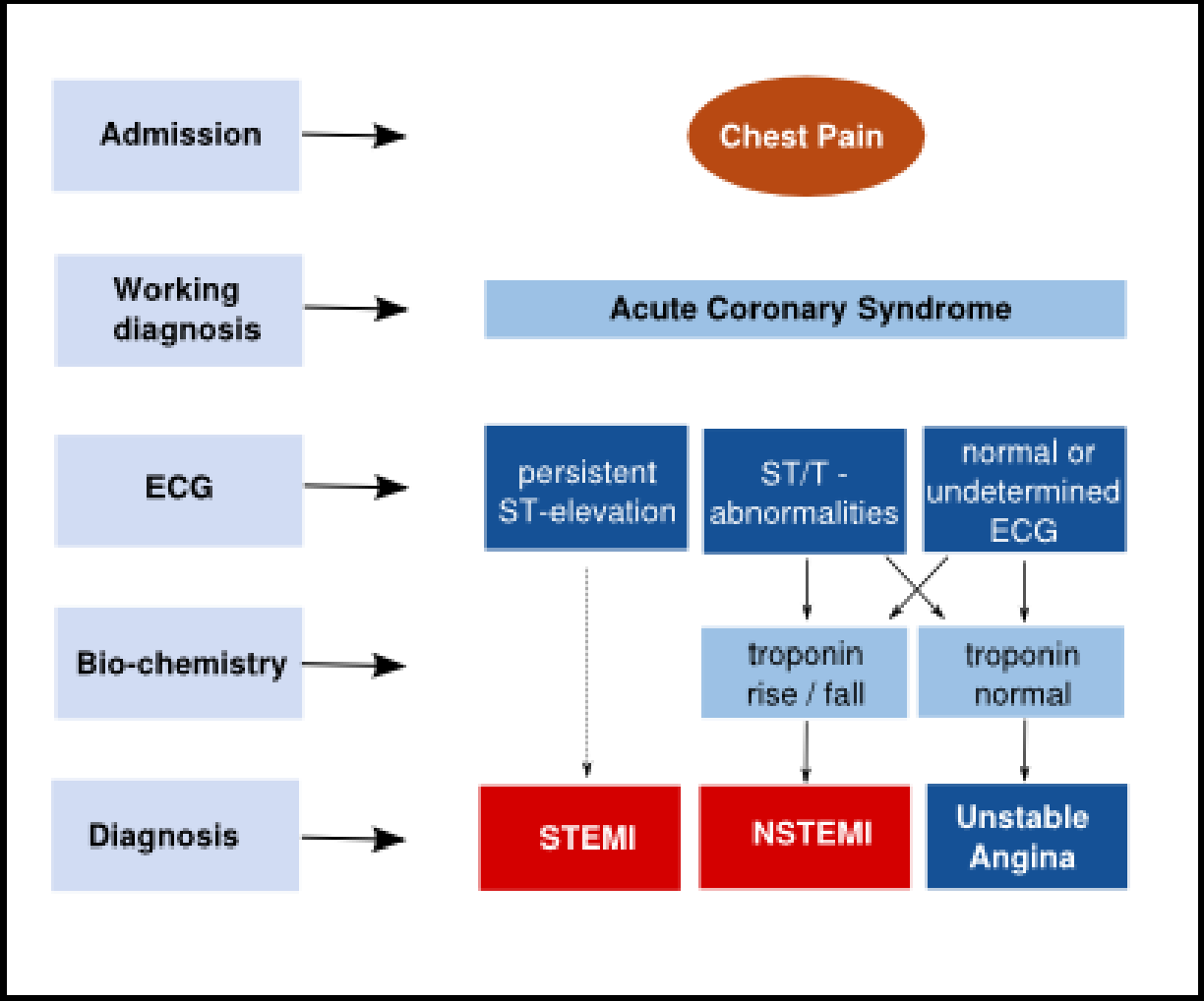
Clinical Presentation

- Patients with UA/NSTEMI are *generally older and have more cardiac risk factors* compared to patients with STEMI.
- Also, more likely to have a *prior history of MI or undergone coronary revacularization.*
- Patients with NSTEMI or UA can have a *variable presentation* from recent-onset angina to progressively worsening angina within the past 48 hours to resting anginal pain (>20 minutes).
- Pain from UA/NSTEMI is *more severe and of longer duration* when compared to stable angina.
- NSTEMI differentiated from UA by presence of + troponin.

Clinical Presentation

- Associated symptoms include SOB, diaphoresis, palpitations, nausea, or vomiting.
- Diabetic patients, females, or the elderly may present without chest pain.
- The physical exam seldom adds to the diagnosis of UA/NSTEMI.
- Documentation of the baseline physical exam is very important to recognize potential complications that may arise during their hospital stay, particularly the presence or absence of cardiac murmurs.

Management Overview



Management Overview

Initial treatment goals include:

1. **Alleviation of ischemic pain**
2. **Optimization of hemodynamics**
3. **Risk stratification**
4. **Choose a management strategy**
5. **Initiation of antithrombotic therapy.**

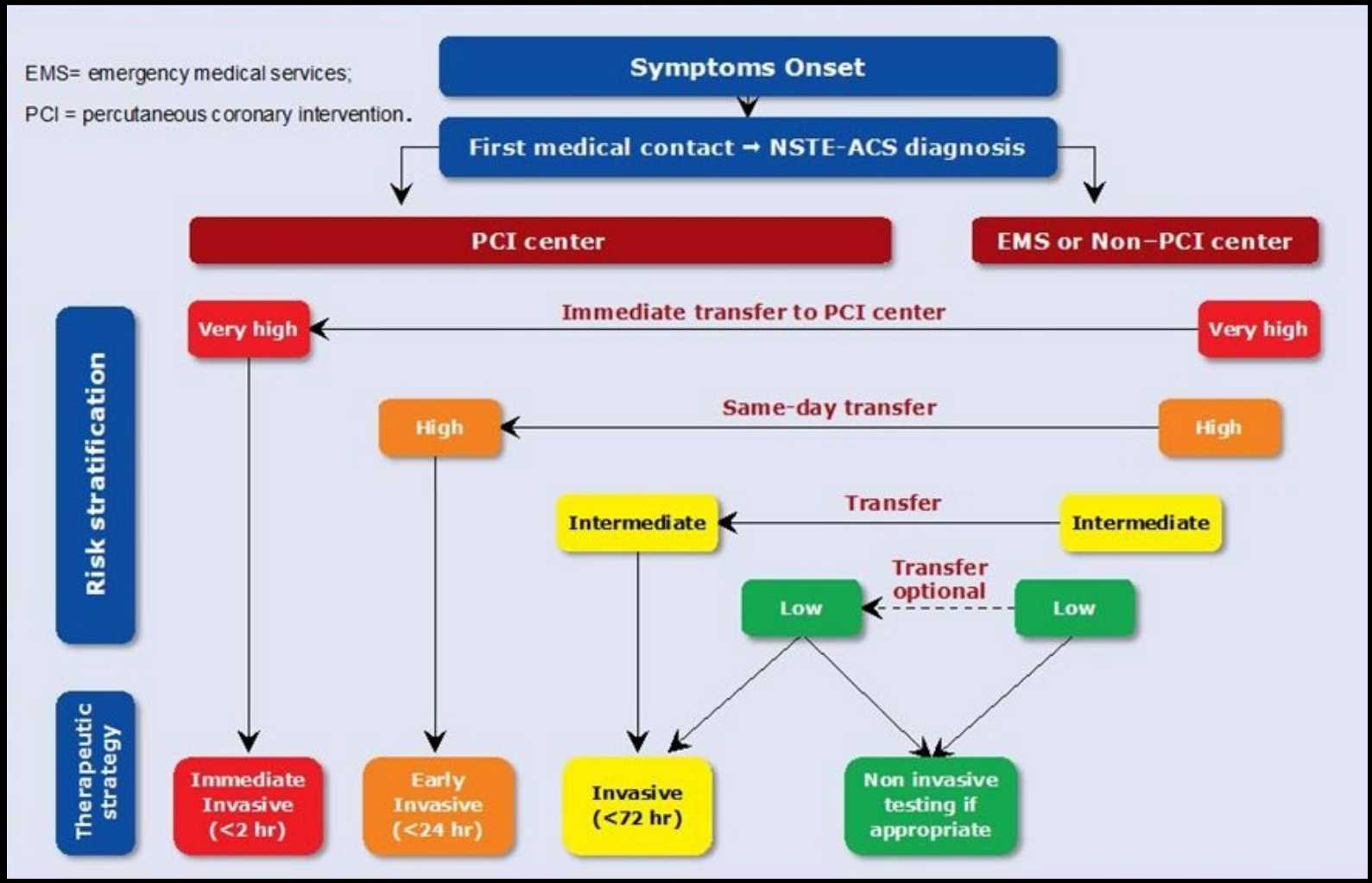
Initial Management

- Aspirin 325 mg chewed
- Anticoagulation – unfractionated heparin, enoxaparin or bivalirudin (preferred because of lower bleeding risk)
- Nitrates +/- morphine
- High-dose statin
- If invasive strategy, clopidrogrel, ticagrelor or prasugrel
- Beta-blockers should be started as soon as feasible and maintained indefinitely

