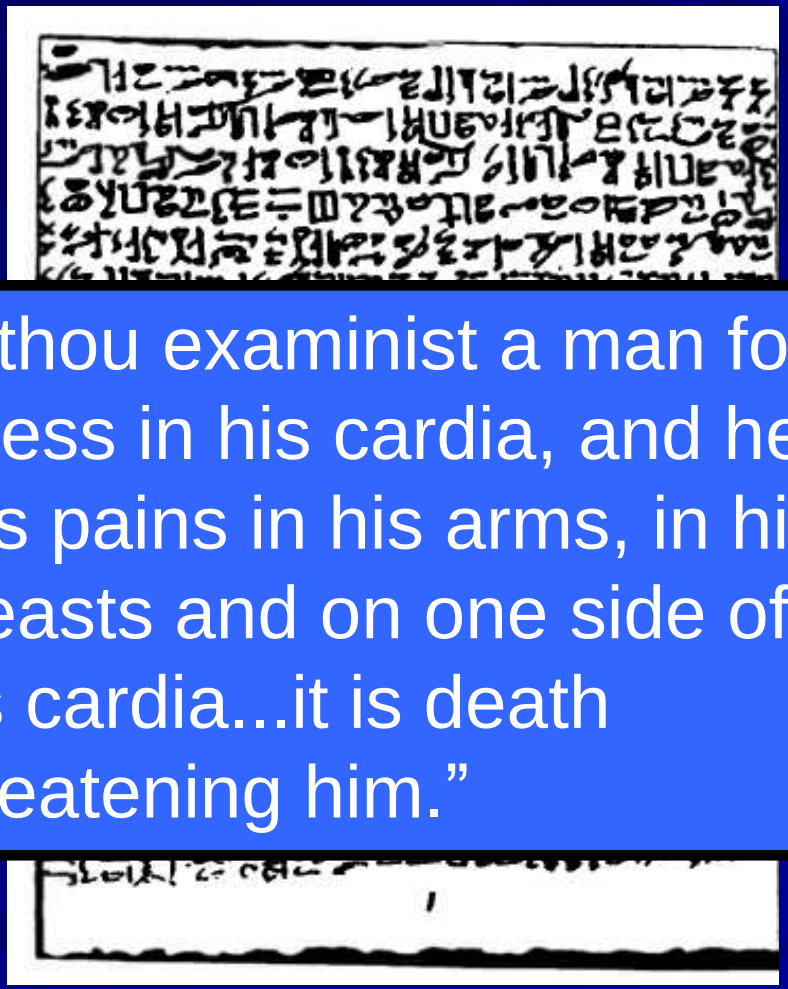
Coronary Artery Disease

Dr. Amitesh Aggarwal

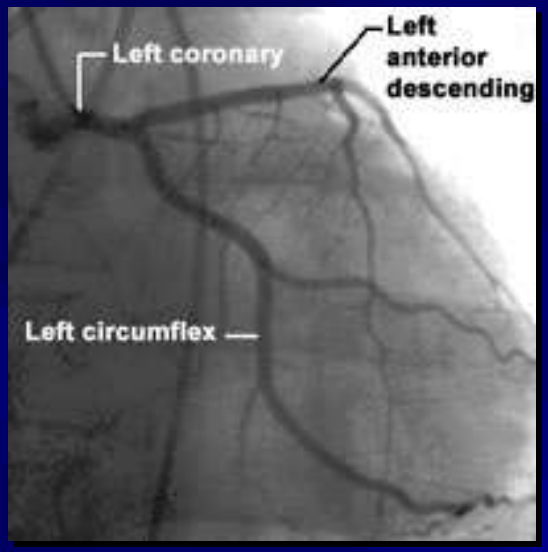
Ebers papyrus, ca. 1555 BCE

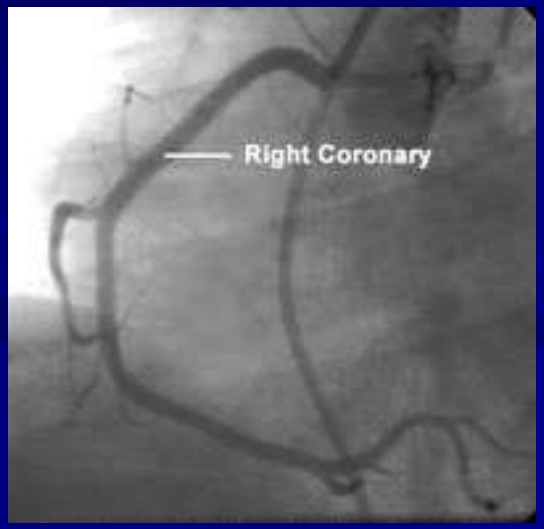


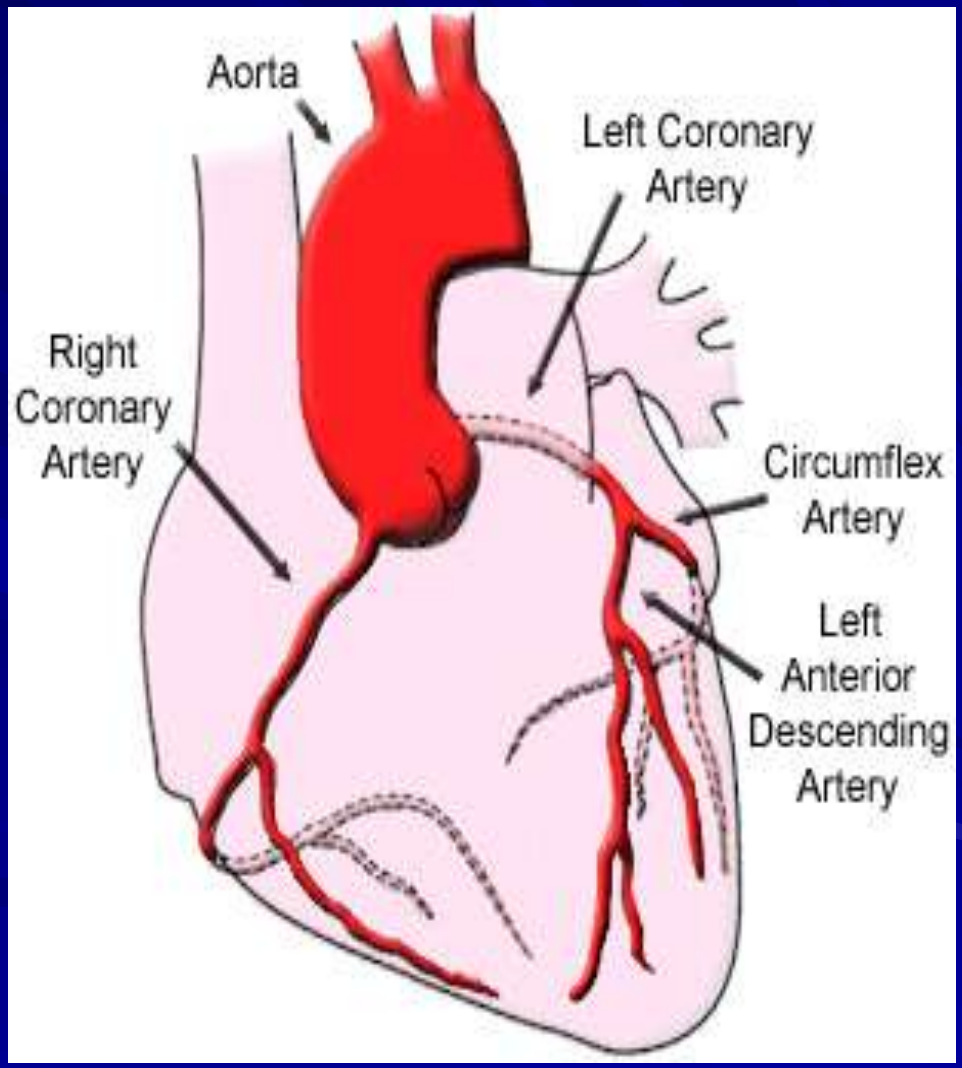
"If thou examinist a man for illness in his cardia, and he has pains in his arms, in his breasts and on one side of his cardia...it is death threatening him."

Ebbell B, The Papyrus Ebers: The greatest Egyptian medical document. 1937, Copenhagen, Levin and Munkfgaard

Coronary anatomy







Spectrum of CAD

- CAD / IHD Stable Angina
- ACS
- UA
- NSTEMI
- STEMI
- Asymptomatic (subclinical)
- ICMP
- SCD

CAD Coronary Artery Disease IHD Ischemic Heart Disease

- Condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium;
- Most common cause is atherosclerotic disease of an epicardial coronary artery sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium.

Angina

Discomfort in the chest or adjacent areas caused by myocardial ischemia but without myocardial necrosis.

- (a) retrosternal pressure, pain, discomfort, or heaviness that
- (b) radiates to the neck, jaw, left arm, or shoulder,
- (c) precipitated by exertion and relieved by rest or nitroglycerin, lasting <10 minutes.

ACS

- Spectrum of clinical presentations ranging from those for STEMI to presentations found in NSTEMI or in UA.
- In terms of pathology, ACS is almost always associated with rupture of an atherosclerotic plaque and partial or complete thrombosis of the infarct-related artery.

Unstable Angina

Angina pectoris or equivalent ischemic discomfort with at least one of three features:

(1) it occurs at rest (or with minimal exertion), usually lasting >10 min;

(2) it is severe and of new onset (i.e., within the prior 4–6 weeks); and/or

(3) it occurs with a crescendo pattern (i.e., distinctly more severe, prolonged, or frequent than previously)

NSTEMI

Clinical features of UA

+

Evidence of myocardial necrosis
(elevated cardiac biomarkers)

+

absence of persistent ST elevation

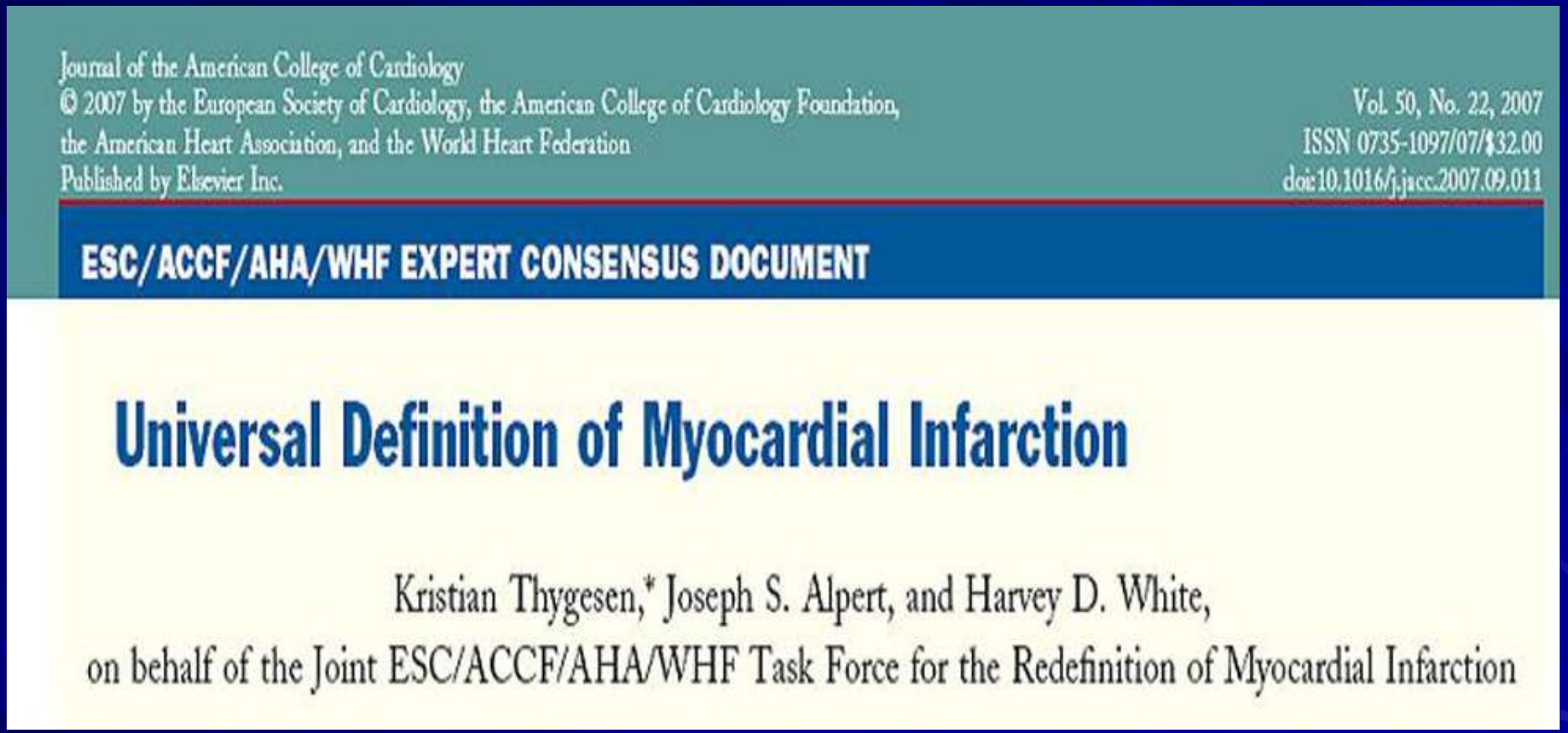
STEMI

WHO DEFINITION (1994)

Two out of three criteria

- Symptoms (chest pain > 20 minutes)
- ECG
- Bio-markers (CK MB, Trop T/I)

Universal Definition of Myocardial Infarction (2007)



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ESC/ACCF/AHA/WHF EXPERT CONSENSUS DOCUMENT

Universal Definition of Myocardial Infarction

Kristian Thygesen,* Joseph S. Alpert, and Harvey D. White,
on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction

Type 1;Type 2;Type 3;Type 4a;Type 4b;Type 5

Clinical Classification of MI
Type 1

- Spontaneous MI related to ischaemia due to primary coronary event such as plaque erosion & / or rupture
- It identifies MI due to atherosclerotic coronary arterial occlusion only
- Rise and/or fall of cardiac biomarkers (preferably troponin) above URL with evidence of at least 1 of the following :

  * Symptoms of ischemia
  * ECG changes indicative of new ischemia
  * Development of pathological Q waves
  * *Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality*

ECG changes in AMI

(In absence of LVH & LBBB)

- ST elevation
  - New ST elevation at the J-point in two contiguous leads with the cut-off points: ≥0.2 mV in men or ≥ 0.15 mV in women in leads V_2-V_3 and/or ≥0.1 mV in other leads
- ST depression &/or T-wave changes
  - New horizontal or down-sloping ST depression ≥0.05 mV in two contiguous leads ; and/or T inversion ≥0.1 mVin two contiguous leads with prominent R-wave or R/S ratio >1
- New LBBB
- Development of Q-waves

ECG changes in AMI

(In absence of LVH & LBBB)

- Hyper- acute T wave amplitude with prominent symmetrical T-wave in at least two contiguous leads is earliest manifestation of AMI

- ST segment equivalent – ST depression in V1-V3 with terminally positive T-wave with reciprocal ST elevation in V7-9

- Pseudonormalisation of previously inverted T-waves

Clinical Classification of MI
Type 2

- MI secondary to ischaemia due to either ↑ O_2 demand or decreased supply, eg, coronary artery spasm, coronary embolism, anemia, arrhythmias, HTN or Hypotension

- There is no coronary artery occlusion
- No scope of reperfusion therapy & antithrombotics

Clinical Classification of MI
Type 3

Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischaemia, accompanied by presumably new STelevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood

Clinical Classification of MI
Type 4a

- Myocardial infarction associated with PCI
  - Embolisation of clot or debris
  - Slow flow or no reflow
  - Dissection
  - Side branch stenosis
  - Disruption of collaterals

(increases > 3X URL of biomarker)