Mnemonic Letter	Treatment
M	Morphine
O	Oxygen
N	Nitrates
A	Aspirin
R	Reperfusion (thrombolysis or primary PCI)
C	Clopidogrel (or prasugrel)
H	Heparin
B	Beta-blocker
A	Anticoagulants (aspirin and clopidogrel)
S	Statin
I	Inhibitors of angiotensin II (ACEi or A2R blocker)
C	Correction of risk factors

PCI = percutaneous coronary intervention; ACEi = angiotensin-converting enzyme inhibitor; A2R = angiotensin 2 receptor.

Management Overview

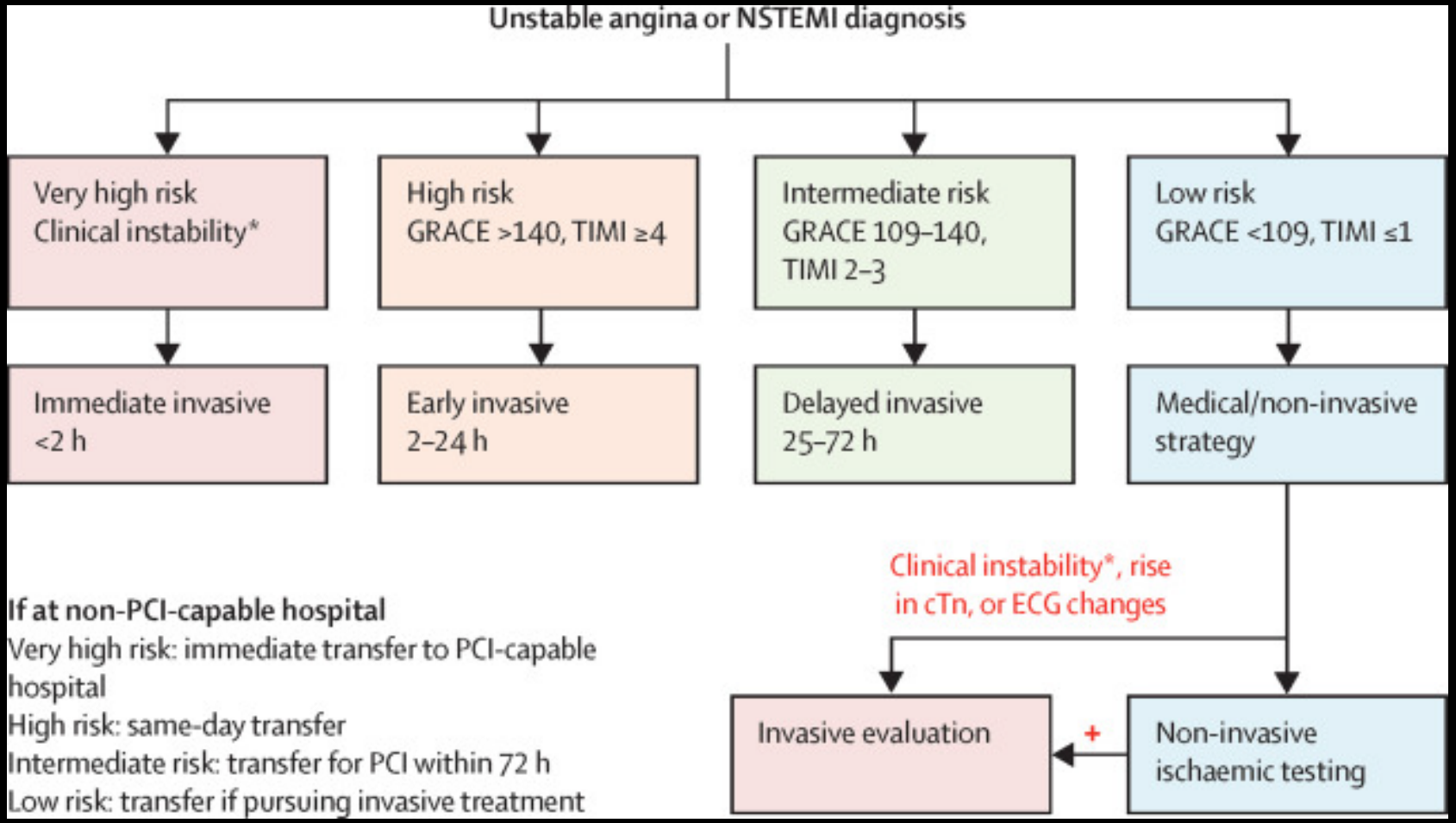


Initial Invasive vs. Conservative Strategy

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 less than 40%)
Conservative	Low risk score (e.g., TIMI, GRACE)
	Patient or physician preference in the absence of high-risk features

CABG = coronary artery bypass graft surgery; GRACE = Global Registry of Acute Coronary Events; HF = heart failure; LVEF = left ventricular ejection fraction; PCI = percutaneous coronary intervention; TIMI = Thrombolysis In Myocardial Infarction; TnI = troponin I; TnT = troponin T.

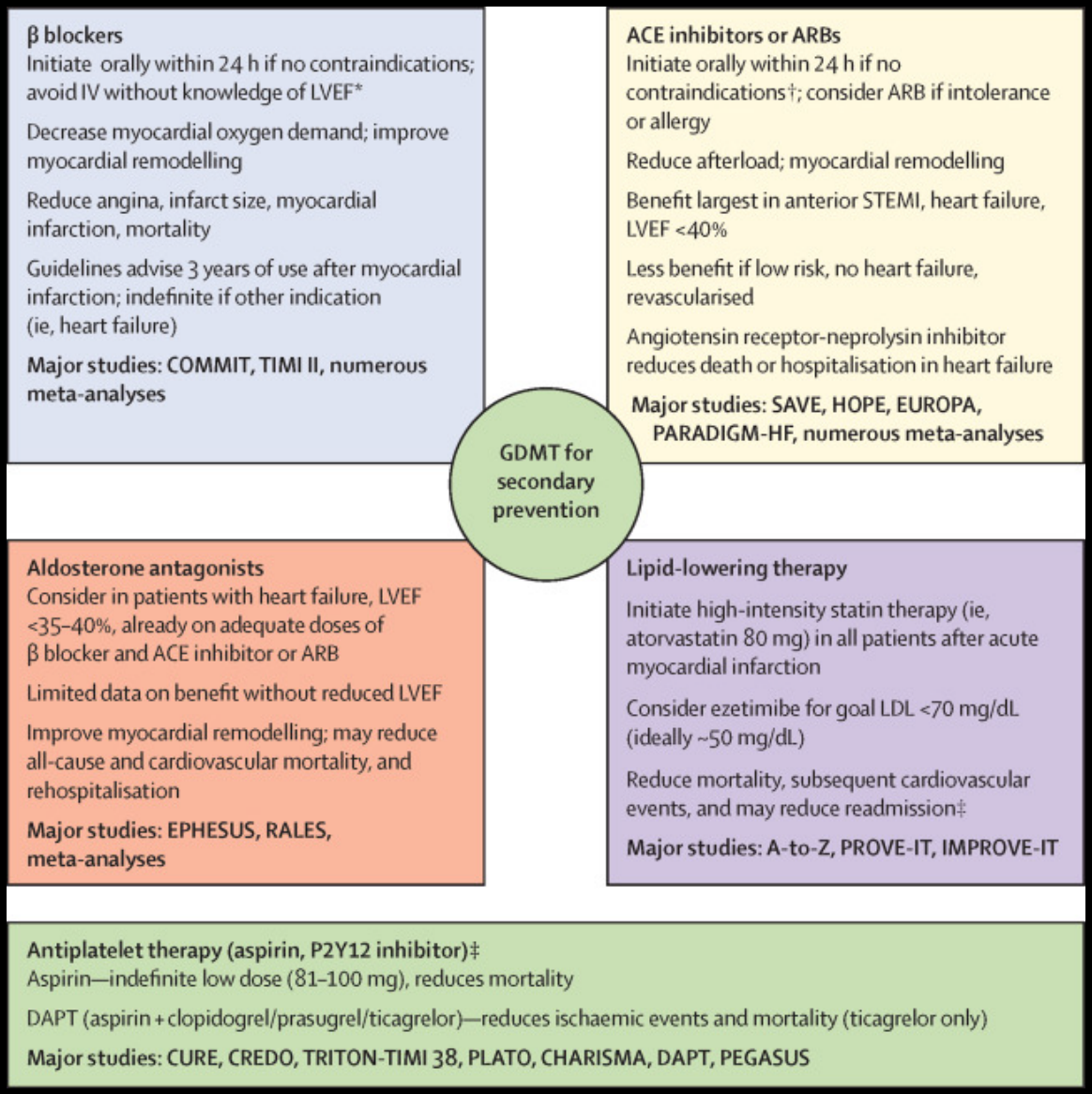
Initial Invasive vs. Conservative Strategy



Post-MI Management

- Echocardiogram
- Lifestyle Modification
- Phase II Cardiac Rehab
- ACE-I if LVEF<40%, Heart Failure, DM or HTN

Post-MI Management



Clinical Pearls

- UA/NSTEMI is typically due to reduced blood flow due to a non-occlusive thrombus that forms on an atherosclerotic plaque.
- UA and NSTEMI are clinically indistinguishable.
- UA/NSTEMI may present with ST segment depression, T wave inversion, or no ECG changes at all.
- UA/NSTEMI patients should be risk-stratified and referred for an early invasive therapy if indicated.
- Risk factor modification and maintenance of an evidence-based medical regimen are essential for secondary prevention.
- Monitor for potential electrical or mechanical complications of ACS.

History

- 62 year old man arrives to the ER complaining of acute, severe precordial chest pain radiating to his arm and neck.
- The pain is described as “an elephant standing on my chest” and accompanied by nausea.
- Chest pain began approximately 30 minutes ago while he was watching television and has not completely resolved.
- PMH: HTN, hyperlipidemia, 50-pack/year tobacco history

Px Exam

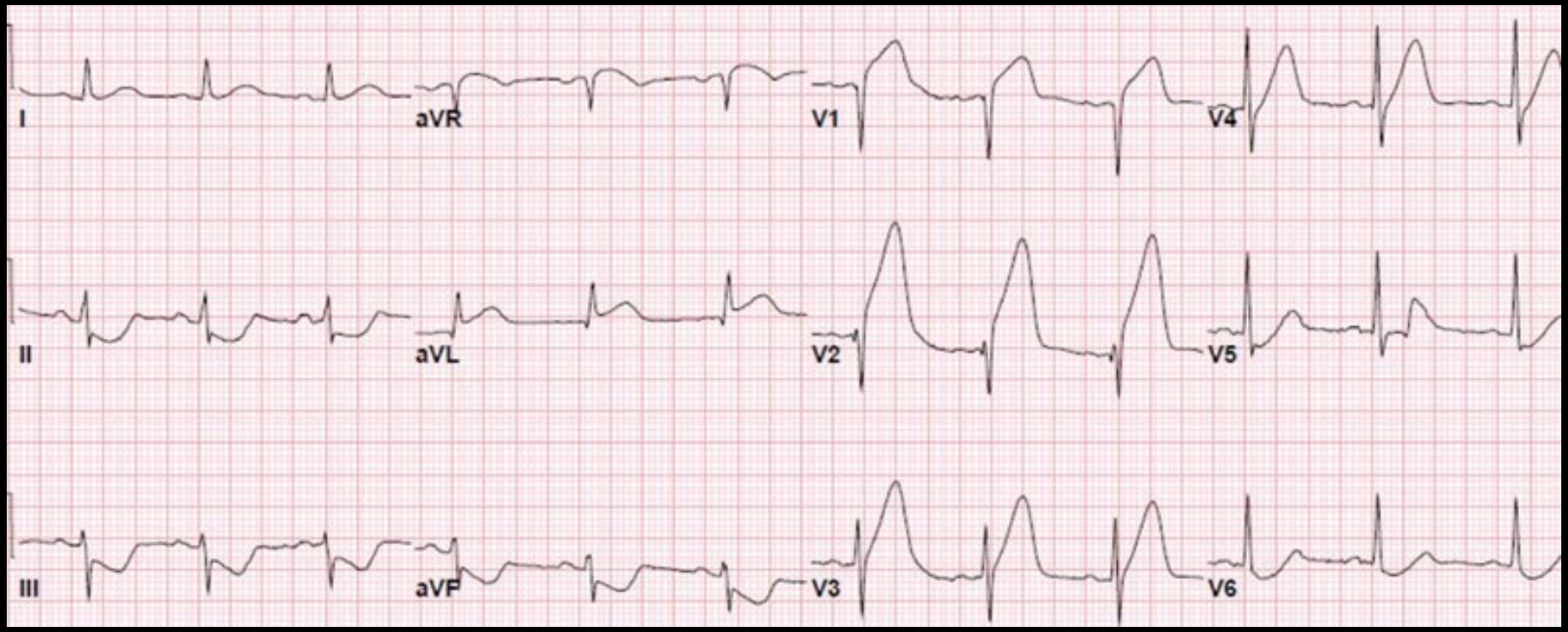
- Vitals – BP 156/97 HR 113 RR 24 RA ox sat 98%
- Gen'l – Appears to be in moderate distress
- Neck – Normal JVP. 2+ carotids with left bruit.
- Lungs – Faint crackles at the bases.
- Abd – Soft, +BS, NT, ND
- Heart – Tachycardic. Regular. Normal S1, S2. No murmur or gallop.
- Ext – No LE edema. 2+ pedal pulses bilaterally.

Questions

- Differential diagnosis?
- Diagnostic work-up?
- Management?

CRITICAL	Emergent	Non Emergent

• Acute Coronary Syndromes
  • STEMI
  • Non-STEMI
  • Unstable angina
• Aortic Dissection
• Cardiac Tamponade
• Pulmonary Embolism
• Tension Pneumothorax
	• Pericarditis
	• Myocarditis
	• Pneumothorax
	• Mediastinitis
	• Cholecystitis
	• Pancreatitis
	• Cocaine chest pain
		• Stable angina
		• Asthma exacerbation
		• Valvular Heart Disease
		• Pneumonia
		• Pleuritis
		• Tumor
		• Esophageal Spasm
		• Gastroesophageal Reflux Disease (GERD)
		• Peptic Ulcer Disease
		• Biliary Colic
		• Rib Fracture
		• Chostochondirits
		• Panic attack

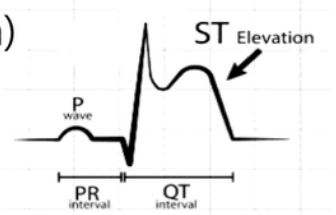

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Differential of ST Elevation