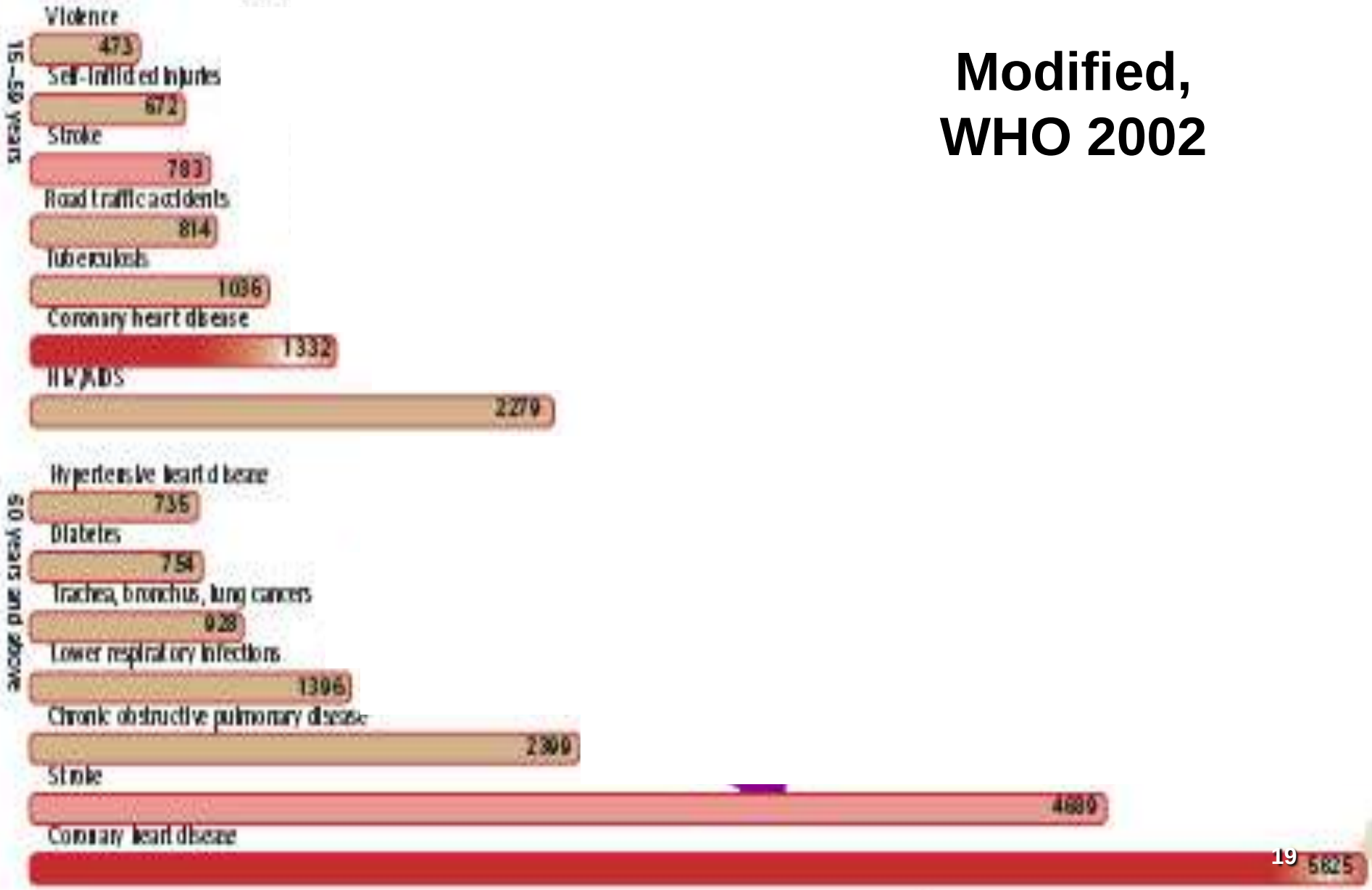
Type 4b

- MI associated with stent thrombosis as documented by angiography or at autopsy

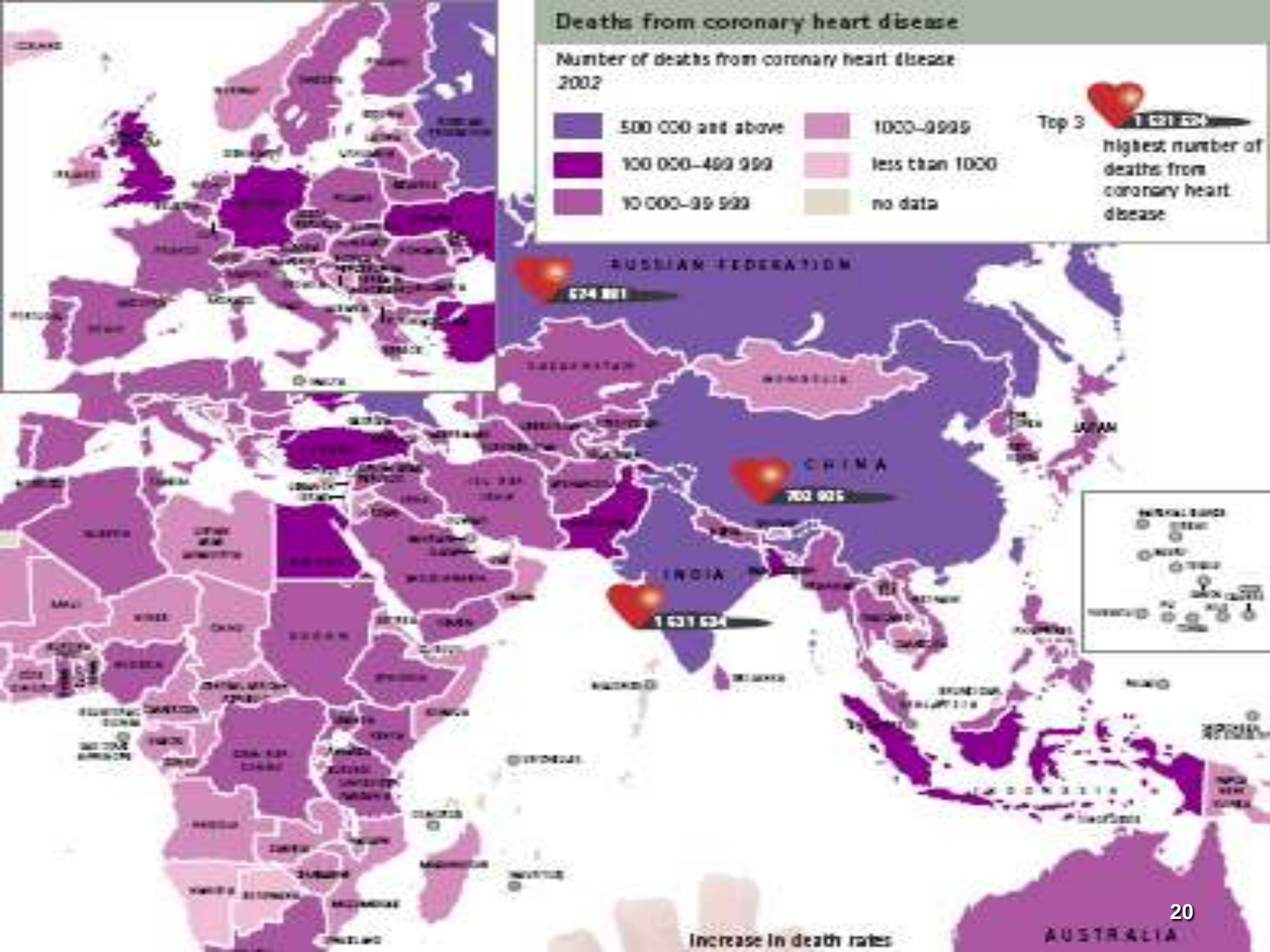
Clinical Classification of MI
Type 5

- Myocardial infarction associated with CABG
- Biomarker > 5 X of URL during first 72 h following CABG, when associated with the appearance of new pathological Q-waves or new LBBB, or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium

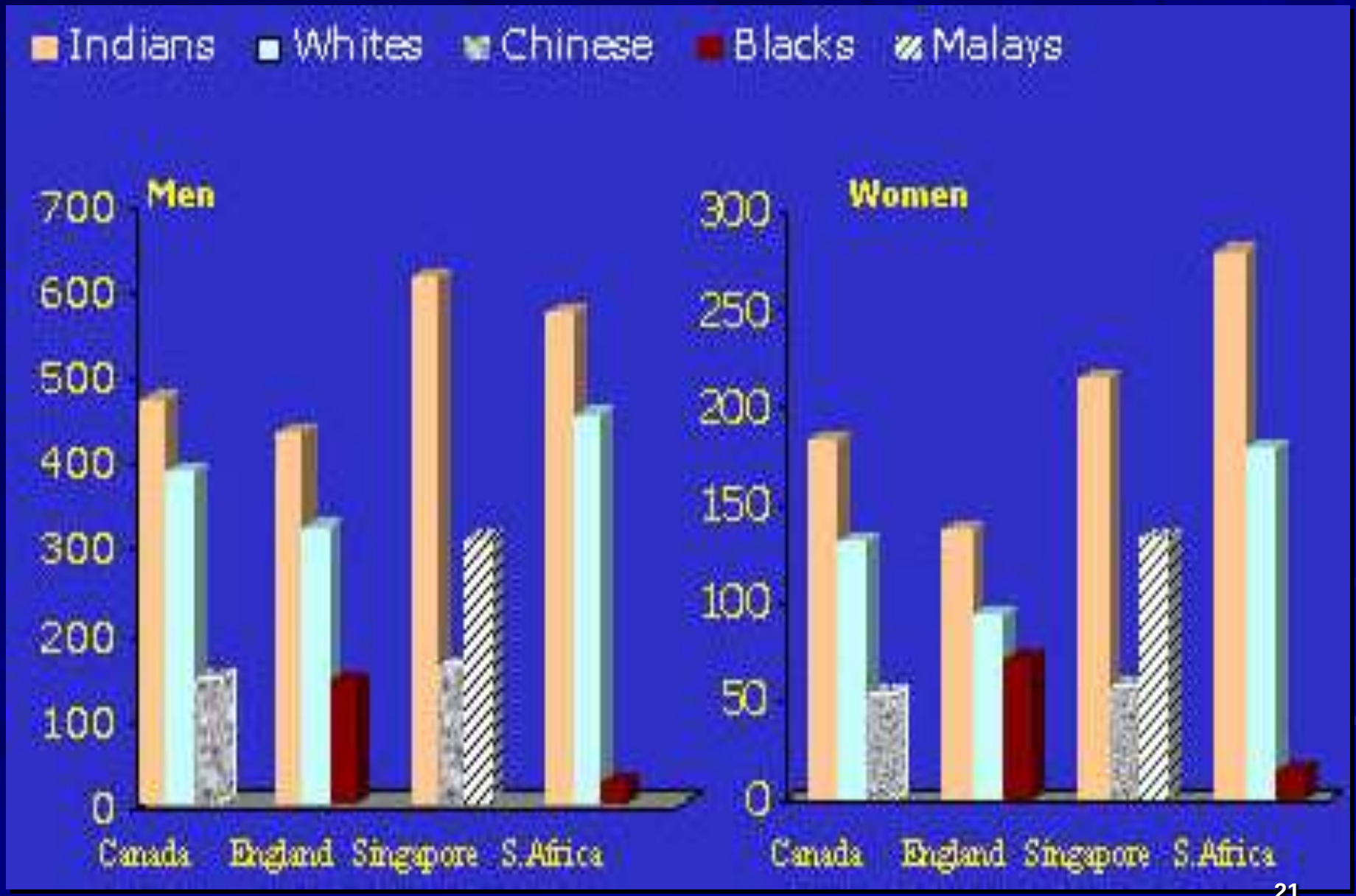
Epidemiology



Modified, WHO 2002


Deaths from coronary heart disease
Number of deaths from coronary heart disease
2002
500 000 and above
100 000–499 999
10 000–99 999
1000–9999
less than 1000
no data
Top 3
highest number of deaths from coronary heart disease
RUSSIAN FEDERATION
634 881
CHINA
702 925
INDIA
1 631 634
AUSTRALIA
Increase in death rates

Mortality rates for CAD in Asian Indians in different countries



Indian Scenario

- Median age of first heart attack is 53 years
- Incidence of CAD in young Indians is 14%–16%
- Mortality attributable expected to rise by 113% in men and 94% in women from 1985 to 2015
- Indian risk of CAD is 3-4 X White Americans 6 X Chinese 20 X Japanese.
- Manifests a decade earlier in ethnic Indians.
- Prevalence Urban (10.5 %) vs. Rural (4.5 %)
- Prevalence Men (7.4 %) vs. Women (4.5 %)

Coronary Artery Disease Other than Atherosclerosis

- Arteritis Takayasu disease
- Trauma Laceration, Radiation
- Coronary mural thickening Homocysteinuria
- Luminal narrowing Spasm (Prinzmetal angina)
- Emboli Infective endocarditis
- Congenital Anomalies ALCAPA
- O2 Demand-Supply Disproportion AS
- Miscellaneous Cocaine abuse

RISK FACTORS OF ATEROSCLEROSIS

Non modifiable

- Age
- Sex
- Menopause
- Family history of DM, HTN, IHD etc.
- Genetic profile
- Vascular anomaly (ostial obstruction)

Modifiable

- Smoking
- Hypertension
- Dyslipidemia
- Diabetes
- Insulin resistance
- Exercise/Obesity
- Sedentary life style
- Mental Stress
- Estrogen status
- Cocaine use

Novel Atherosclerosis Risk factors

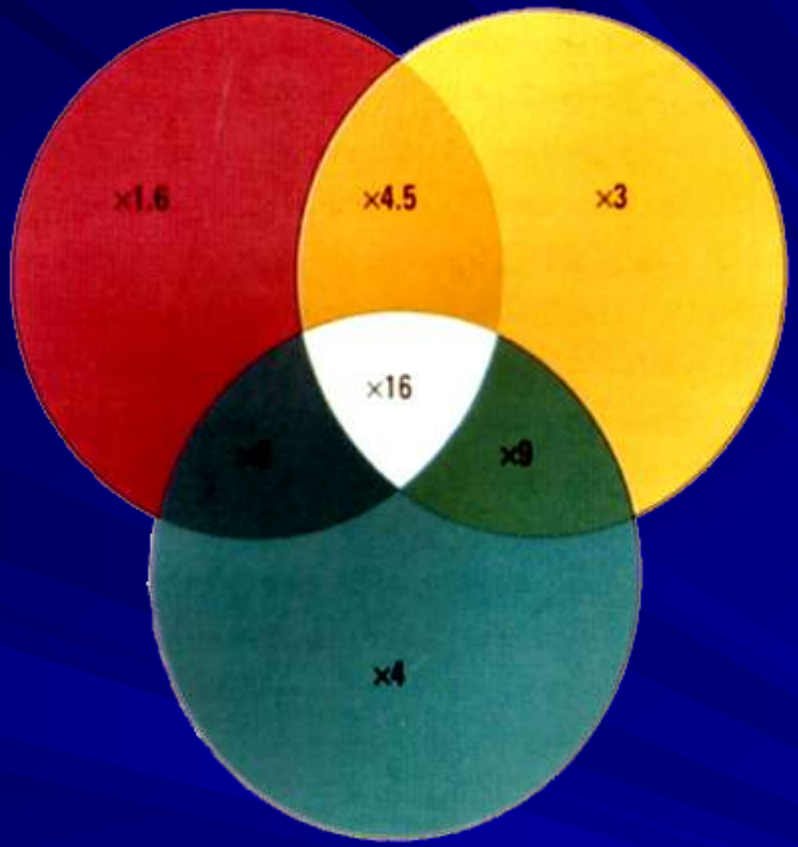
- Homocysteine
- Fibrinogen
- Lipoprotein ‘a’
- Markers of Fibrinolytic function
- Markers of Inflammation (hs – CRP, IL – 6, PAI)

Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study

*Salim Yusuf, Steven Hawken, Stephanie Ôunpuu, Tony Dans, Alvaro Avezum, Fernando Lanas, Matthew McQueen, Andrzej Budaj, Prem Pais, John Varigos, Liu Lisheng, on behalf of the INTERHEART Study Investigators**

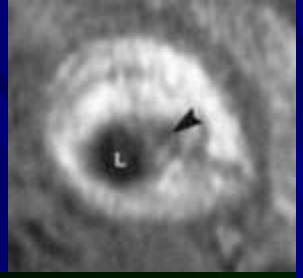
Lancet 2004; 364: 937–52
Published online
September 3, 2004

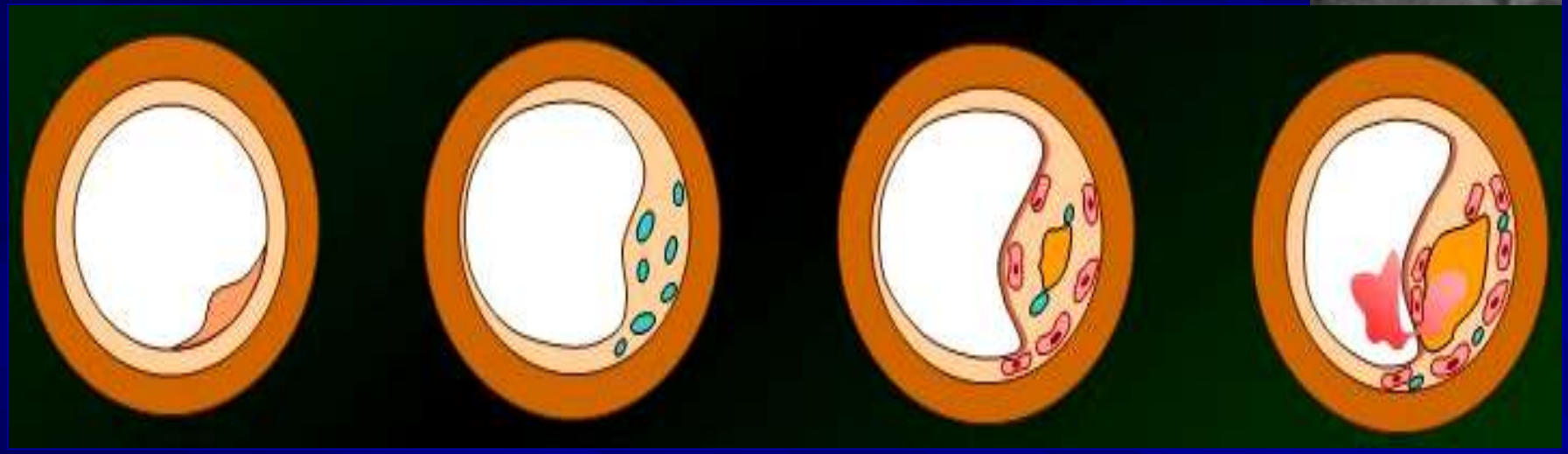
Abnormal lipids, smoking, hypertension, diabetes, abdominal obesity, psychosocial factors, consumption of fruits & vegetables, alcohol and regular physical activity account for most of the risk of MI worldwide in both sexes and at all ages in all regions



- Each of these factors significantly increases the risk of CAD
- When these factors combine in single individual, their effects become multiplicative

Vascular Atherosclerosis is an ongoing process from day of creation to day of destruction