EMNote

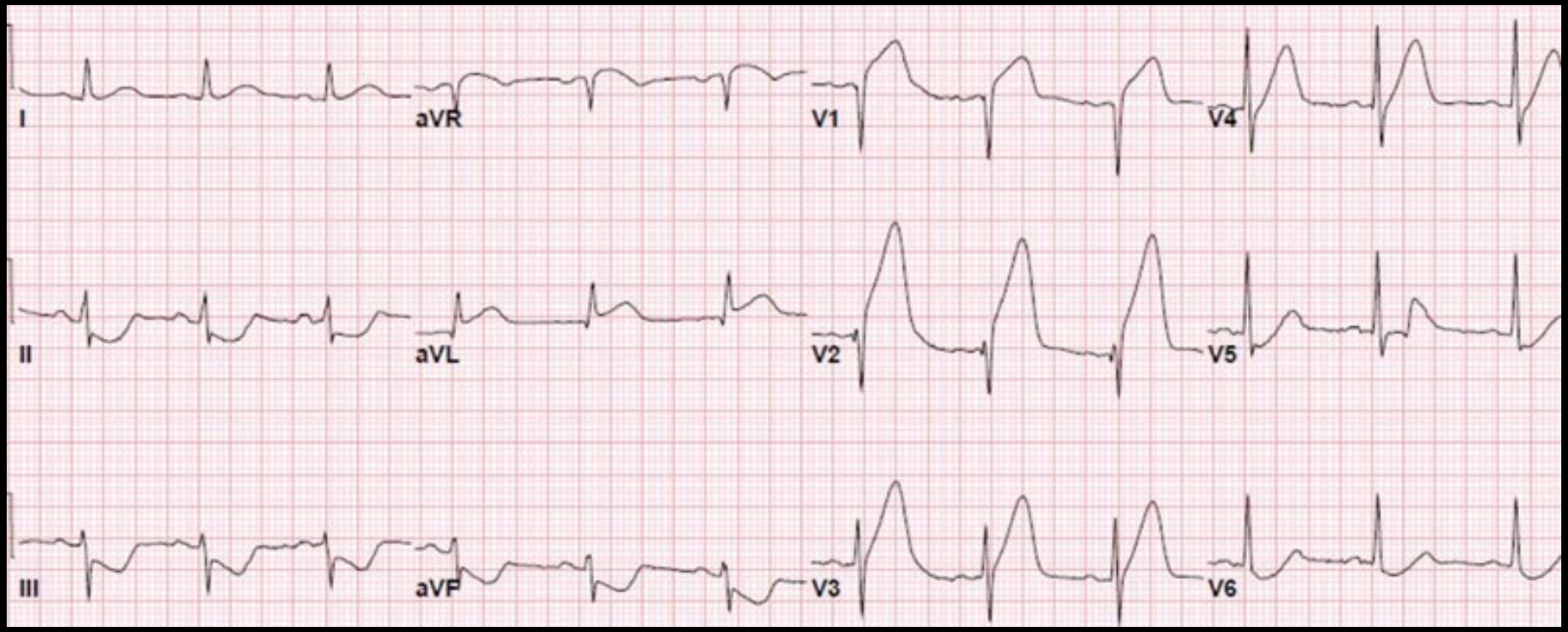
ST Elevation - Not Always AMI

Mnemonic for causes of ST elevation : "ELEVATION"



E : Electrolytes (e.g. hyperkalemia)
L : Left bundle branch block
E : (Benign) Early repolarization
V : Ventricular hypertrophy
A : Arrhythmia (Brugada, VT), Aneurysm of LV, Aortic dissection
T : Takotsubo disease, Traumatic brain injury (ICH)
I : Infarct (MI), Injury(contusion), Inflammation (myo/peri-carditis)
O : Osborn (J) waves (hypothermia or hypercalcemia)
N : Non-atherosclerotic vasospasm (Prinzmetal's angina)

@ jackcfchong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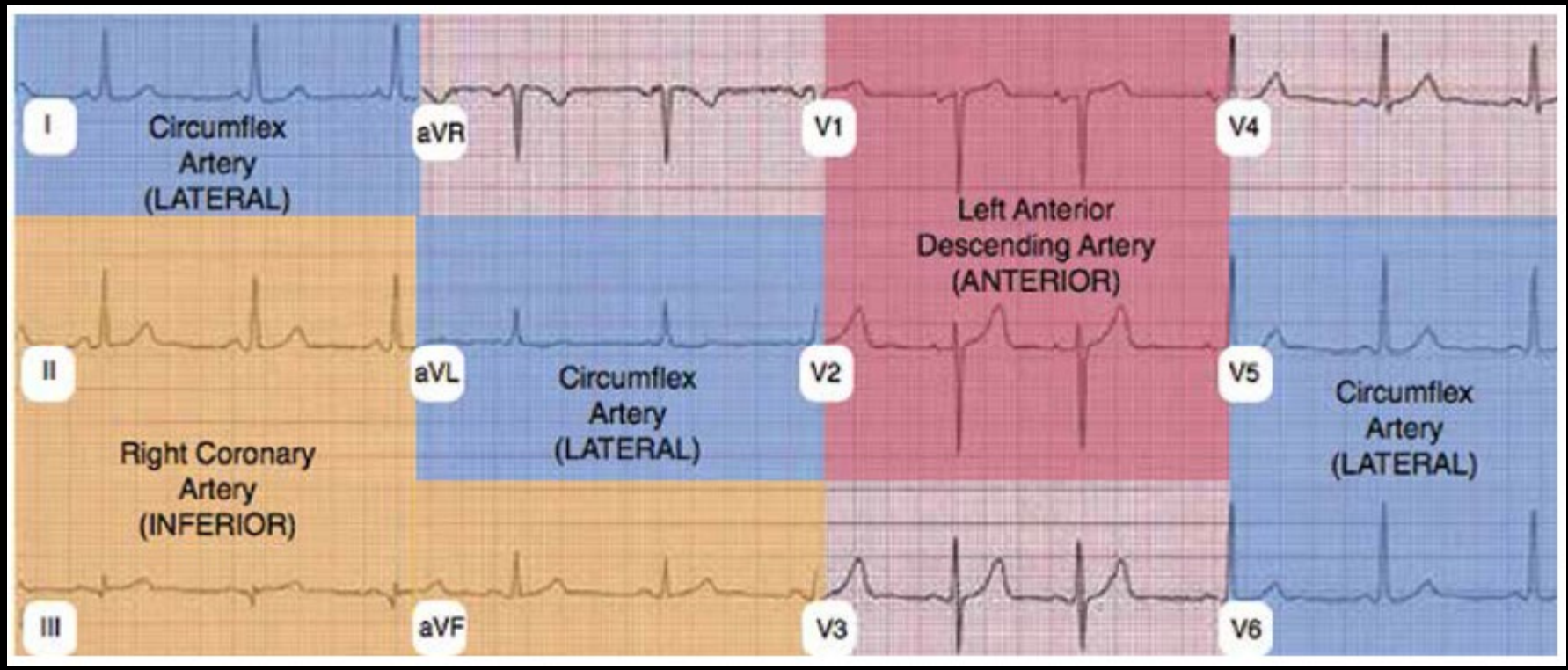
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(ANTERIOR)
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(LATERAL)
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Artery
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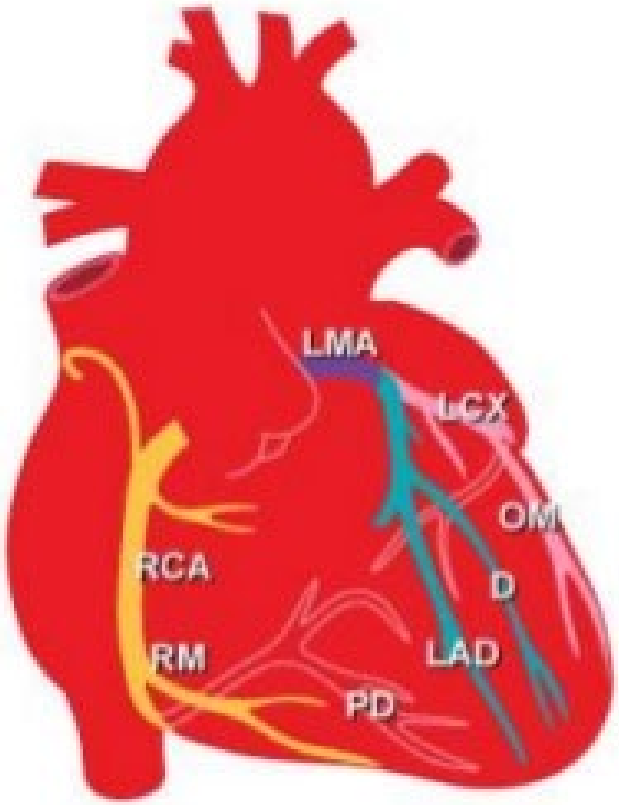
III
aVF
V3
V6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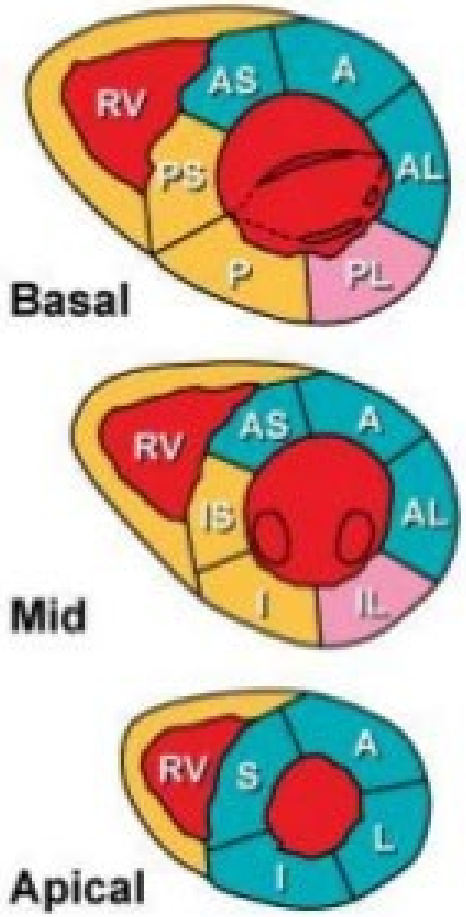
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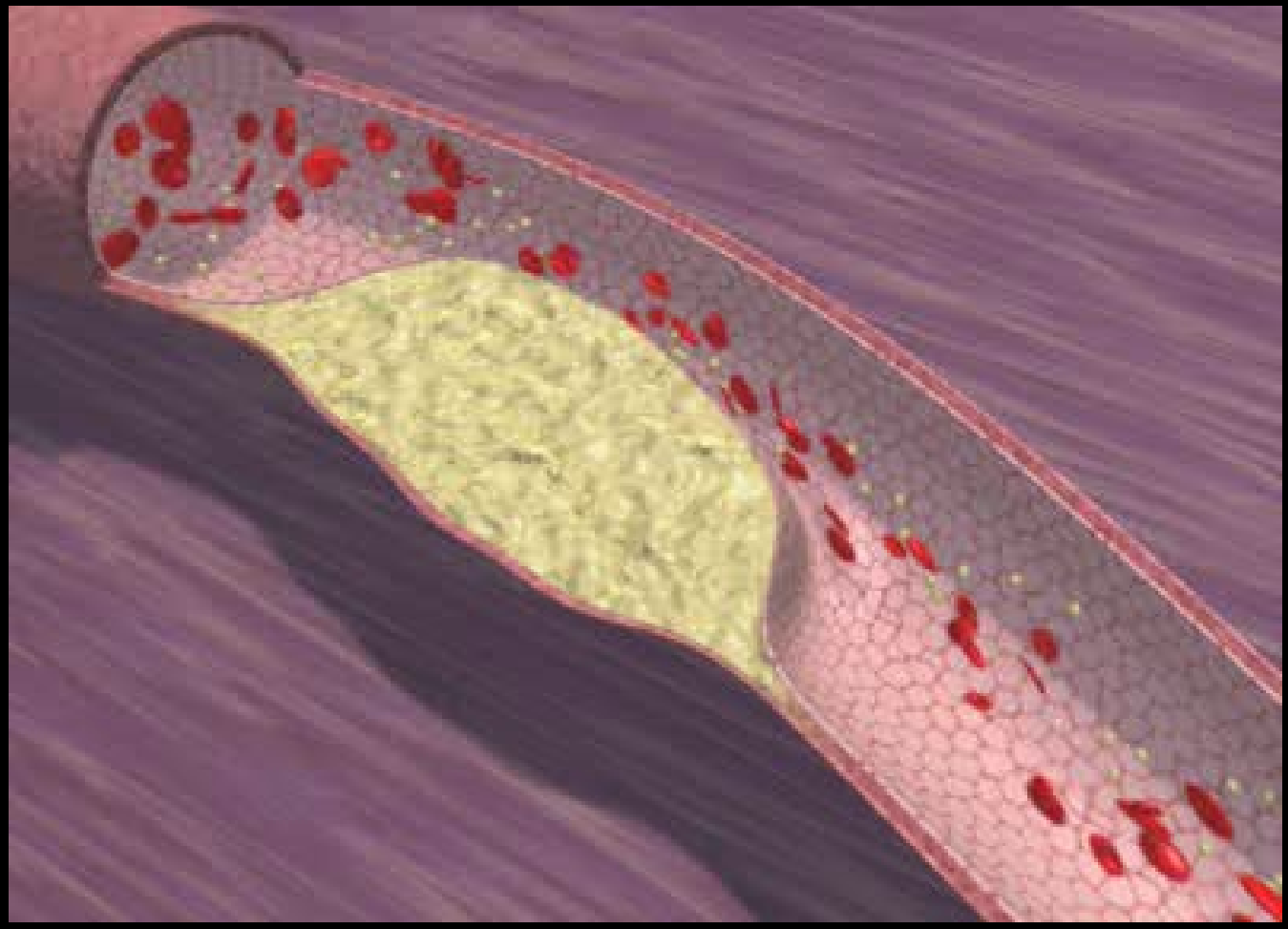
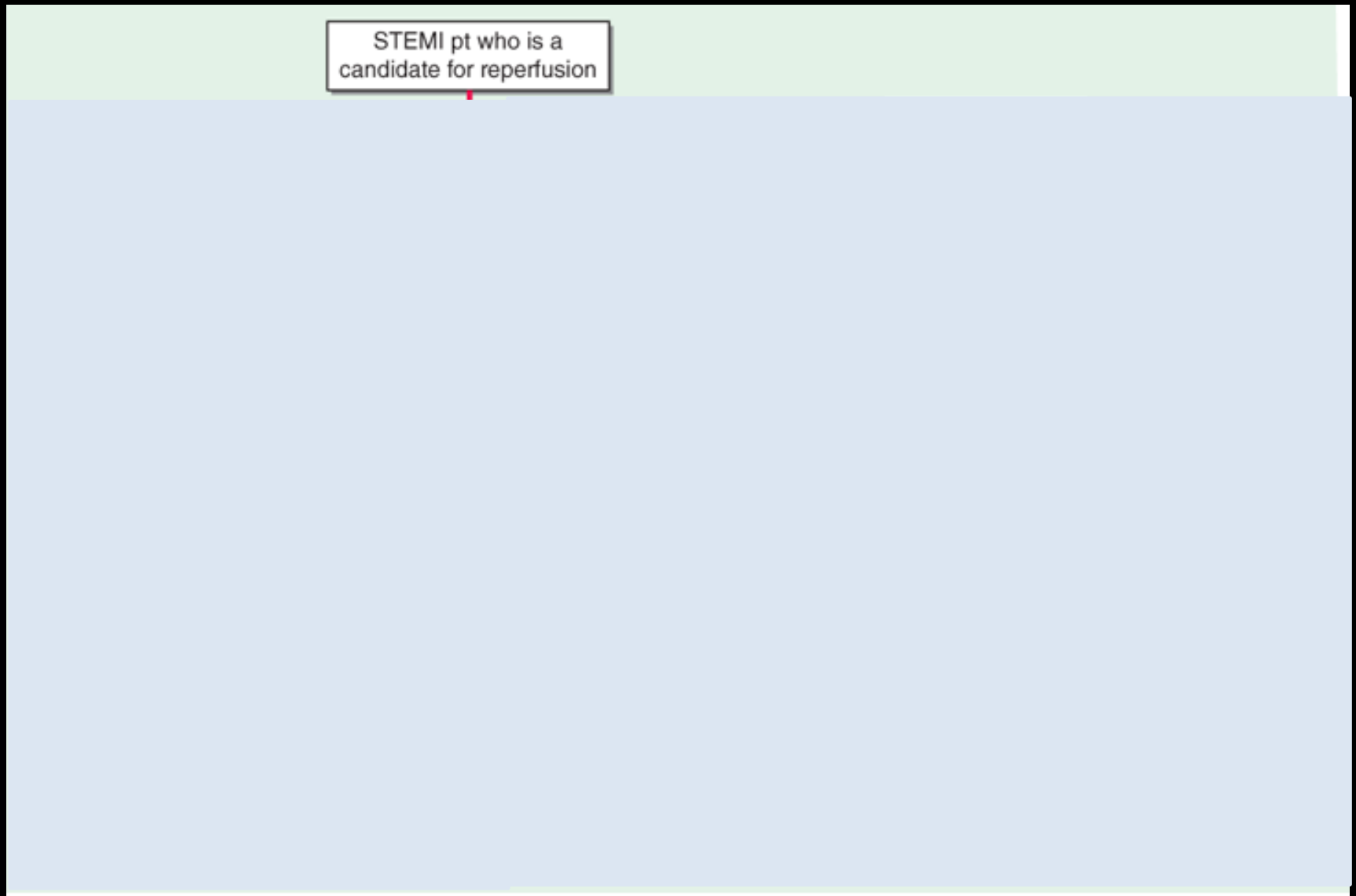