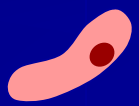


Fatty streak

Transitional plaque

Mature plaque

Ruptured plaque with thrombus formation



Thrombus

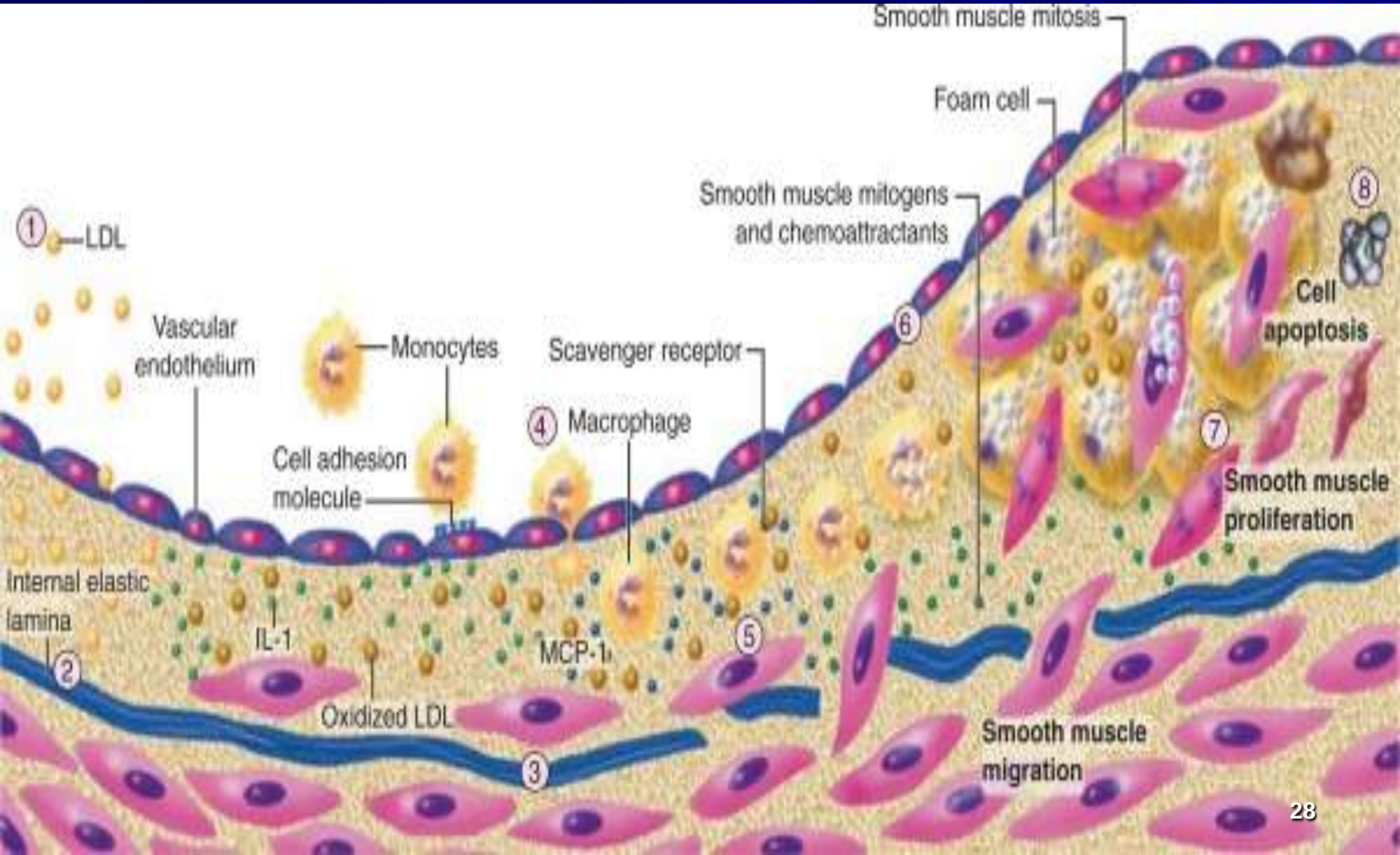
Extra cellular lipid pool

Foam cells

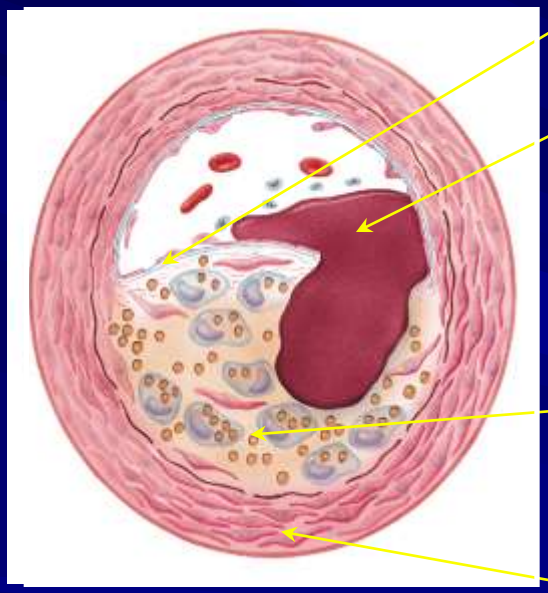
Smooth muscle cells

Fibrous cap

Evolution of Atherosclerotic Plaque



Pathophysiology of Stable and Unstable Plaques



Thin fibrous cap

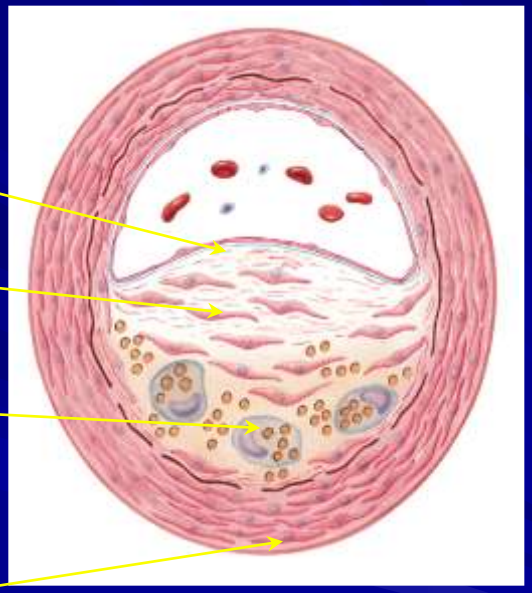
Thrombus

Thick fibrous cap

Smooth muscle cells

Lipid rich core and macrophages

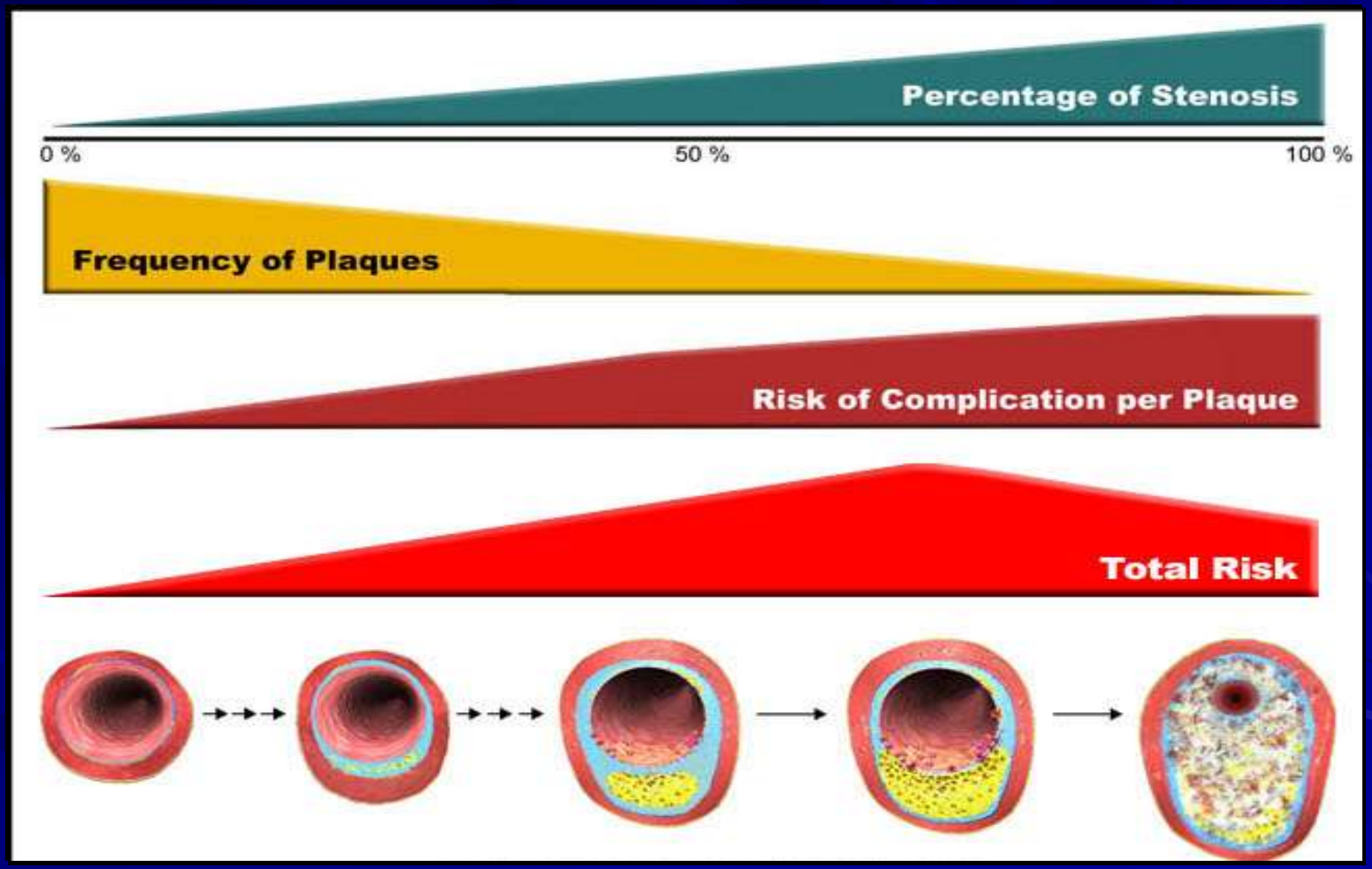
Media



Unstable plaque

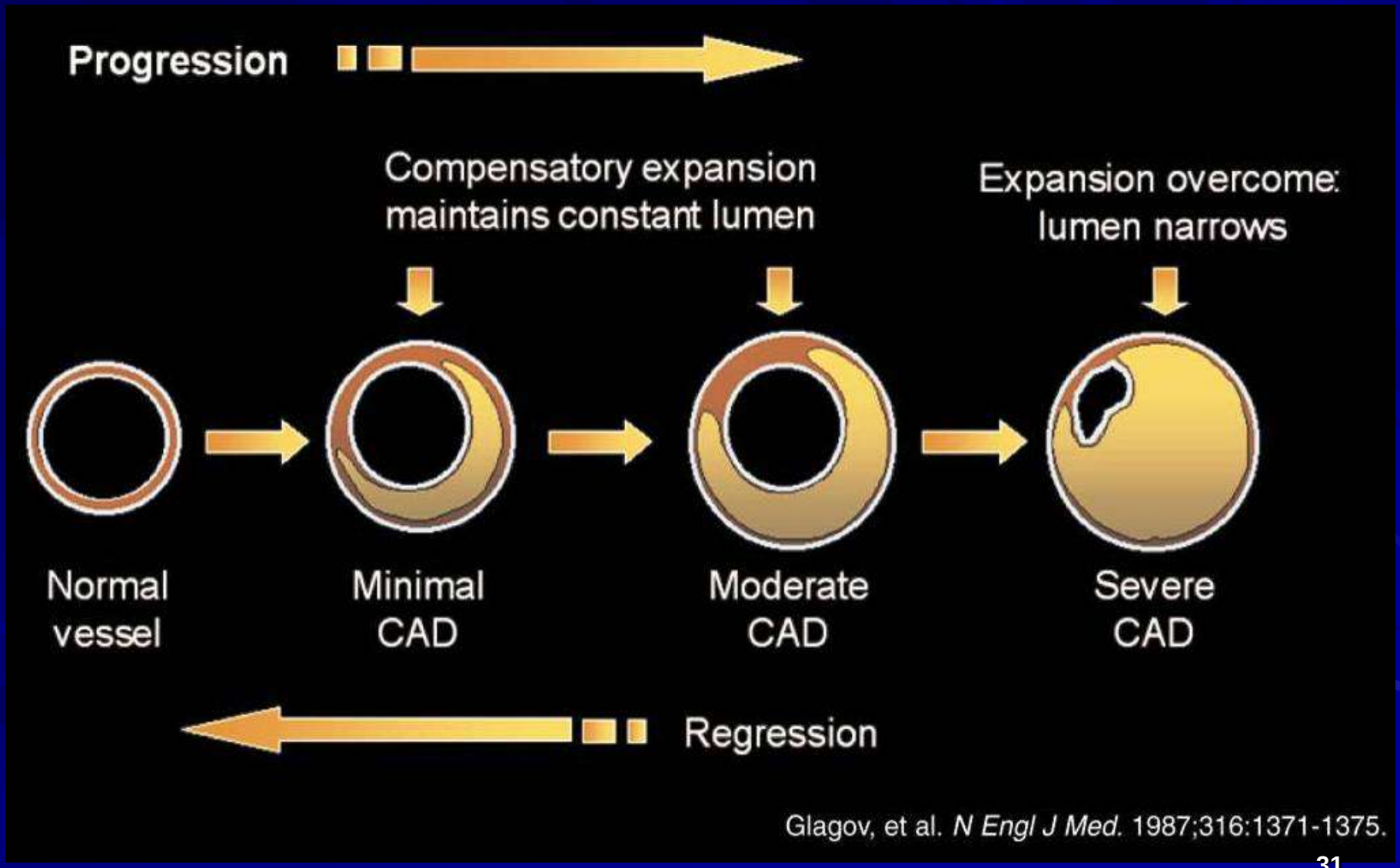
Stable plaque

Non-Stenotic Vulnerable Plaques overall are More Dangerous Since they are far More Frequent than Stenotic Ones



Naghavi et al. *Circulation.* 2003;108:1664

Plaque remodeling



Glagov, et al. *N Engl J Med.* 1987;316:1371-1375.

Angina

Discomfort in the chest or adjacent areas

- (a) retrosternal pressure, pain, discomfort, or heaviness that
- (b) radiates to the neck, jaw, left arm, or shoulder,
- (c) precipitated by exertion, emotional stress, medical or surgical illness, morning and relieved by rest or nitroglycerin, lasting <10 minutes.

Anginal equivalents

- Dyspnea without pain
- Atypical location of the pain – epigastric
- Apprehension and nervousness
- Sudden mania or psychosis
- Syncope
- Profound weakness
- Acute indigestion

Unstable Angina/ NSTEMI

Angina pectoris or equivalent ischemic discomfort with at least one of three features:

(1) it occurs at rest (or with minimal exertion), usually lasting >10 min;

(2) it is severe and of new onset (i.e., within the prior 4–6 weeks); and/or

(3) it occurs with a crescendo pattern (i.e., distinctly more severe, prolonged, or frequent than previously)

STEMI

- Angina pectoris or equivalent ischemic discomfort
- May be accompanied by weakness, vomiting, sweating, dizziness, palpitations, cold perspiration, and sense of impending doom
- Pronounced circadian periodicity for the time of onset of STEMI, with peak incidence of events between 6 AM and 12 noon

Atypical presentations of STEMI

- heart failure - dyspnea without pain
- classic angina pectoris without severe or prolonged episode
- atypical location of the pain
- central nervous system manifestations
- apprehension and nervousness
- sudden mania or psychosis
- Syncope
- overwhelming weakness
- acute indigestion
- peripheral embolization

MI patients without chest discomfort

- More likely to be
  - Older
  - Women
  - Diabetic and/or have prior heart failure
  - Delayed longer before they went to the hospital
- Less likely to be diagnosed as having an MI when admitted
- Less likely to receive fibrinolysis or primary PCI, aspirin, beta-blockers or heparin
- 2.2 times more likely to die during the hospitalization

ANGINA / MI WITH NORMAL CORONARY ARTERIES

- Seen in 6% of pt.
- More common in women (mechanism difficult to establish)
- Tend to be young and have relatively few coronary risk factors, except smoking.
- Usually no history of angina pectoris prior to the infarction.
- The infarction in these patients is usually not preceded by any prodrome,
- clinical, laboratory, and ECG features of STEMI are otherwise indistinguishable from classical STEMI
- In patients who recover, areas of localized dyskinesis and hypokinesis can often be demonstrated by left ventricular angiography.

PHYSICAL EXAMINATION

- Frequently negative
- But careful search for
  - Valve disease (aortic valve)
  - Left ventricular dysfunction (cardiomegaly , gallop rhythm)
  - Manifestation of arterial diseases (carotid bruits peripheral vascular diseases)
  - Unrelated condition that exacerbate angina (anemia, thyrotoxicosis)

Findings & Implications in MI

CVS

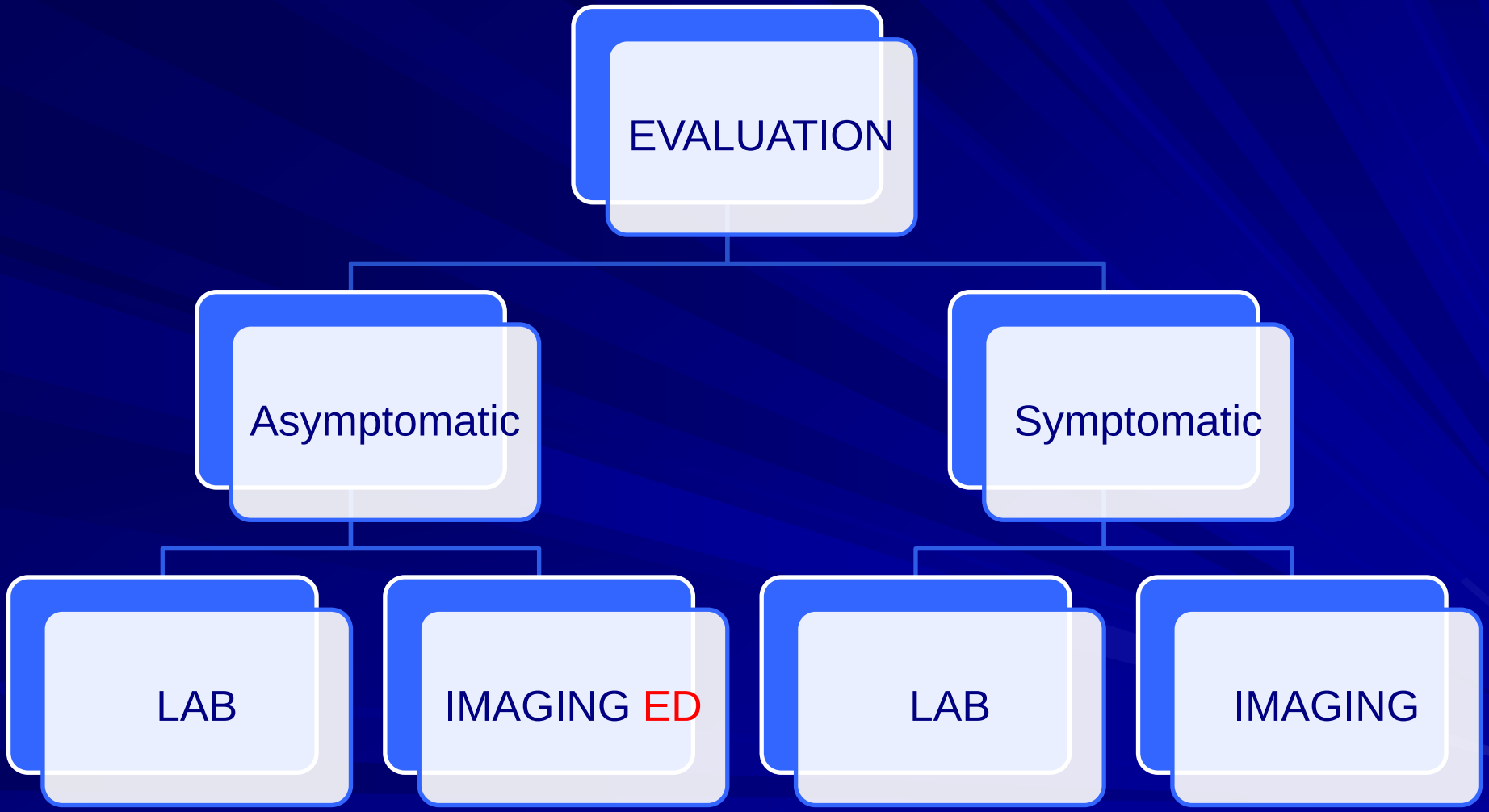
- General: Restless, agitated, Levine's sign
- Skin: Cool, clammy, pale, ashen
- Low-grade fever: response to myocardial necrosis
- Hypertension, tachycardia: ↑ sympathetic tone (AWMI)
- Hypotension, bradycardia: ↑ vagal tone (I/P MI)
- Small-volume pulses: Low cardiac output
- Irregular pulse: Atrial / vent arrhythmias, CHB
- Paradoxical "ectopic" systolic impulse: LV dyskinesis, ventricular aneurysm (AWMI)

Findings & Implications in MI

- **Soft S1:** ↓ LV contractility; 1° AV block (IW MI)
- **S4 gallop:** ↓ LV compliance
- **Paradoxically split S2:** Severe LV dys, LBBB
- **S3 gallop, pulmonary rales, pulsus alternans:** LV systolic dys (CHF >25% of myocardium)
- **↑ JVP, Kussmaul's sign, hypotension, RV S4 and S3 gallops, clear lungs:** RVMI
- **Pericardial friction rub:** Pericarditis
- **Absent pulses and murmur of AR:** Aortic dissection

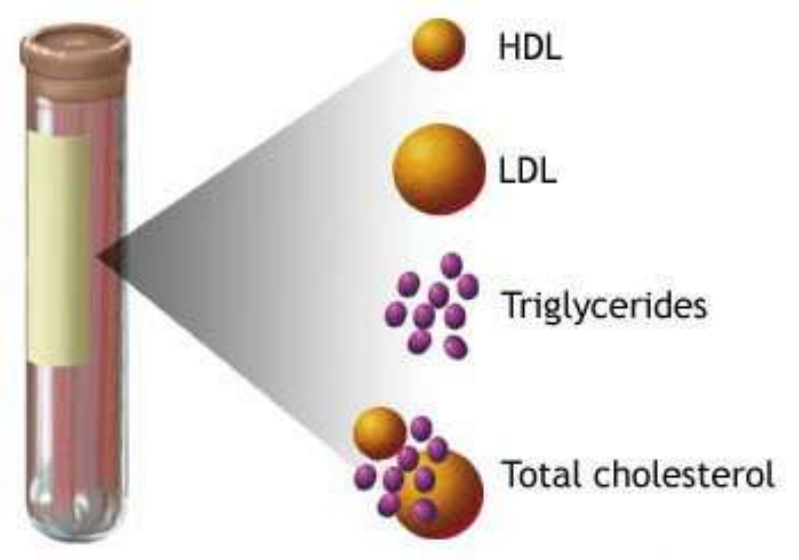
CHEST

May be normal or **few basal crackles**: Pulmonary congestion

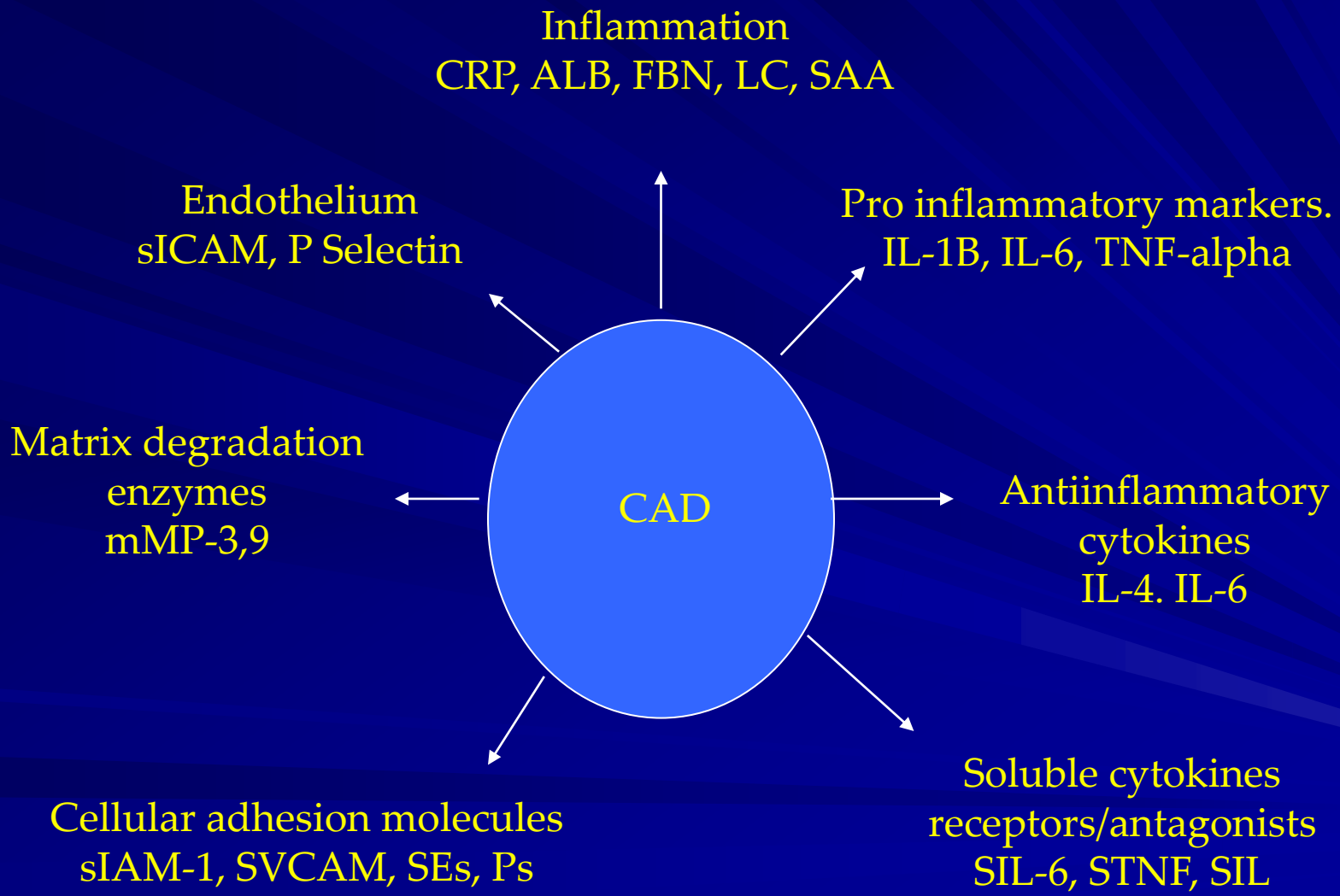

EVALUATION
Asymptomatic
Symptomatic
LAB
IMAGING ED
LAB
IMAGING

RISK FACTORS

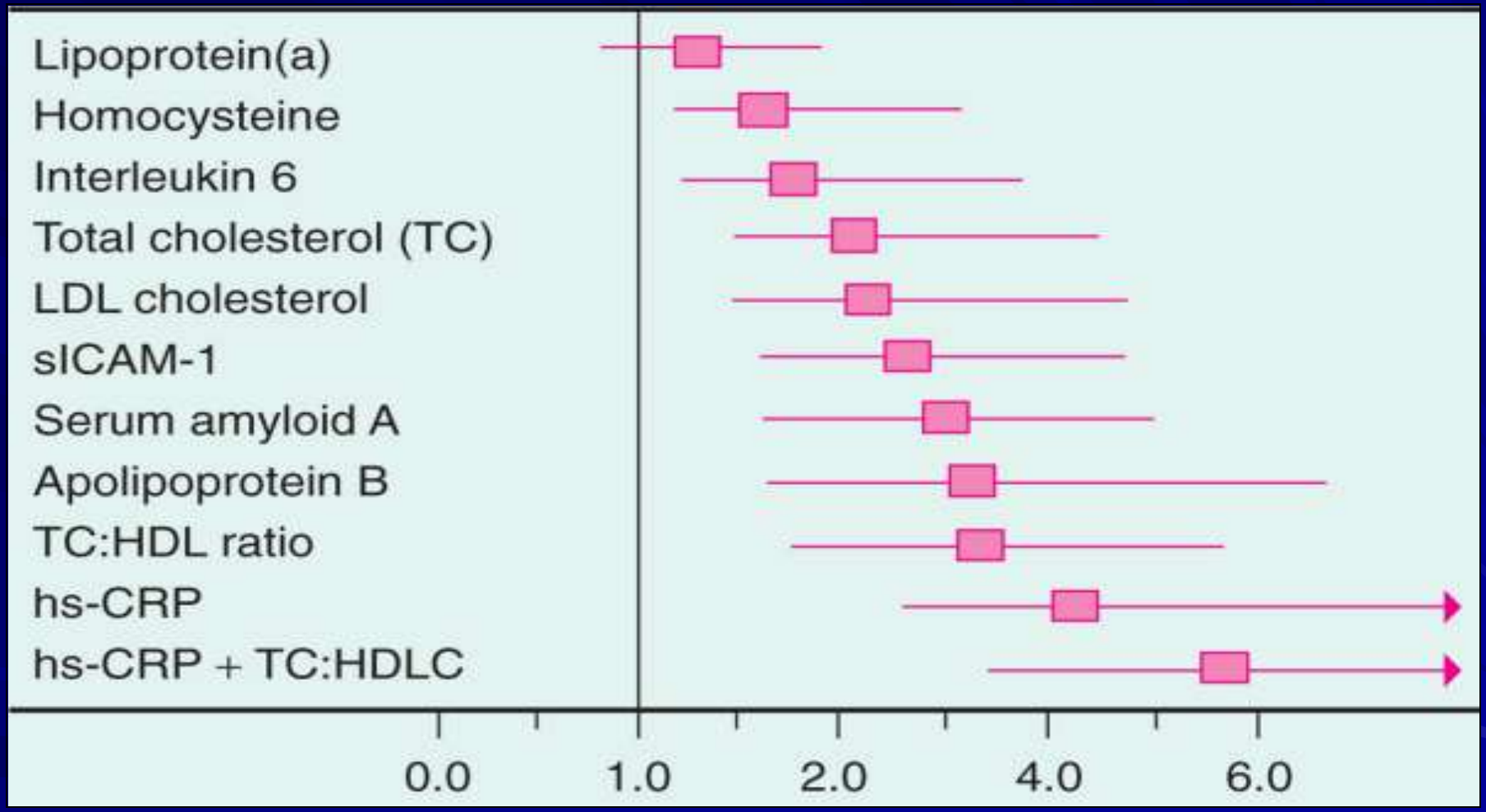
- Blood Suger
- Serum Lipids
- hs-CRP
- Homocysteine
- Fibrinogen
- Lipoprotein (a)



Biomarkers in CAD

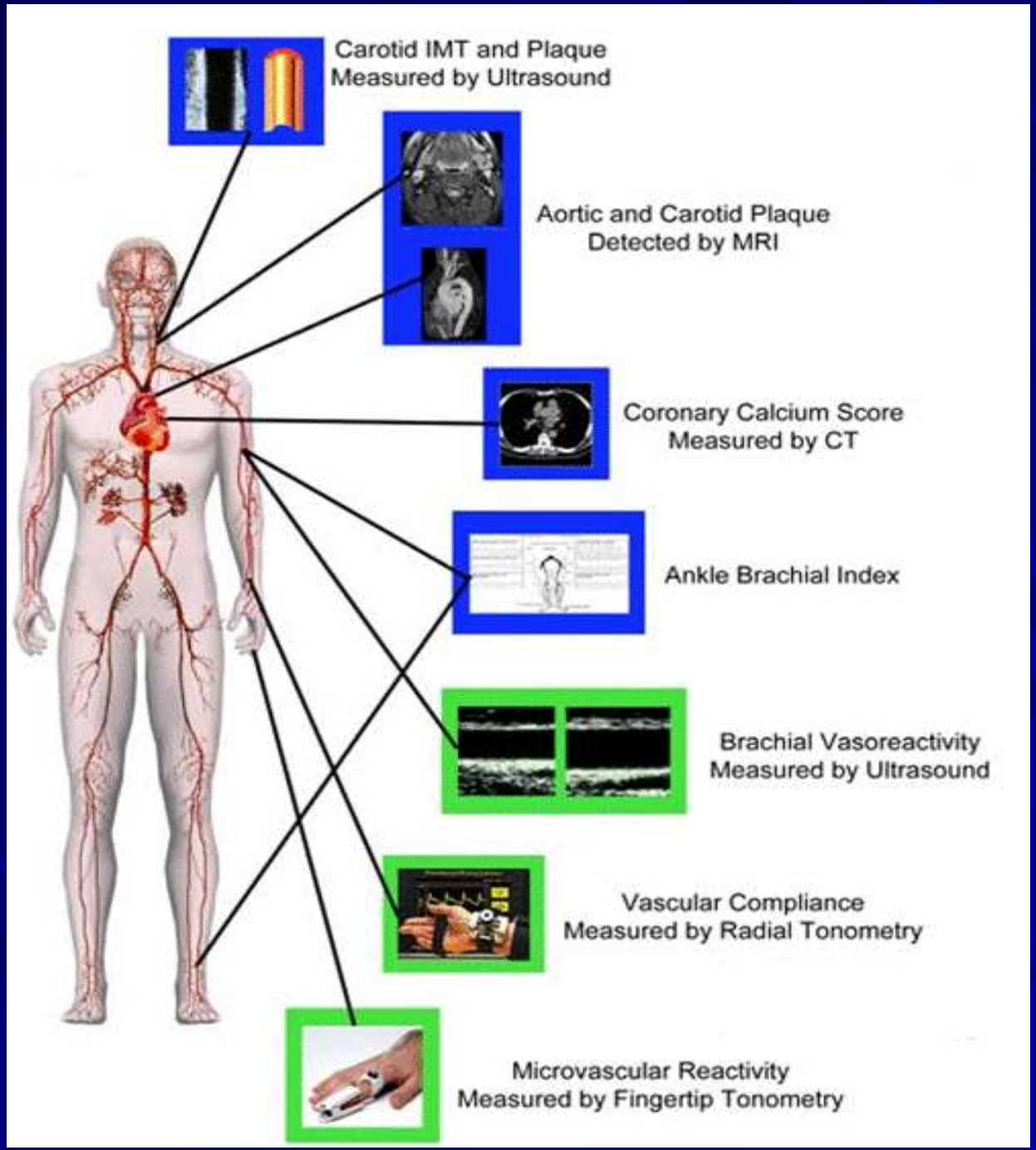


Prognosis with Biomarkers



Relative risks of future myocardial infarction among apparently healthy men

Imaging

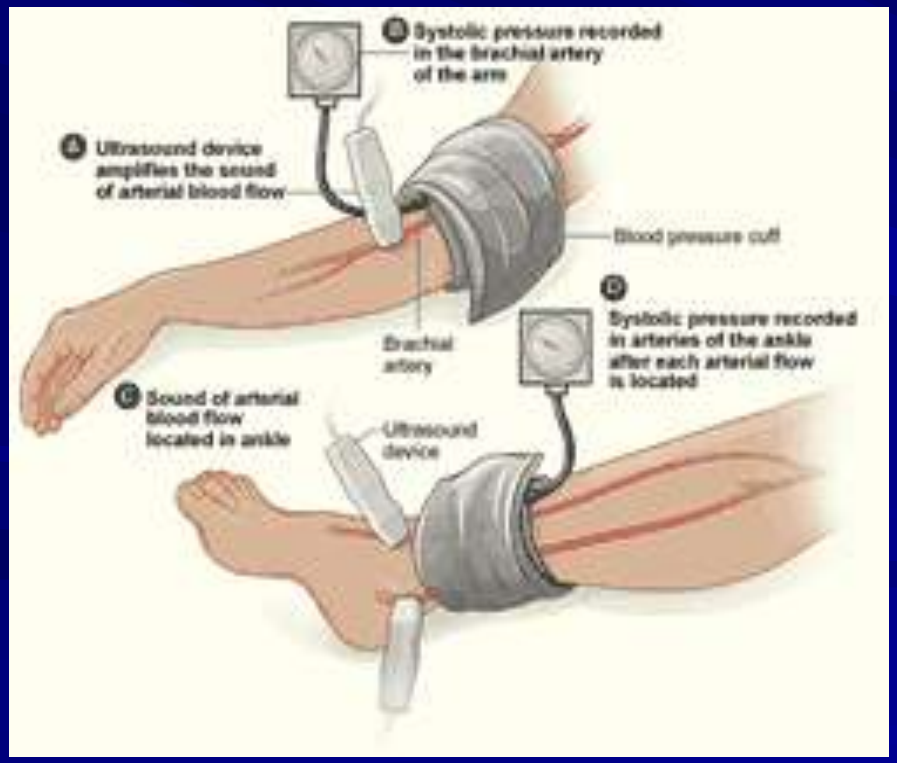


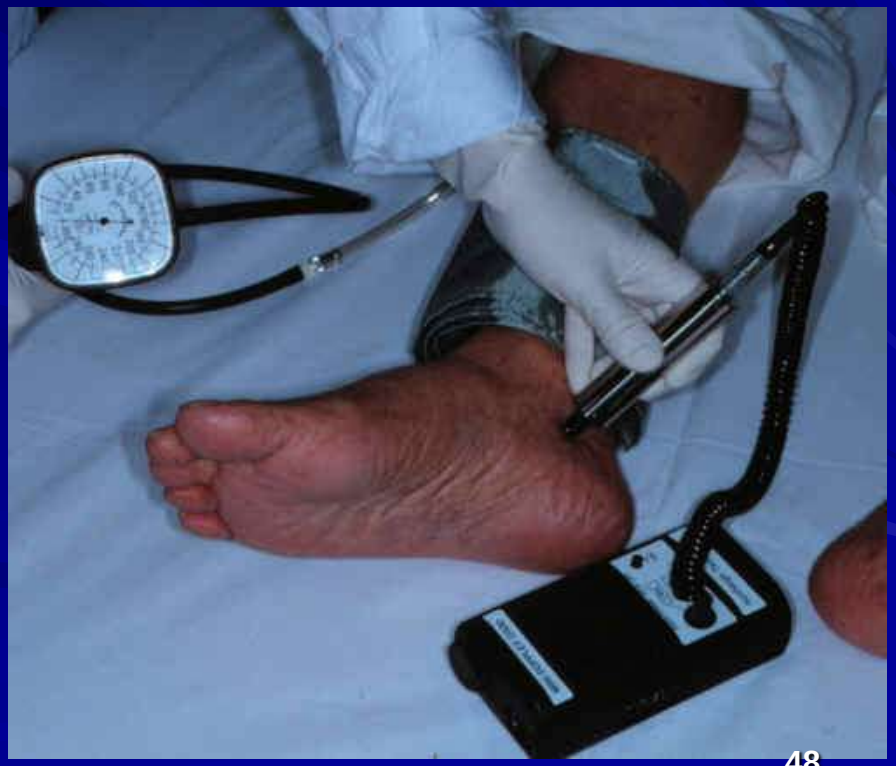
combination of tests to diagnose the extent and spread of atherosclerosis

- ABI
- Stress testing
- CIMT
- CTA
- Doppler study
- IVUS
- MRA
- Angiography
- CCS

Ankle Brachial Index

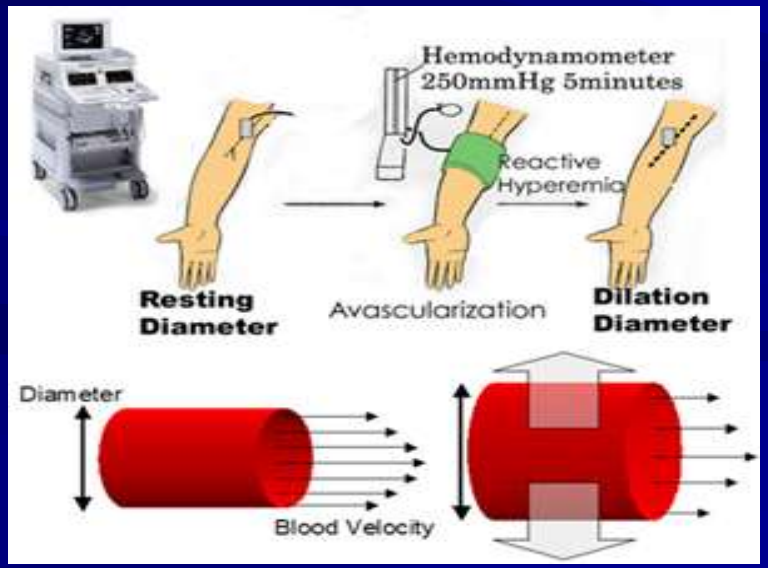
- This index is ratio of SBP measured at ankle to SBP measured at brachial artery
- Normal ABI should be ≥ 1.0

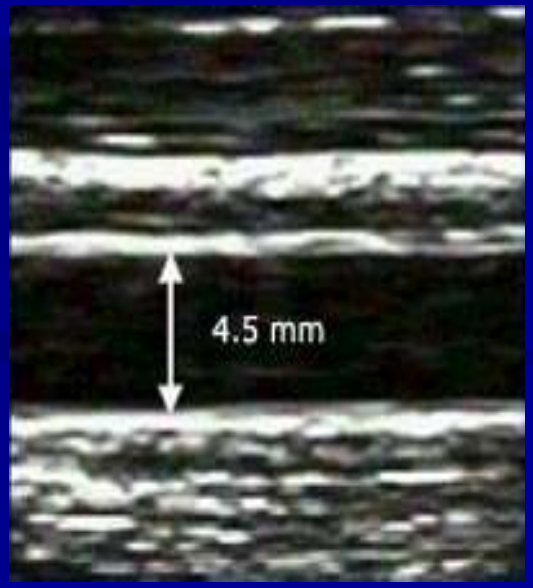


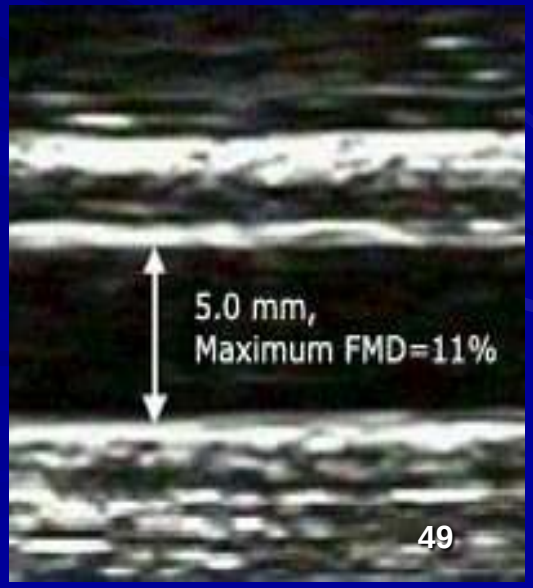


Flow Mediated Dilatation

- Early assessment of atherosclerosis
- Endothelial dysfunction is considered to be the first stage of atherosclerosis.
- Determining efficacy of treatment
- Cuff is inflated to 50 mm Hg above subject's resting systolic pressure and remains inflated for 4 min. The cuff is then deflated and the 2-min image data are acquired