Clinical Presentation

- Majority of patients describe a crushing, heavy, pressure, or squeezing sensation.
- Radiation to the left arm or jaw is classic, although radiation to the back, right arm, shoulder, and epigastric region are also seen.
- The character can be similar to stable angina; however, the pain associated with STEMI is of longer duration, not relieved by NTG and more intense.
- Associated symptoms include dyspnea, diaphoresis, palpitations, nausea, vomiting, extreme fatigue, or an impending sense of doom.
- Women, diabetics or the elderly may present without chest pain, only describing a nonspecific discomfort in combination other sxs



Preferred for FMC to ECG and diagnosis	**≤10 min**
Preferred for FMC to fibrinolysis ('FMC to needle')	**≤30 min**
Preferred for FMC to primary PCI ('door to balloon') in primary PCI hospitals	**≤60 min**
Preferred for FMC to primary PCI	**≤90 min** (≤60 min if early presenter with large area at risk)
Acceptable for primary PCI rather than fibrinolysis	**≤120 min** (≤90 min if early presenter with large area at risk) if this target cannot be met, consider fibrinolysis.
Preferred for successful fibrinolysis to angiography	**3–24 h**

FMC = first medical contact; PCI = percutaneous coronary intervention.

Table 2: Contraindications to Thrombolytic Therapy

Absolute Contraindications
Any prior intracranial hemorrhage
Known intracranial malformation or neoplasm
Ischemic stroke <3 month
Suspected dissection
Recent surgery
Recent head trauma
Bleeding diathesis
Relative Contraindications
>75 years of age
Current anticoagulants
Pregnancy
Cardiopulmonary resuscitation >10 minutes
Recent internal bleed (2–4 weeks)
Uncontrolled hypertension (180/110 mmHg)
Remote ischemic stroke
Major surgery within 3 weeks

Modified from Jaff et al.[39]

Initial Management

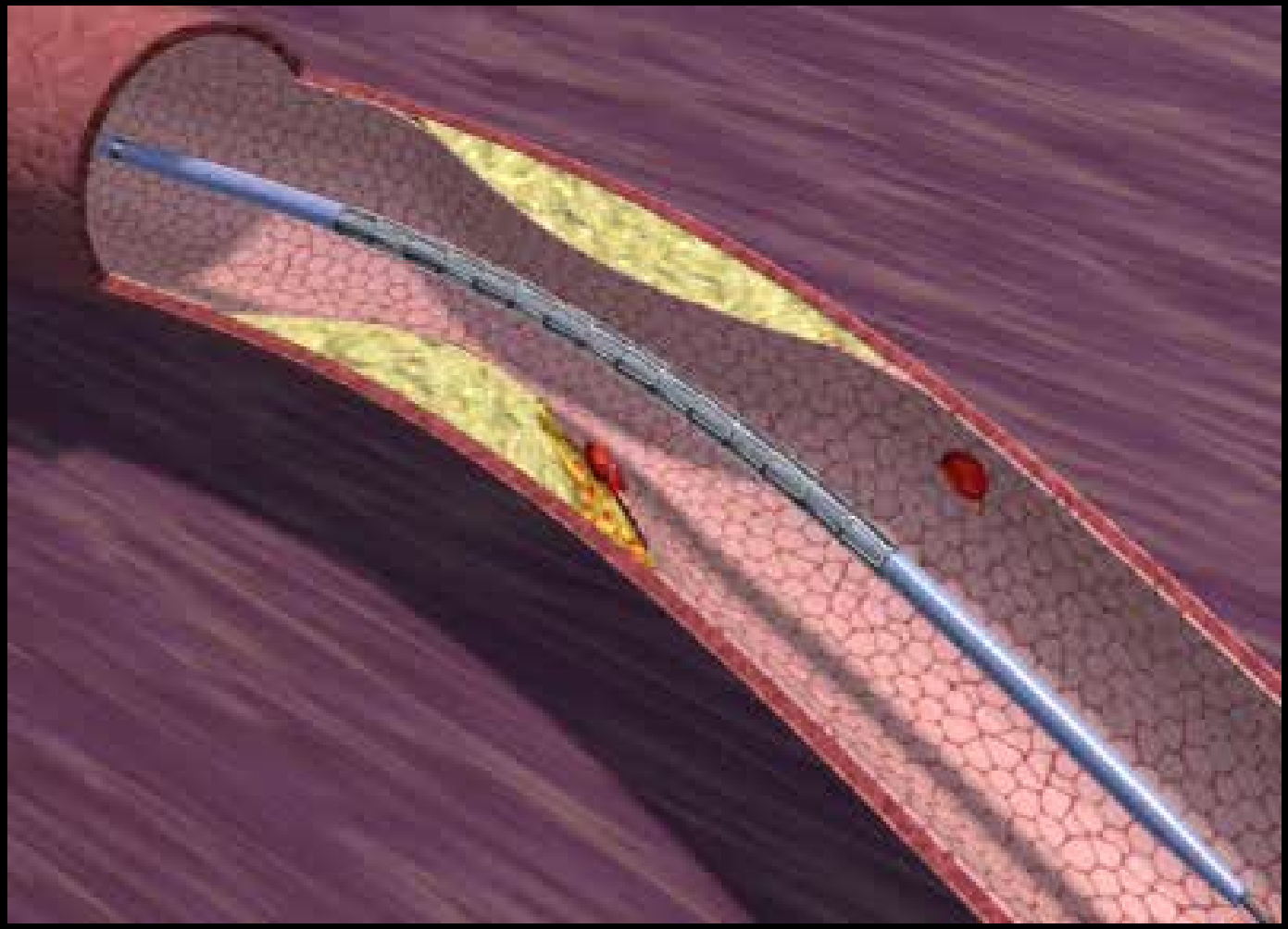
- Aspirin 325 mg chewed
- Anticoagulation – unfractionated heparin, enoxaparin or bivalirudin (preferred because of lower bleeding risk)
- Nitrates +/- morphine
- High-dose statin
- Beta-blockers should be started as soon as feasible and maintained indefinitely

Treatment

Mnemonic Letter	Treatment
M	Morphine
O	Oxygen
N	Nitrates
A	Aspirin
R	Reperfusion (thrombolysis or primary PCI)
C	Clopidogrel (or prasugrel)
H	Heparin
B	Beta-blocker
A	Anticoagulants (aspirin and clopidogrel)
S	Statin
I	Inhibitors of angiotensin II (ACEi or A2R blocker)
C	Correction of risk factors

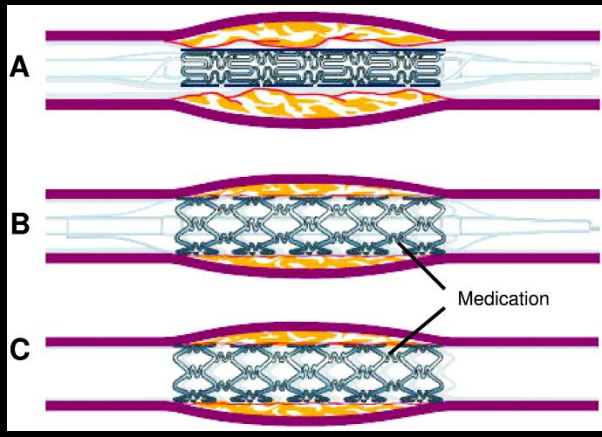
PCI = percutaneous coronary intervention; ACEi = angiotensin-converting enzyme inhibitor; A2R = angiotensin 2 receptor.