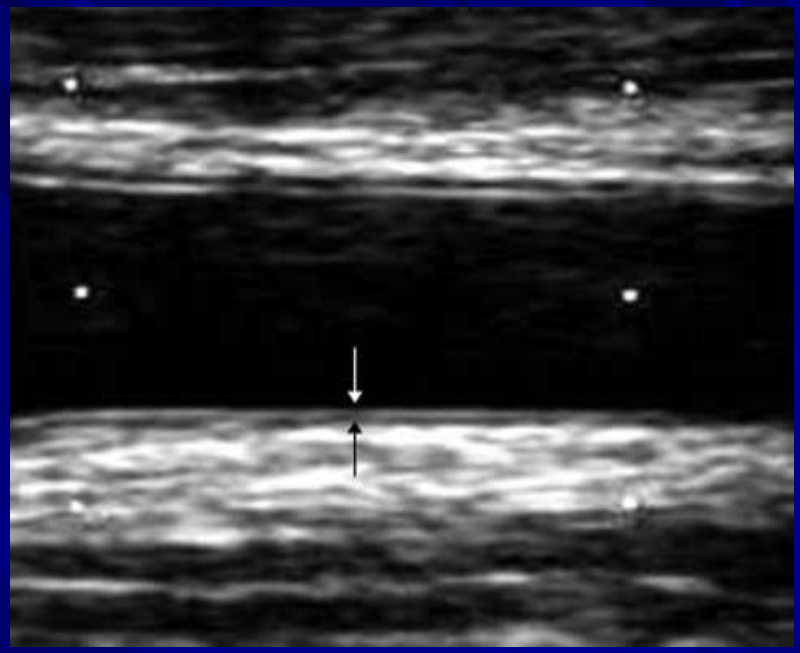


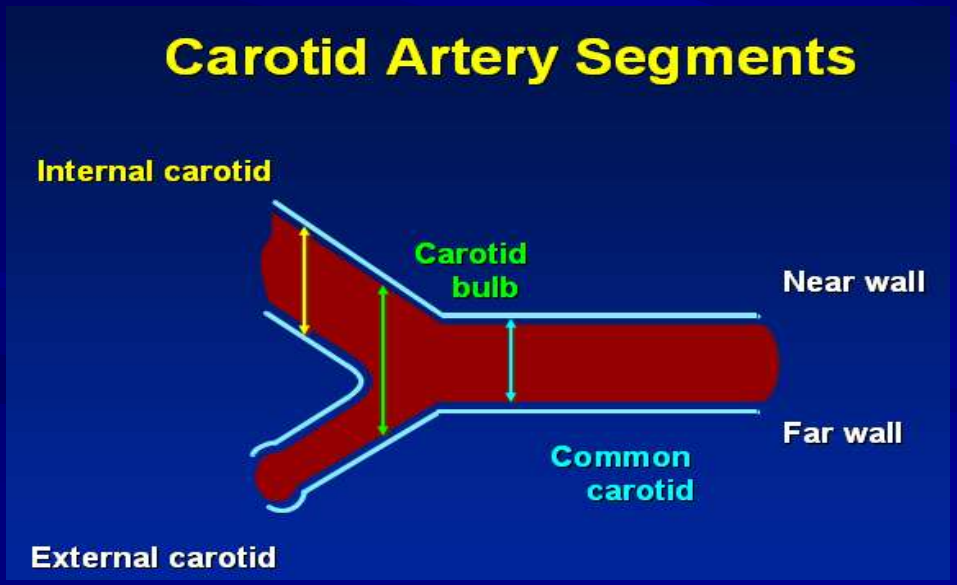


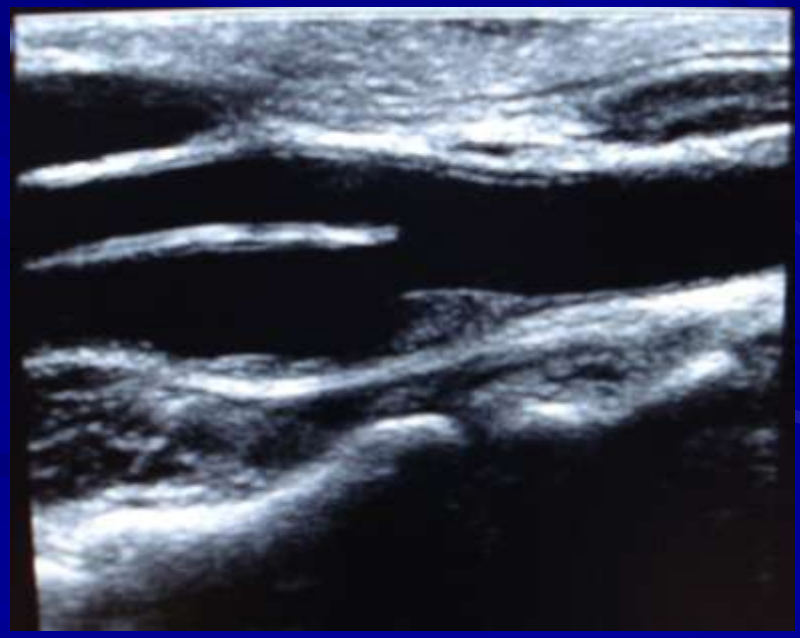


CIMT

- Carotid IMT measurement is a viable predictor of the presence of coronary atherosclerosis and its clinical sequelae



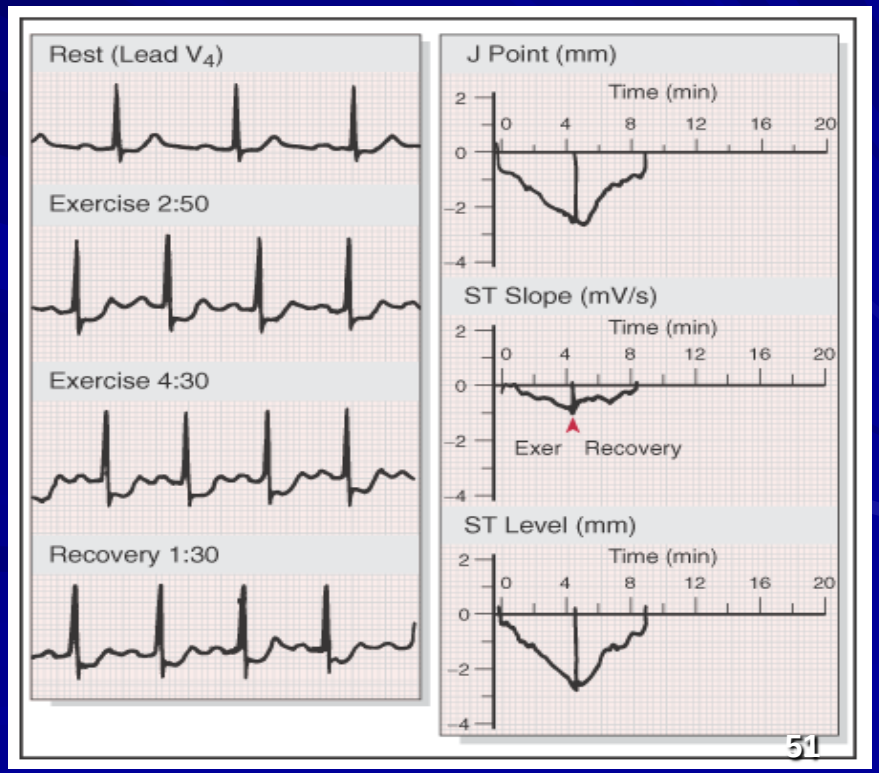




Exercise ECG / TMT

- to assess patients with suspected or proven cardiovascular disease
- to estimate prognosis and to determine functional capacity, the likelihood and extent of CAD and the effects of therapy.

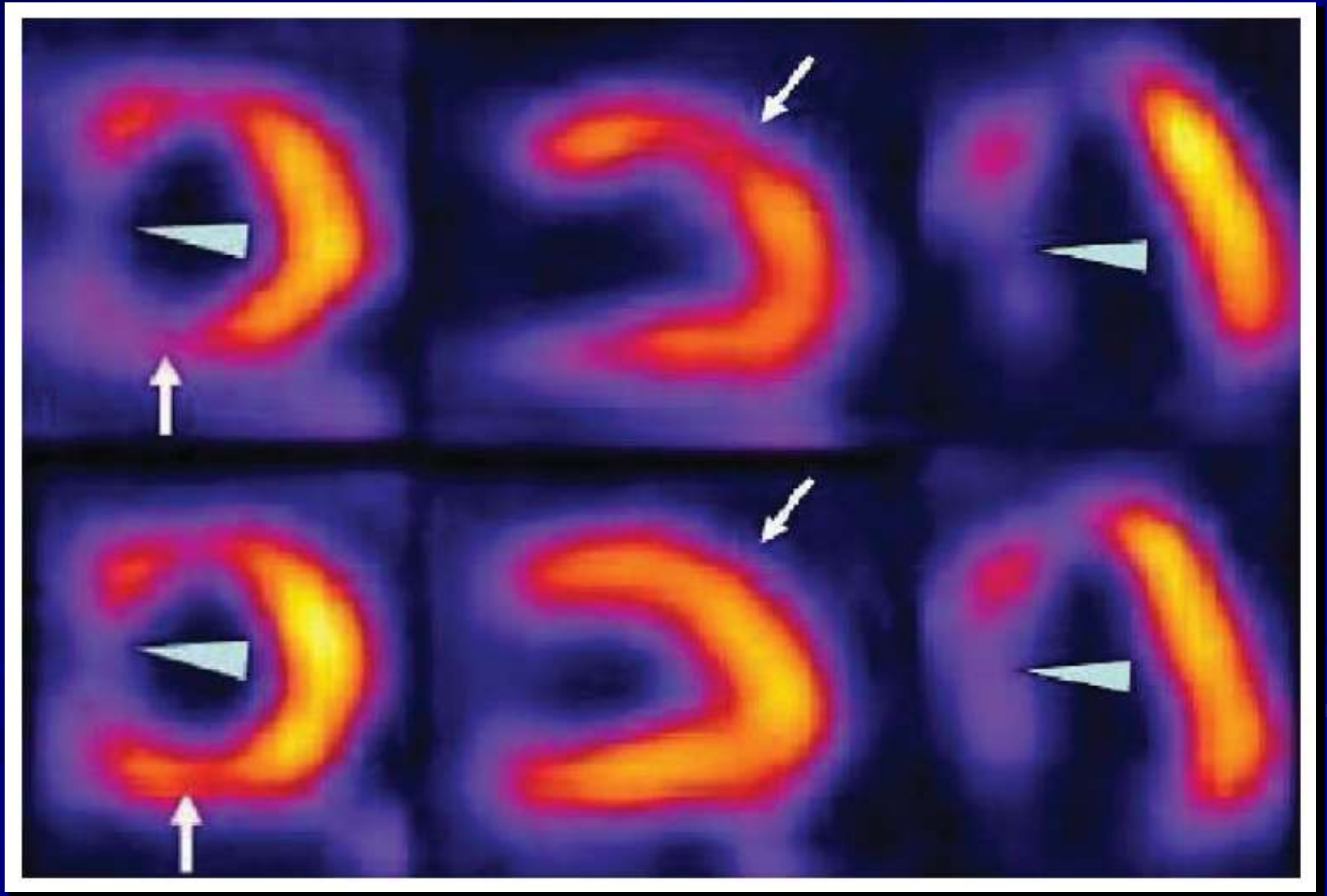
In lead V_4 , the TMT is abnormal early in the test, reaching 0.3 mV (3 mm) of horizontal ST segment depression at the end of exercise. The ischemic changes persist for at least 1 minute and 30 seconds into the recovery phase. This type is consistent with a severe ischemic response.



MYOCARDIAL PERFUSION SCANNING

- Helpful in pt with uninterpretable exercise test
- Accuracy higher than exercise ECG
- scintiscan of myocardium at rest and during stress after iv radioactive isotopes thallium.
- If Perfusion defect present during stress but not rest -reversible myocardial ischemia
- Persistence perfusion defect during both phase -previous MI

Patient with reversible perfusion defect in the inferior and anterior wall and an irreversible perfusion defect in the septum

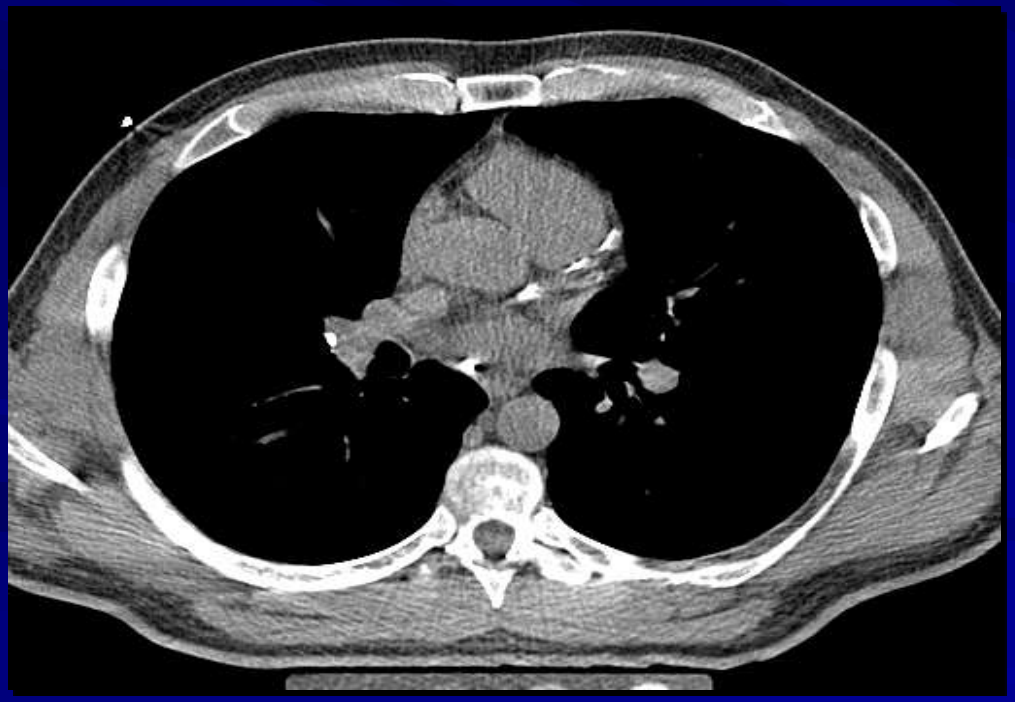


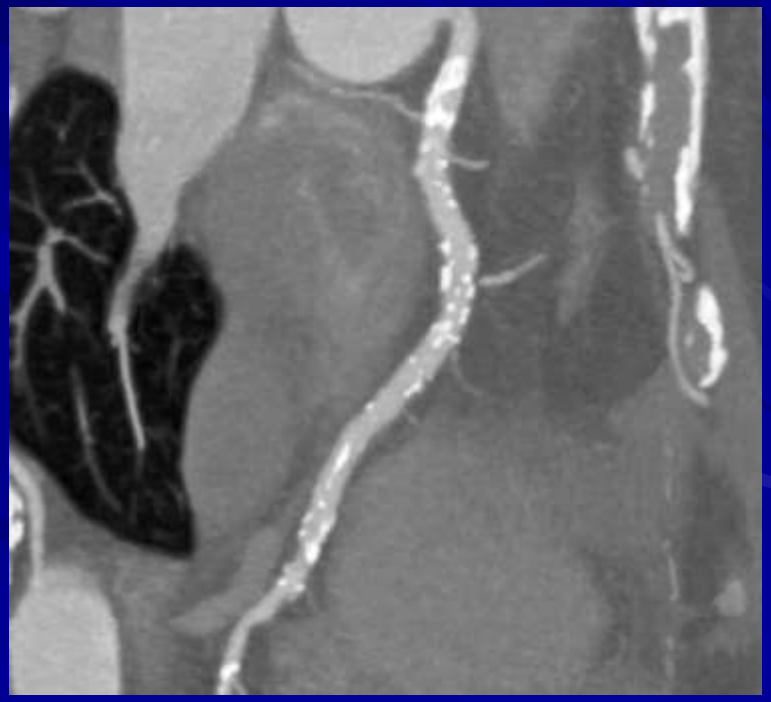
Coronary calcium Scoring

- quantitatively assess coronary calcium using Agatston CCS
- surrogate for plaque burden
- shown to provide powerful prognostic information
- absence of coronary calcium (CCS 0), while not excluding the presence of noncalcified plaque, virtually excludes significant coronary atherosclerosis

Significant Coronary Artery Calcium

(Score >400)





IVUS

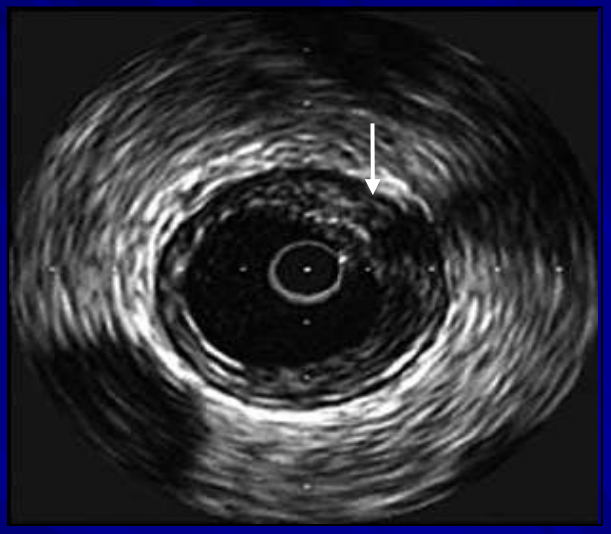
Advantage

- Vessel wall + lumen visualization
- Plaque characterization

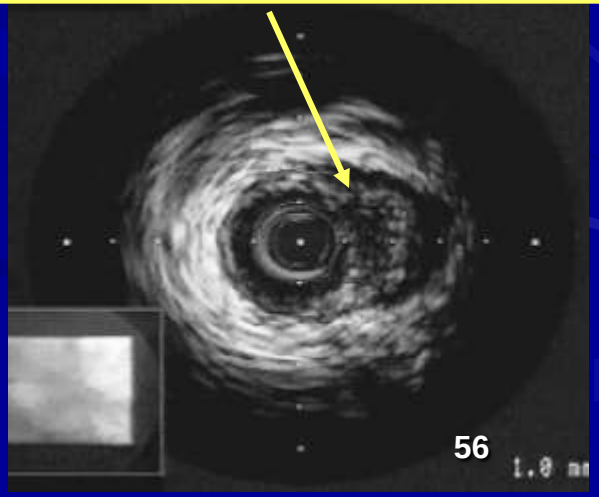
Disadvantage

- Need to instrument vessels
- Limited to proximal segments
- Not as well validated for clinical decision making

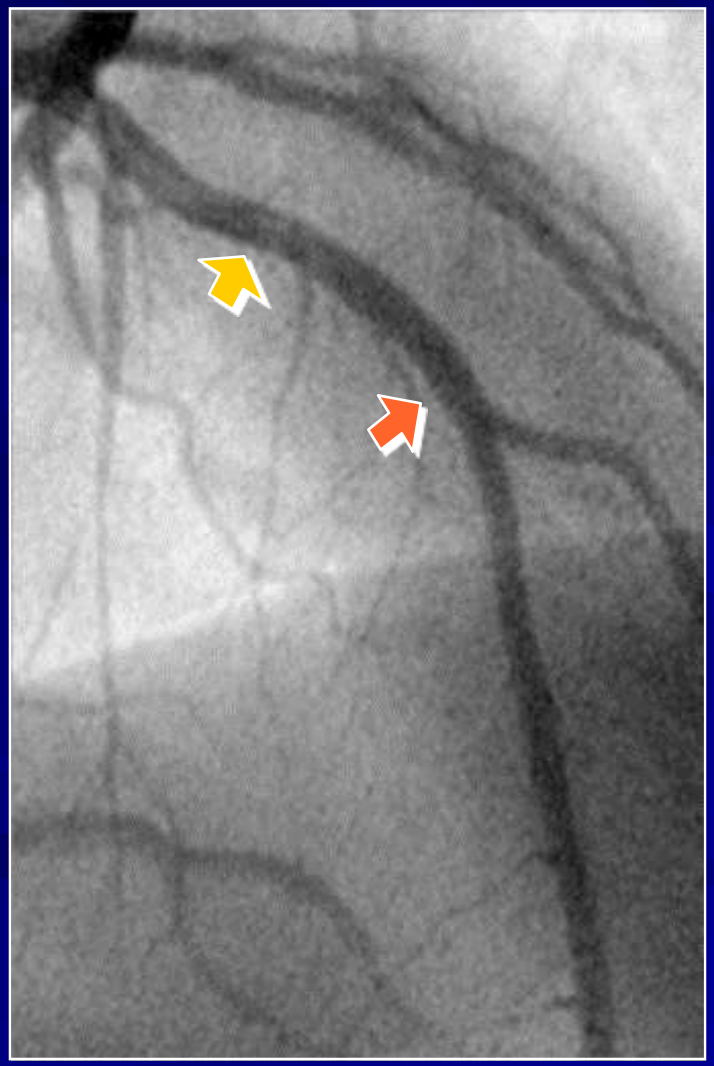
Atheroma

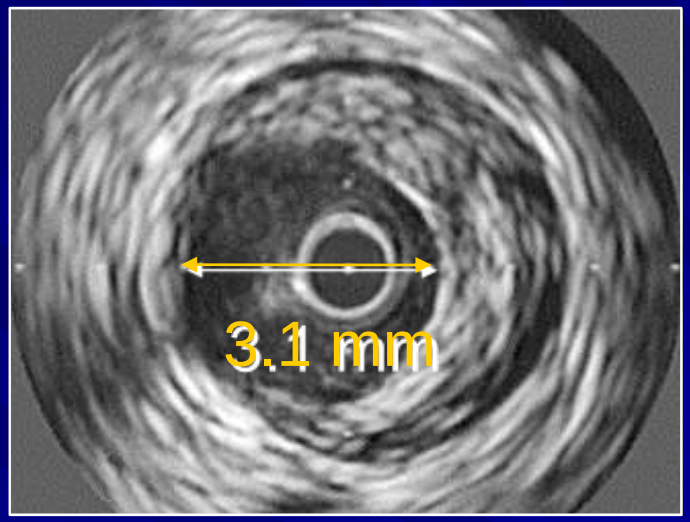


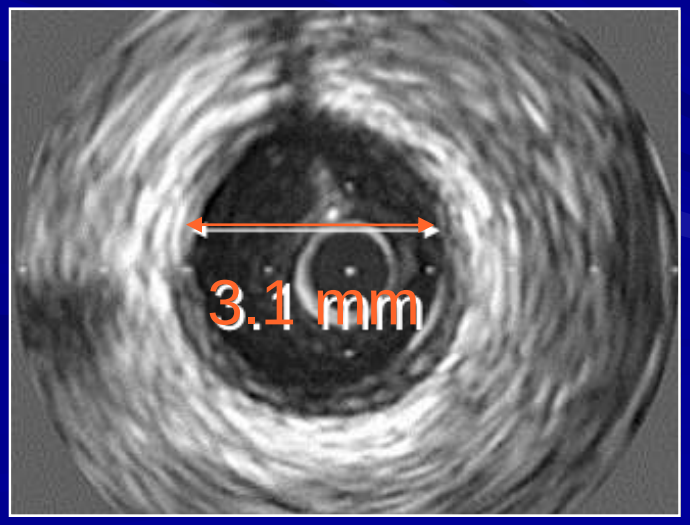
Potentially unstable
Echolucent



Angiography Fails to Depict Coronary Arterial Remodeling



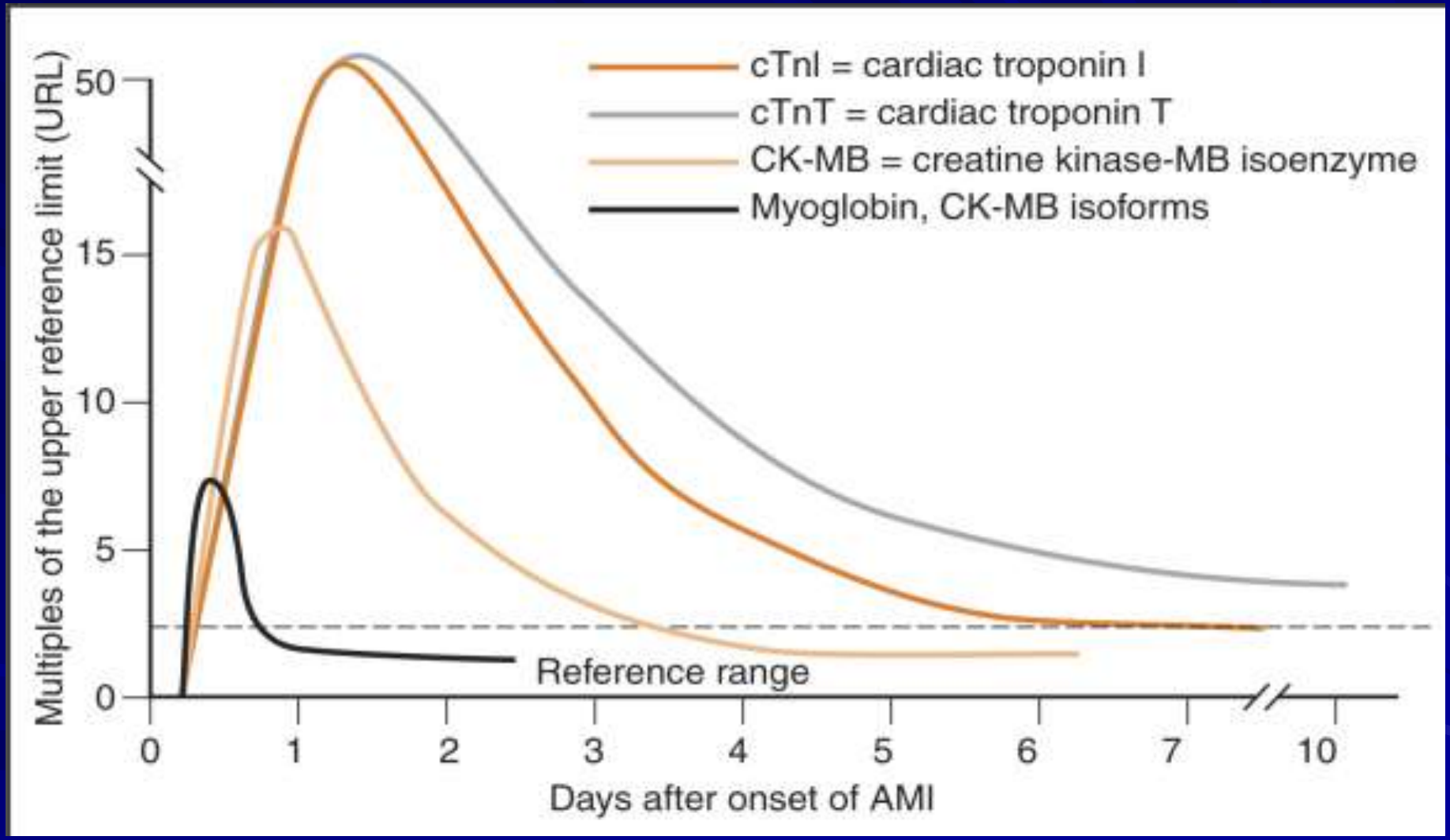




Symptomatic

- Serum biomarkers for cardiac damage
- Serum lipids
- Blood sugar
- ECG
- Echo
- Myocardial perfusion scans
- CTA/ MRA
- CAG

post-acute MI and Biomarkers



Normal - UA Increased - NSTEMI / STEMI

Should be measured at 0hrs, 6-9hrs, 12-24 hrs after admission

Biomarkers of Cardiac Damage

Biomarker	Range of Times to Initial Elevation, h	Mean Time to Peak Elevations (Nonreperfused)	Time to Return to Normal Range
CK-MB	3-12 h	24 h	48-72 h
cTnI	3-12 h	24 h	7-10 d
cTnT	3-12 h	12 h–2 d	7-14 d
Myoglobin	1-4 h	6-7 h	24 h

CK-MB

- Rapid, cost-efficient, accurate assays
- Ability to detect early reinfarction
- Lack of specificity with skeletal muscle disease/injury
- Low sensitivity during early MI (<6 h) or late (>36 h) after symptom onset and for minor myocardial damage

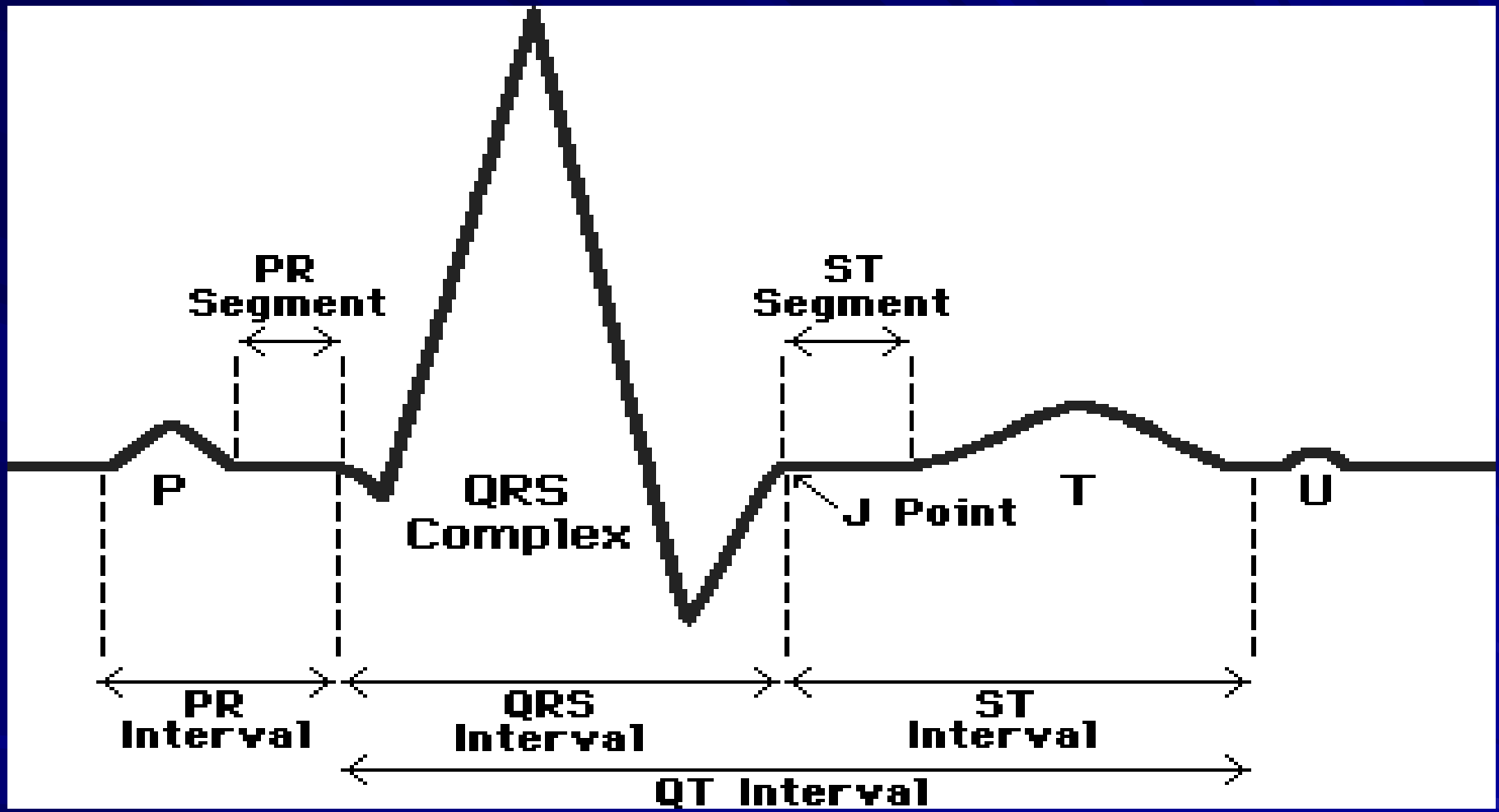
Myoglobin

- High sensitivity
- Useful in early detection of MI
- Detection of reperfusion
- **Most useful in ruling out MI**
- Very low specificity with skeletal muscle injury or disease
- Rapid return to normal

Troponins

- Greater sensitivity and specificity than CK-MB
- **Detection of recent MI up to 2 weeks after onset**
- Detection of reperfusion

ECG

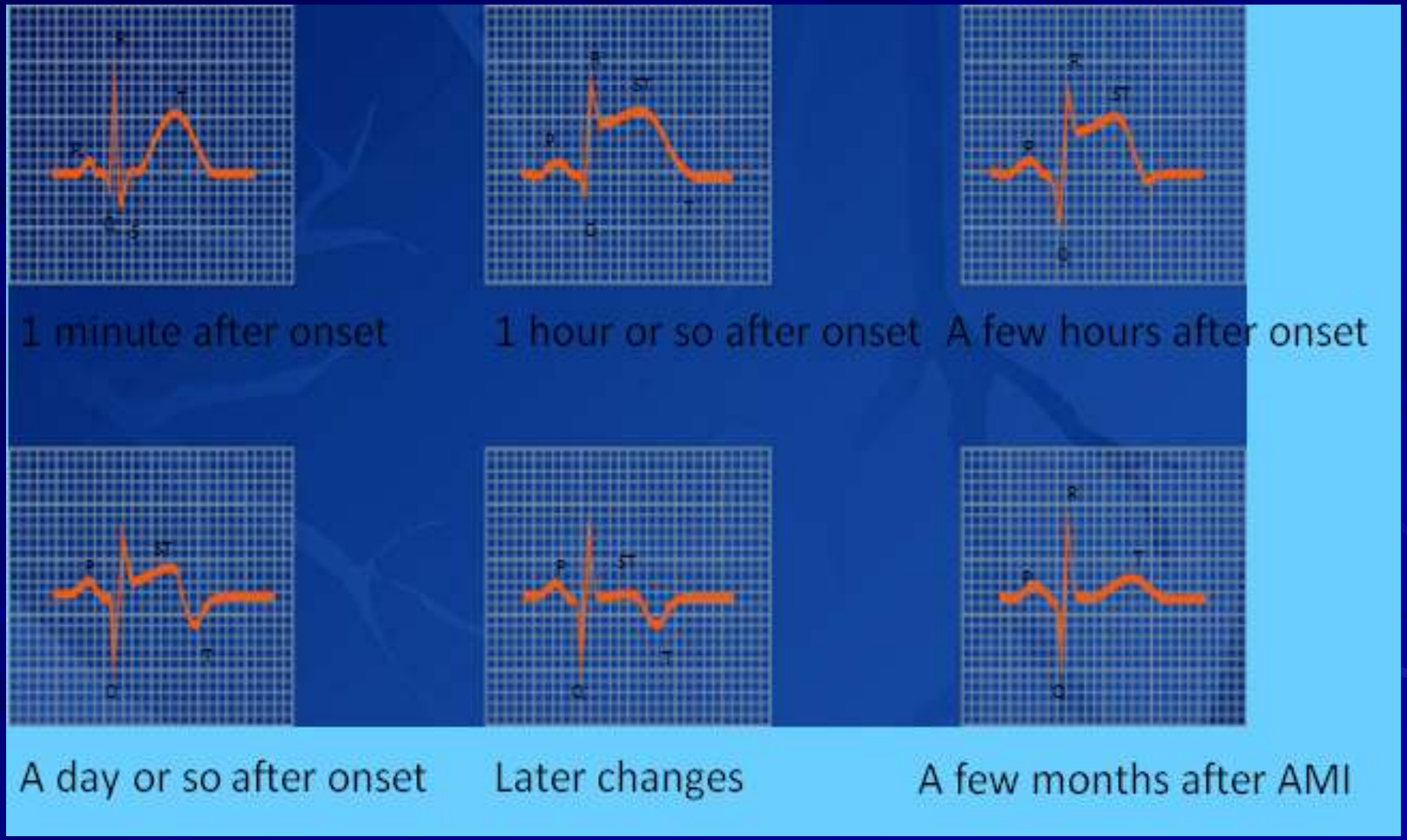


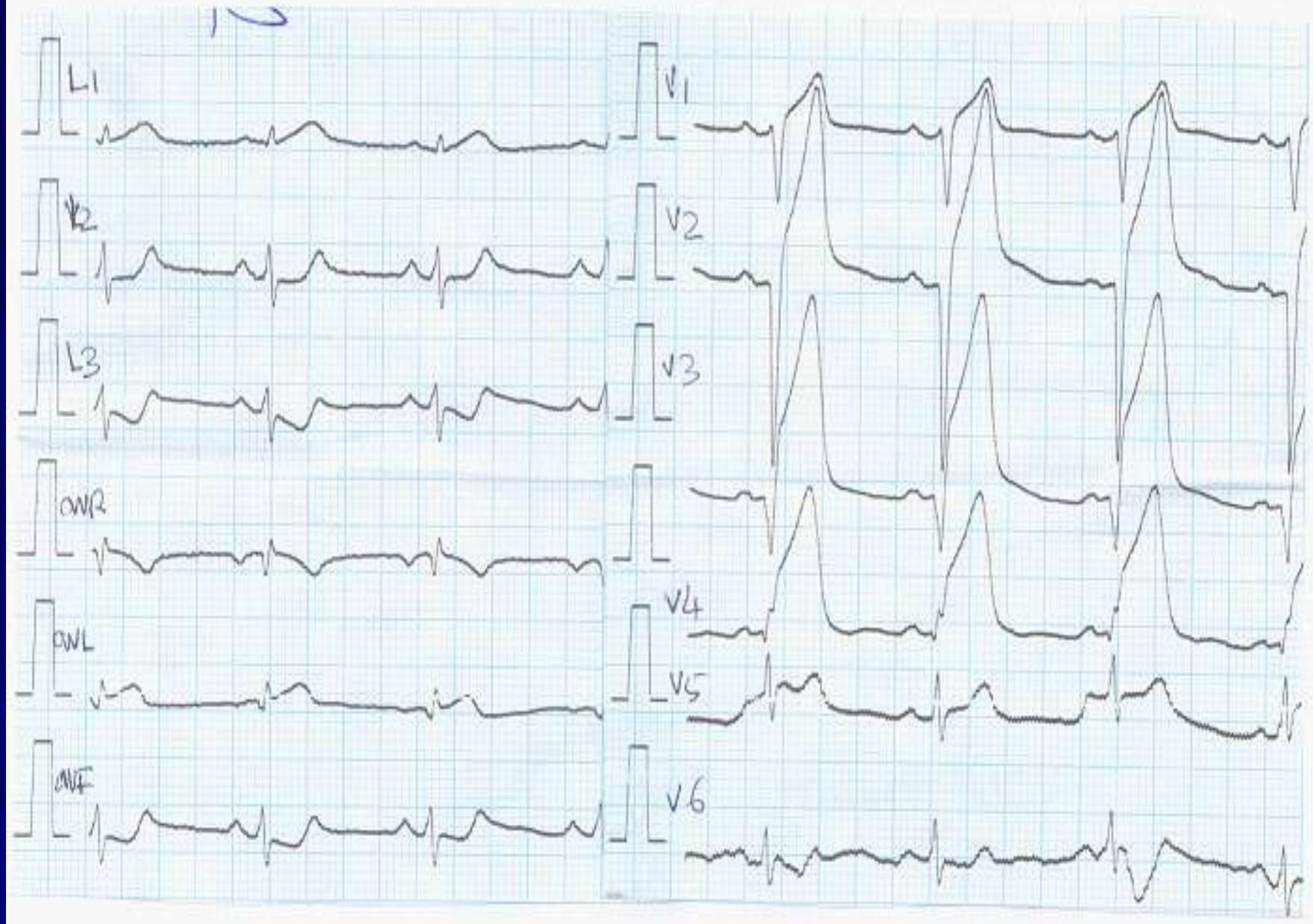
Classic changes of necrosis (Q waves), injury (ST elevation), and ischemia (T wave inversion)