M
10-Mar-2016
Ser: -
Img: 1/1 Fr: 26/54
RAO: -36.1°
CAUD: -17.03°
DFOV: 13.2 x 13.2 cm



Cardiac Stents

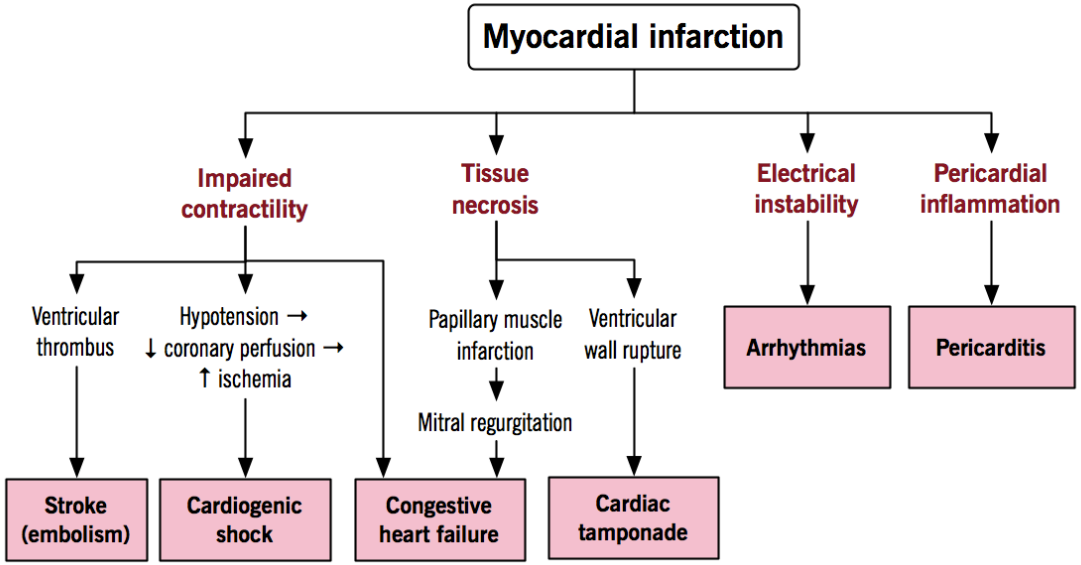
- **Drug-eluting stents (DES)**
  - Anti-proliferative drug to help prevent restenosis
  - Used in 80% of PCI
  - Aspirin and plavix at least 6 months, ideally 1 year