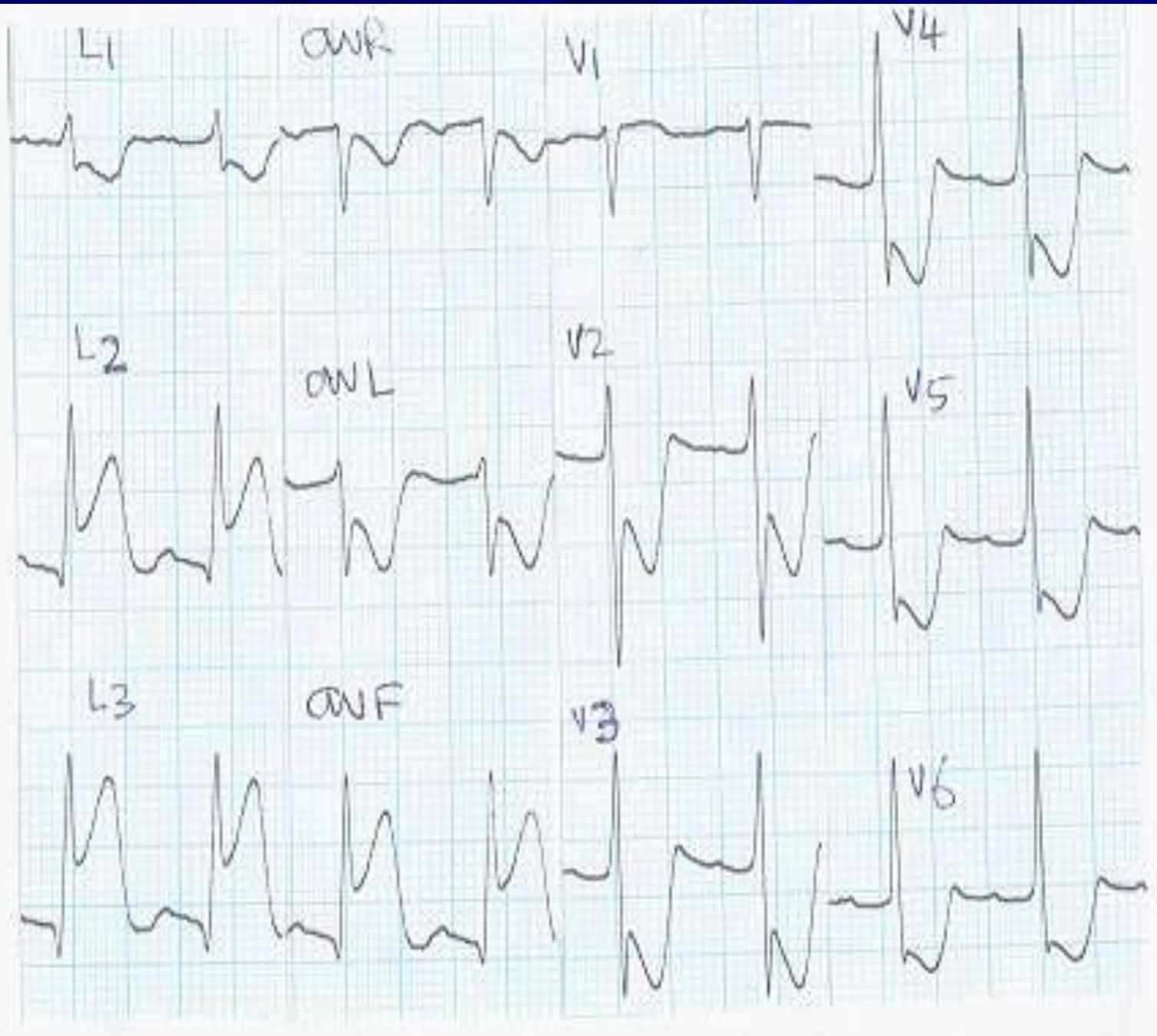
In recovery, the ST segment is earliest change to normalizes, then T wave; the Q wave usually persists for years after infarction

- Hyperacute T Waves (over 50% of preceding R)
- ST-T elevation (>1mm in limb or precordial leads)
- ST depression in Lead V1, Lead V2 (Posterior MI)
- T Wave inversion
- Q Waves (.04 sec and 1/3 height of R Wave)
- New left ventricular strain pattern
- New Left Bundle Branch Block

Sequence of ECG Changes

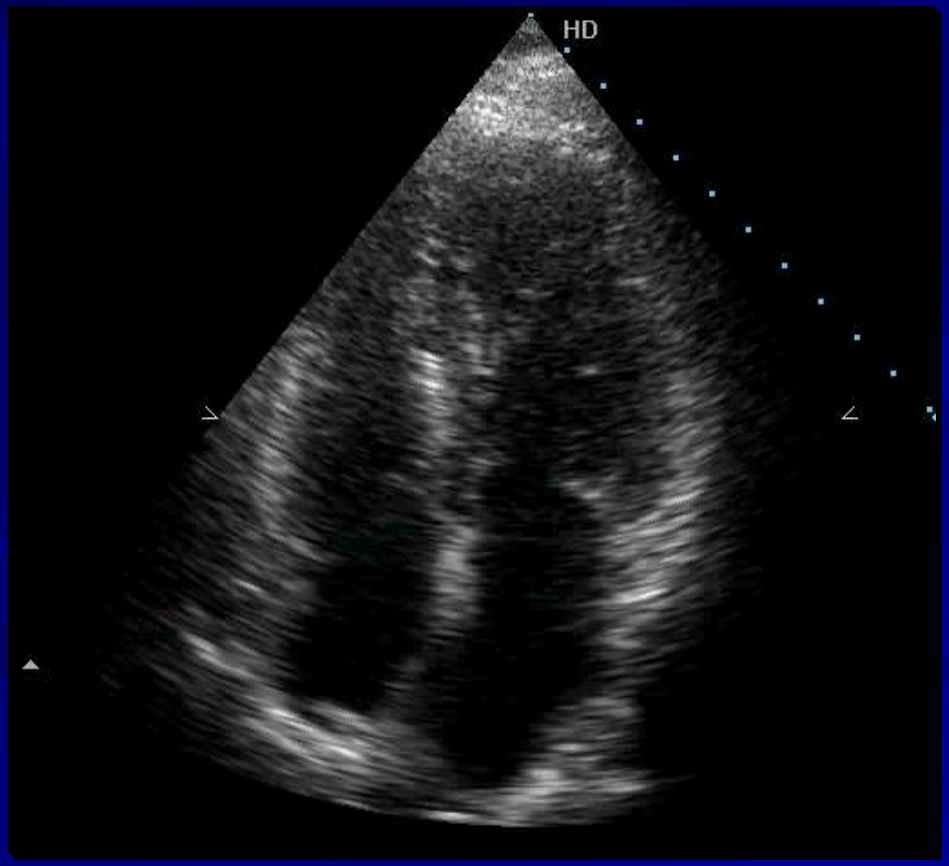


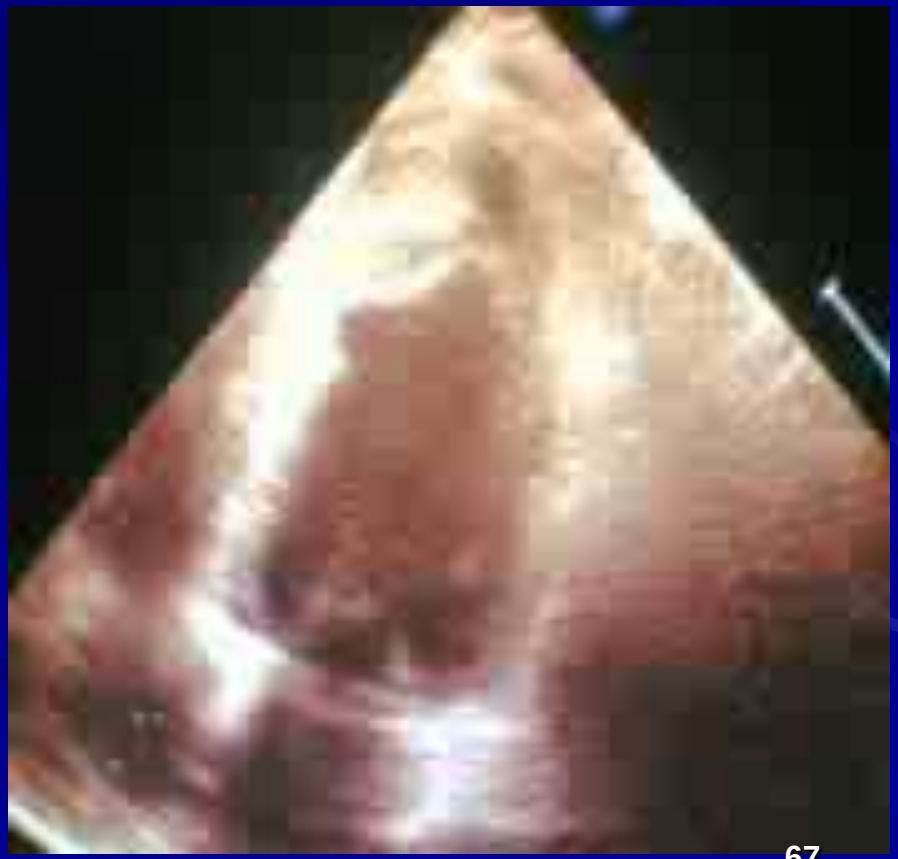

I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6


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

ECHO
Stress ECHO

Alternative to Myocardial perfusion scanning
Superior to exercise ECG

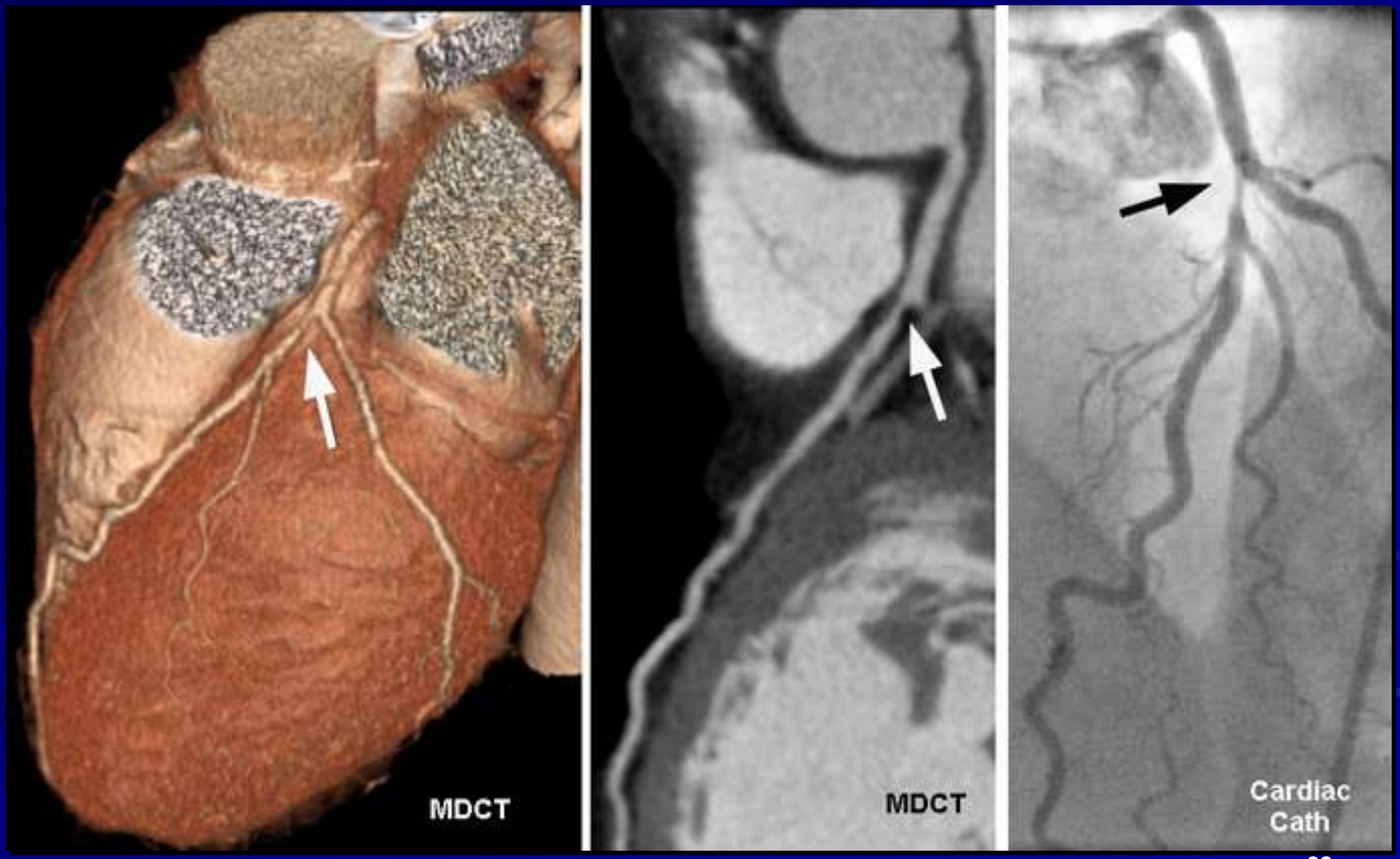




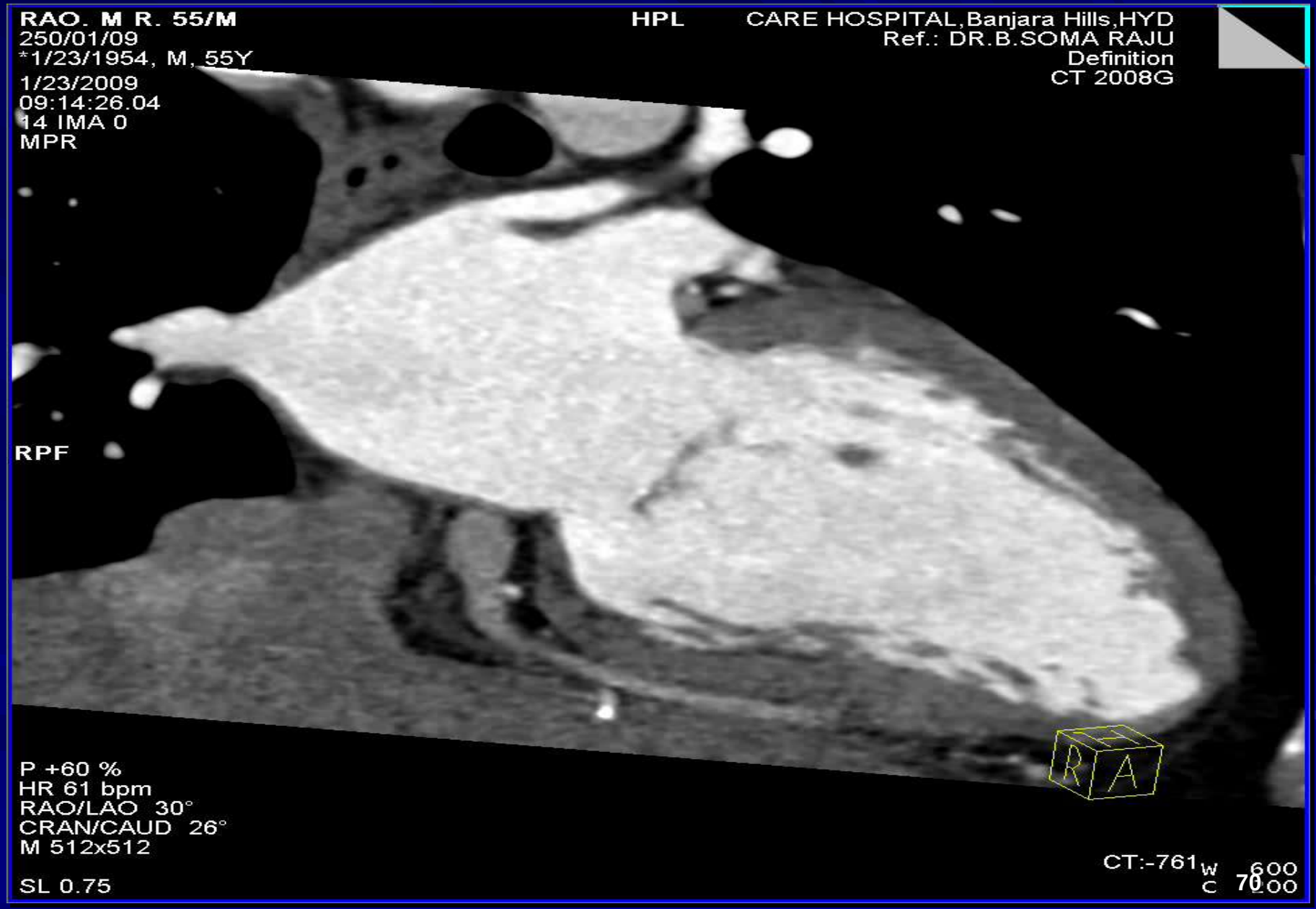
CT Angiography

- assessment of symptomatic patients for the assessment of obstructive disease
- higher radiation dosages contraindicate its use as a screening tool for asymptomatic patients
- demonstrate the morphological consequences of ischemic heart disease
- can assess ventricular function and perfusion
- visualizing coronary arteries