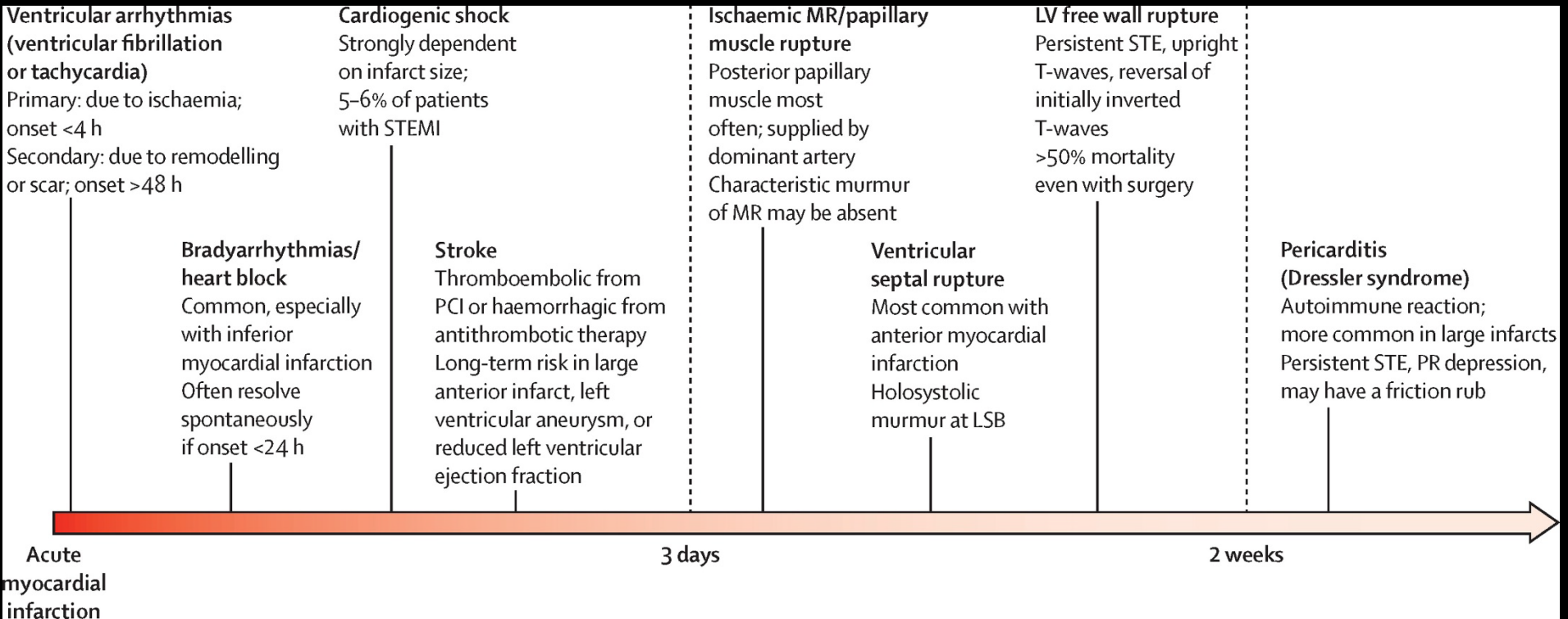


- **Bare metal stents (BMS)**
  - Aspirin and clopidogrel for at least 1 month

Complications of myocardial infarction

Dominique Yelle

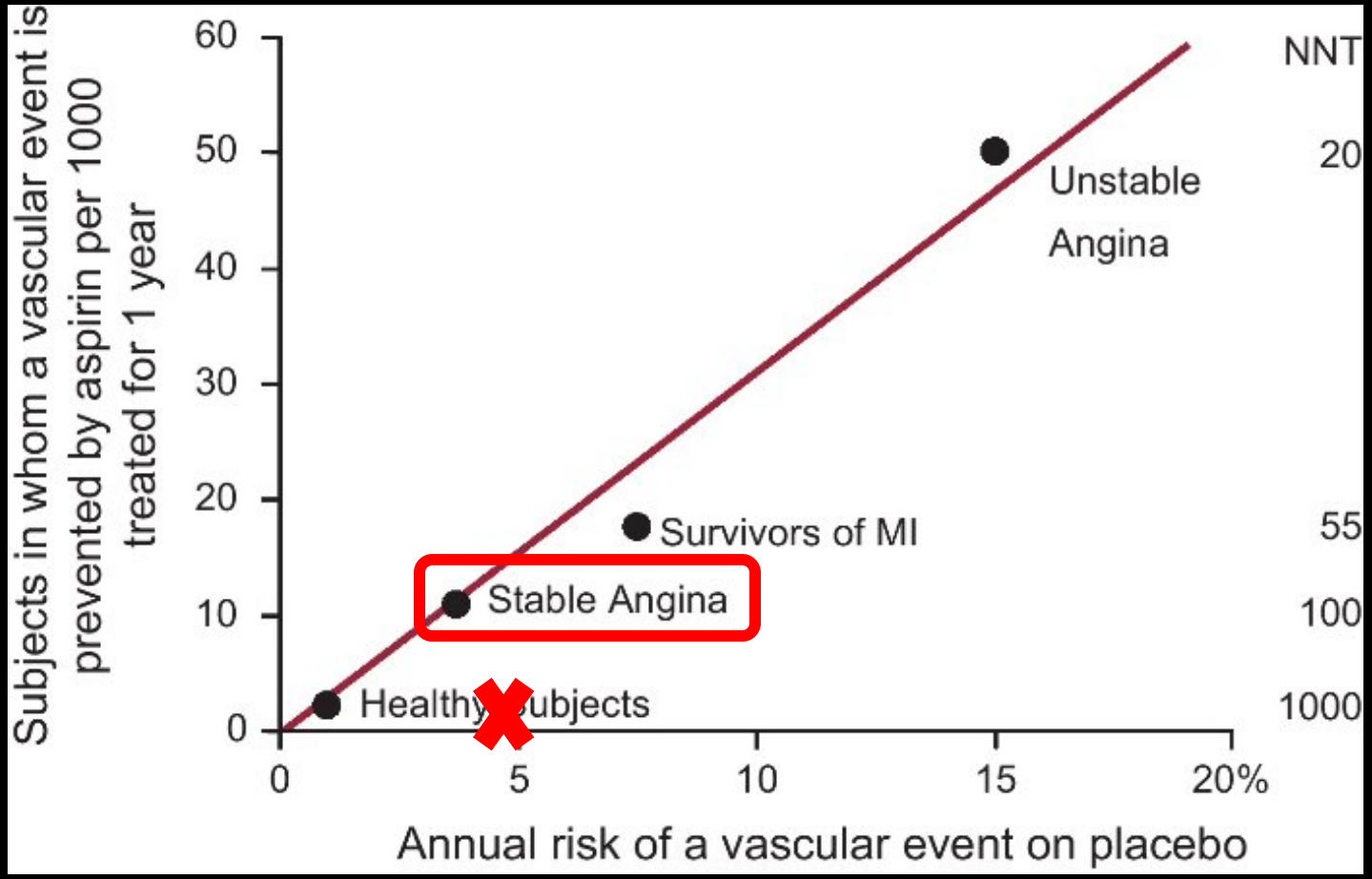




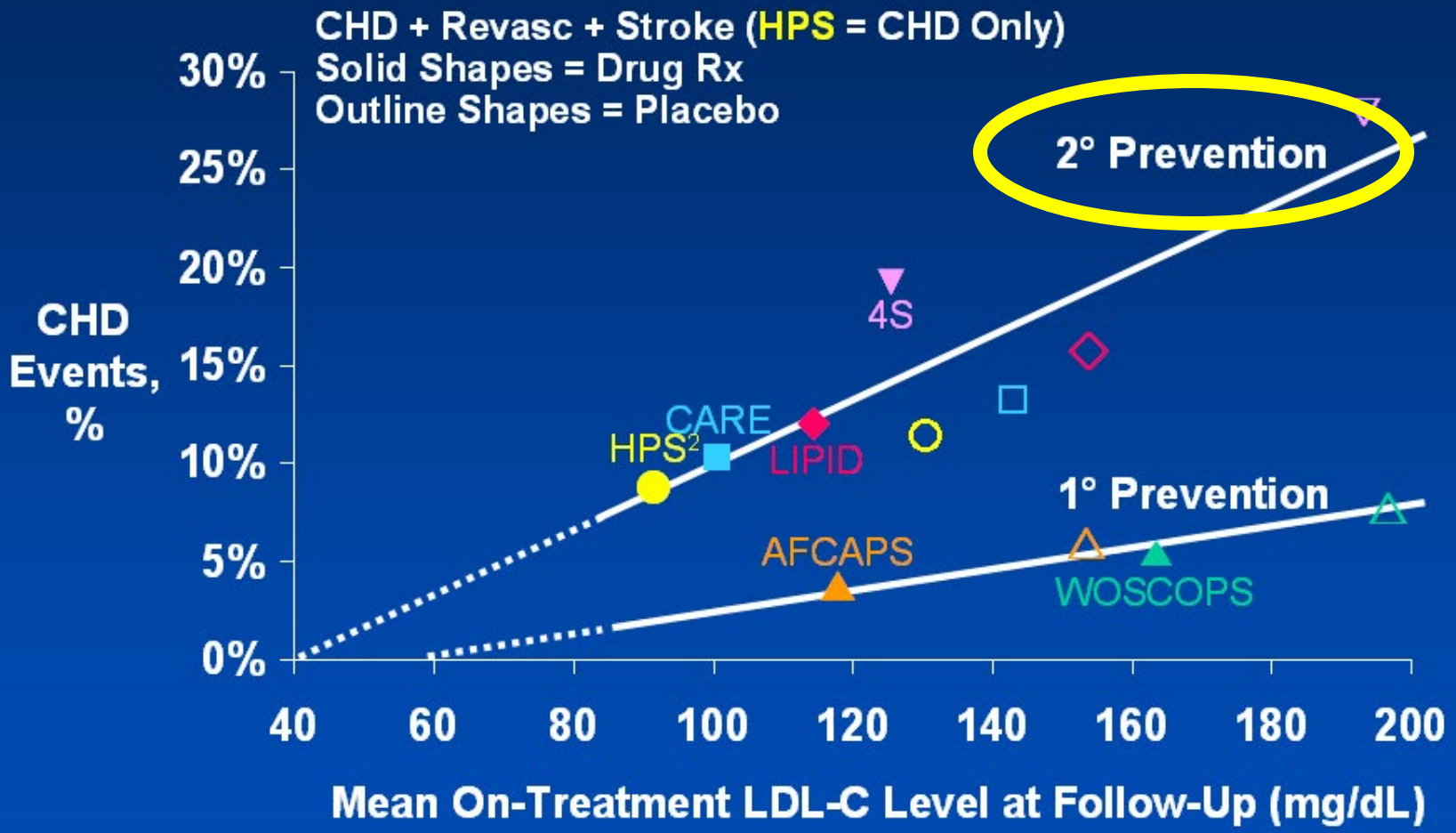
Disease Modifying (Secondary Prevention)

- Aspirin 81 mg QD – lifelong
- Beta Blocker (anti-arrhythmic & remodeling)
- ACE-I (remodeling)
- Statin (Goal <70 mg/dl) – lifelong

Aspirin


Subjects in whom a vascular event is prevented by aspirin per 1000 treated for 1 year
60
50
40
30
20
10
0
NNT
20
55
100
1000
Unstable Angina
Survivors of MI
Stable Angina
Healthy subjects
0
5
10
15
20%
Annual risk of a vascular event on placebo

LDL-C Is Closely Related to CHD Events[1]



1. Adapted from Ballantyne CM. Low-density lipoproteins and risk for coronary artery disease. *Am J Cardiol.* 1998;82:3Q–12Q, with permission from Excerpta Medica.
2. Heart Protection Study Collaborative Group. *Lancet.* 2002;360:7–22.

Clinical Pearls

- **STEMI is due to complete occlusion of a coronary artery characterized by ≥1 mm ST elevation in 2+ contiguous leads.**
- **The location of ST elevation correlates with the occluded artery.**
- **Women, diabetics and the elderly may present with atypical or vague symptoms.**
- **Reperfusion therapy is of the essence and should be initiated ASAP. Ideal "door-to-balloon" time is < 90 minutes or, if PCI is not available, fibrinolytics should be administered within 30 minutes.**
- **After STEMI, risk factor modification and an evidence-based cardioprotective medical regimen consisting of aspirin, ADP inhibitor, high-dose statin, BB and ACE-I are essential.**
- **Monitor for potential electrical or mechanical complications of ACS.**