CT Angiography

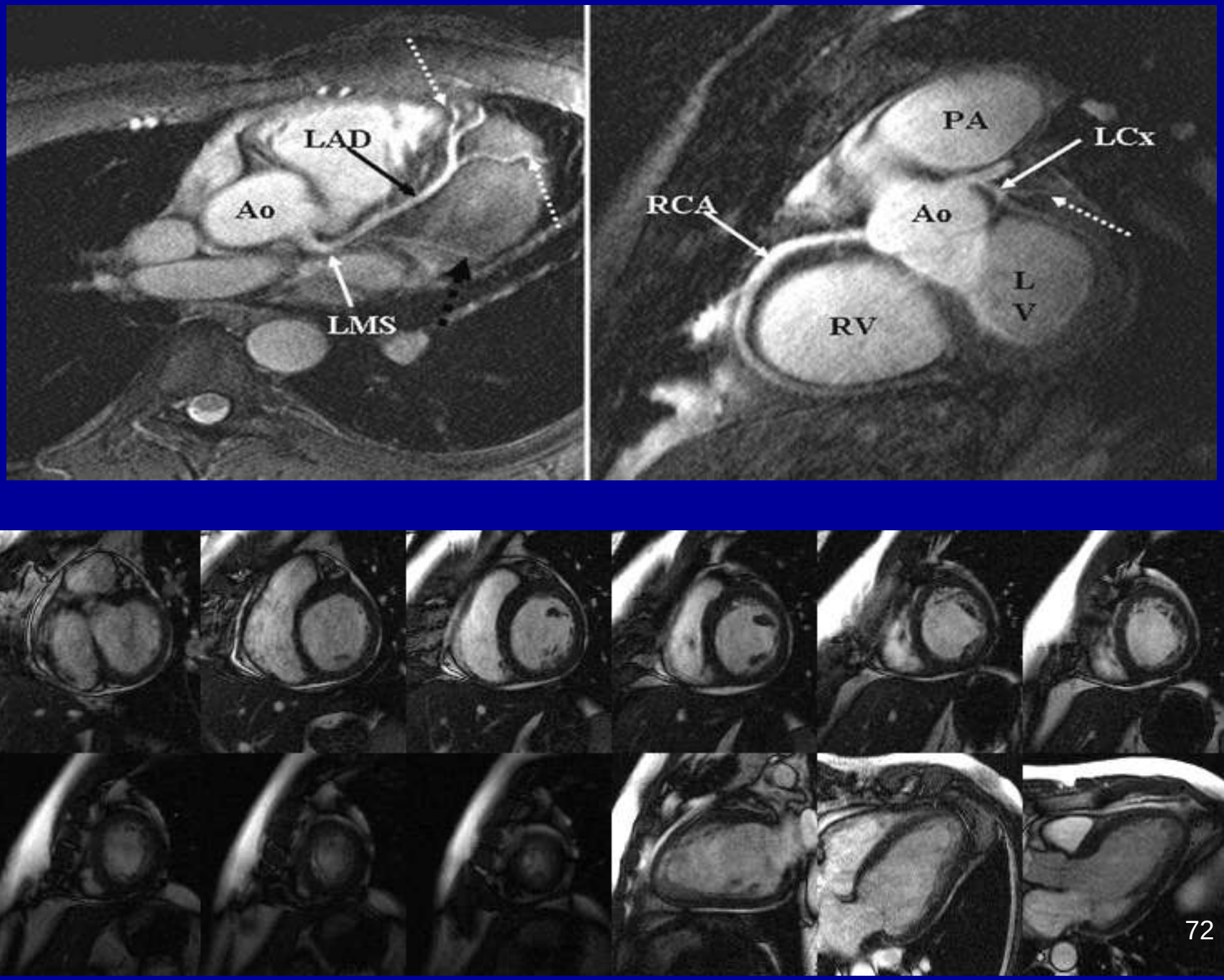


CTA


RAO. M R. 55/M
250/01/09
*1/23/1954, M, 55Y
1/23/2009
09:14:26.04
14 IMA 0
MPR
HPL
CARE HOSPITAL,Banjara Hills,HYD
Ref.: DR.B.SOMA RAJU
Definition
CT 2008G
RPF
P +60 %
HR 61 bpm
RAO/LAO 30°
CRAN/CAUD 26°
M 512x512
SL 0.75
R
A
CT:-761
W 600
C 70

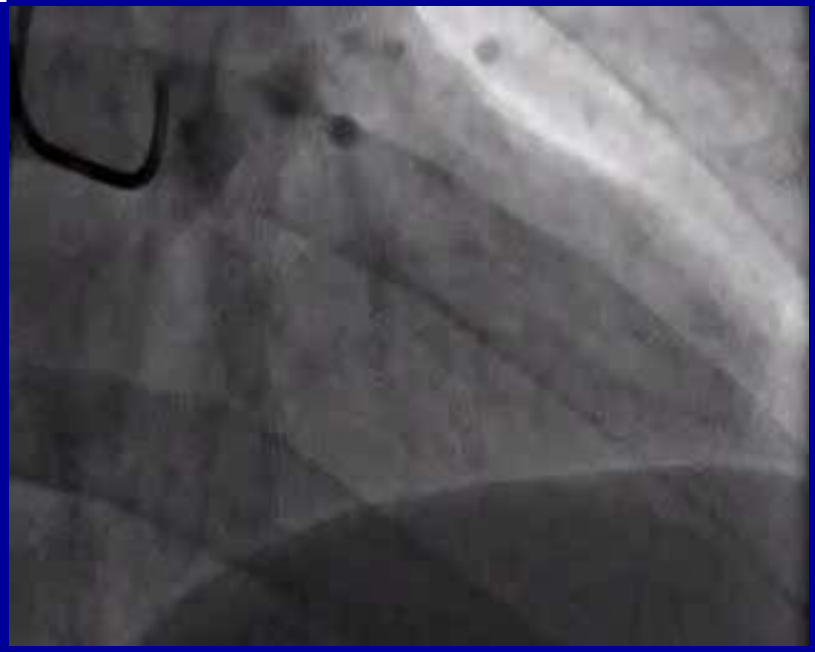
MRA

- Assessment of Ventricular volumes, mass, function
- Assessment of myocardial infarction and viability
- Stress ventriculography
- Coronary angiography and flow
- Identification of plaque components
- Non-invasive and no radiation
- Not useful for screening purpose


LAD
Ao
LMS
PA
LCx
RCA
Ao
L
V
RV

Coronary angiography

- Provide detailed information about the extent & nature of CAD
- Presence of dynamic coronary vascular lesions, such as spasm or thrombosis
- Consequences of CAD, - IMR or LV dysfunction
- Quantification of severity of both diastolic and systolic dysfunction



Treatment (Stable Angina)

- Identification and Treatment of Aggravating Conditions - AV disease, HCM, anemia
- Treatment of Risk Factors – HTN, smoking
- Drug Therapy
- PCI/ CABG
- Enhanced ECP
- Transmyocardial laser revascularization

Treatment (Stable Angina)

Nitrates

– systemic venodilation, reduction in LV EDV/P, reducing oxygen requirements; dilation of epicardial coronary vessels; increased blood flow in collateral vessels, improve exercise tolerance

– relieve ischemia in UA, Prinzmetal's variant angina

Beta-blockers

- reduce myocardial oxygen demand, inhibiting increases in heart rate, BP, contractility, esp during exercise, relief of angina, ischemia

- reduce mortality and reinfarction in patients after MI

Treatment (Stable Angina)

Calcium antagonists

- Coronary vasodilators, variable reductions in myocardial oxygen demand, contractility, BP
- Indicated when BB contraindicated, poorly tolerated, ineffective. sick-sinus syndrome, AV conduction disturbances, symptomatic PAD, Prinzmetal's angina
- Amlodipine and beta blockers have complementary actions on coronary blood supply and myocardial oxygen demands.
- Beta blockers have shown to improve life expectancy following acute MI while CCB have not

Treatment (Stable Angina)

- Aspirin/ Clopidogrel
- (ACE) inhibitors - post MI; HTN, chronic IHD, DM, diabetes, LV dysfunction
- Potassium channel activators- nicorandil - open ATP-sensitive potassium channels in myocytes
- Metabolic modulators

Trimetazidine - exert anti-ischaemic properties without affecting myocardial oxygen consumption and blood supply, affects myocardial substrate utilization by shifting energy production from FFA to glucose oxidation.

Ranolazine - symptomatic chronic angina max Rx, inhibits late inward sodium current (I_{Na})

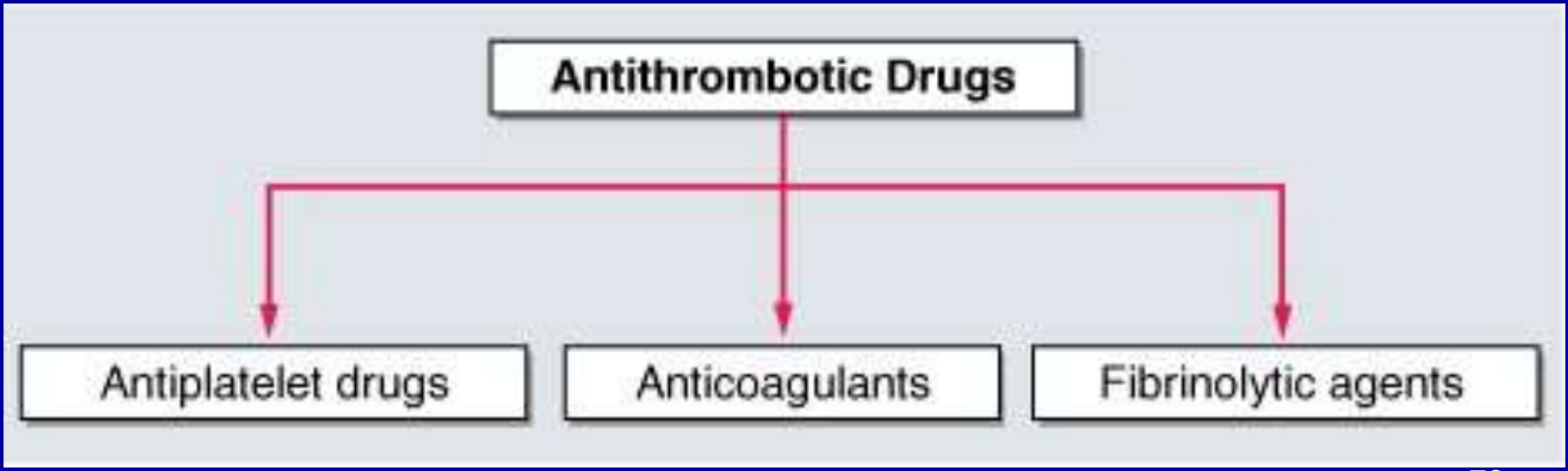
Perhexiline, etomoxir

Treatment Goals (ACS)

- Restore blood flow to prevent infarct expansion - STK/ PCI/ CABG
- Prevent death, complications
- Relieve ischemic chest discomfort
- Prevent coronary artery reocclusion

Early Pharmacotherapy for ACS

- Intranasal oxygen
- IV NTG - control ischemia
- Morphine
- β-blocker - control ischemia



Antiplatelet

- Thromboxane A2 inhibitor
  - -- aspirin
- ADP receptor blockers
  - – Irreversible (ticlopidine, clopidogrel, prasugrel)
  - – Reversible (cangrelor)
- Phosphodiesterase inhibitors
  - – Dipyridamol
  - – Cilostazol
- Glycoprotein IIb/IIIa antagonists indicated for patients undergoing 1 ˚ PCI in combination with ASA, clopidogrel, & UFH
  - – Abciximab
  - – Tirofiban
  - – Eptifibatide

Anticoagulant

- UFH
- LMWH (enoxaparin, dalteparin)
- Factor Xa Inhibitor (fondaparinux)
  - alternative to UFH in patients not undergoing reperfusion
  - receiving fibrinolytics
  - not recommended for use alone in 1˚ PCI
- DTI (Lepirudin, Bivalirudin, Argatroban)
  - option in patients undergoing planned 1˚ PCI
  - Inhibit clot-bound & circulating thrombin
  - Antiplatelet activity
- VKA (warfarin)

Fibrinolytic

Fibrin non-specific agents

- Streptokinase
- Anistreplase
- Urokinase

Fibrin-specific agents

- rt-PA (alteplase)
- Variants of t-PA
  - Substitution (monoteplase, tenecteplase)
  - Deletion (reteplase, lanoteplase, pamiteplase)

Comparison of Fibrinolytic Agents

Agent	Fibrin Specificity	Complete Perfusion at 90 Minutes	Bleeding risk	Administration
Streptokinase	+	35%	+++/+	Infusion over 60 minutes
Alteplase	+++	50-60%	++/++	Bolus followed by infusions over 90 minutes
Reteplase	++	50-60%	++/++	Two bolus doses, 30 minutes apart
Tenecteplase	++++	50-60%	+/++	Single bolus dose

Contraindications to Fibrinolysis

Absolute contraindications

– active internal bleeding (not including menses)

– previous intracranial hemorrhage at any time

– ischemic stroke within 3 months

– intracranial neoplasm

– structural vascular lesion (e.g., AVM)

– suspected aortic dissection

– closed head or facial trauma within 3 months

Relative Contraindications

- uncontrolled HTN (BP > 180/110 mm Hg)
- ischemic stroke > 3 months
- dementia
- intracranial pathology
- current anticoagulant use
- bleeding diathesis
- traumatic or prolonged CPR (> 10 min)
- major surgery (< 3 wks)
- noncompressible vascular puncture
  - recent liver biopsy
  - carotid artery puncture
- recent internal bleeding (within 2 to 4 wks)
- previous streptokinase use (> 5 days) or prior allergic reaction
- pregnancy
- active peptic ulcer
- history of severe, chronic, poorly controlled HTN

Successful thrombolysis

Clinical

Resolution of chest pain

ECG

> 50 % decrease in max ST elevation at 90 minutes after start of STK

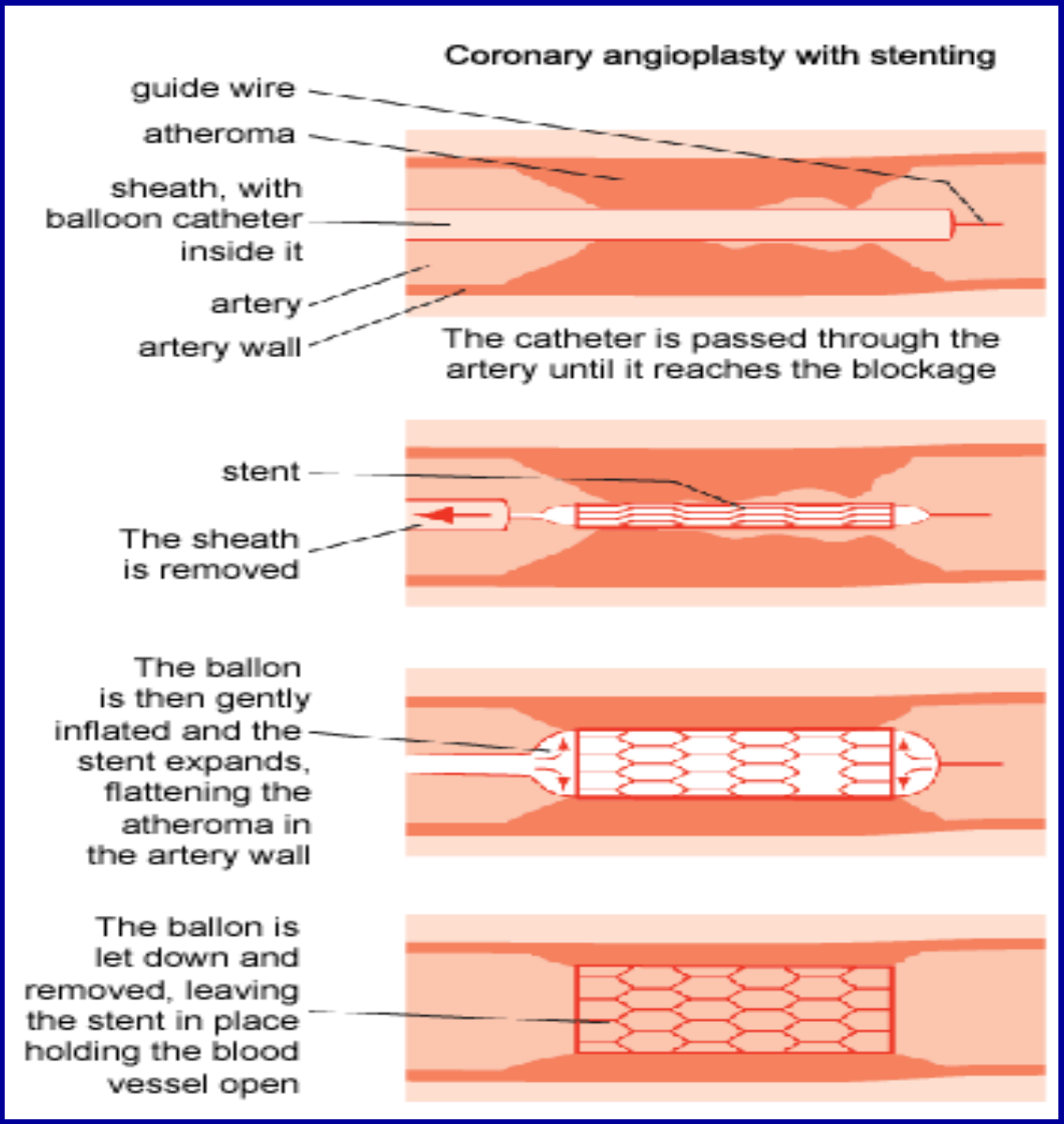
Enzymes

Early peak (3 hrs) of CK-MB

CAG

TIMI III flow

PCI



Percutaneous Coronary Interventions

- Balloons 10%
- Stents 80%
- Atherectomy 10%

PCI

- As an alternative to thrombolytic therapy in patients with AMI and STEMI or new or presumed new LBBB who can undergo angioplasty of the infarct artery <12 hr from the onset of ischemic symptoms or >12 hr if symptoms persist, if performed in a timely fashion (performance standard: balloon inflation within 90 ± 30 min of hospital admission) by individuals skilled in the procedure
- In patients who are within 36 hr of an acute STEMI/Q wave or new LBBB MI who develop cardiogenic shock, are younger than 75 yr, and revascularization can be performed within 18 hr of the onset of shock by individuals skilled in the procedure
- As a reperfusion strategy in candidates who have a C/I to thrombolytic therapy
- Objective evidence for recurrent infarction or ischemia

PCI

- Cardiogenic shock or hemodynamic instability
- Recurrent angina without objective evidence of ischemia or infarction
- Angioplasty of the infarct-related artery stenosis within hours to days (48 hr) following successful thrombolytic therapy in asymptomatic patients without clinical and/or inducible evidence of ischemia
- Spontaneous or provocable myocardial ischemia during recovery from infarction
- Patients with LV ejection fraction <0.4, CHF, or serious ventricular arrhythmias
- All patients after a non-Q-wave MI

CABG

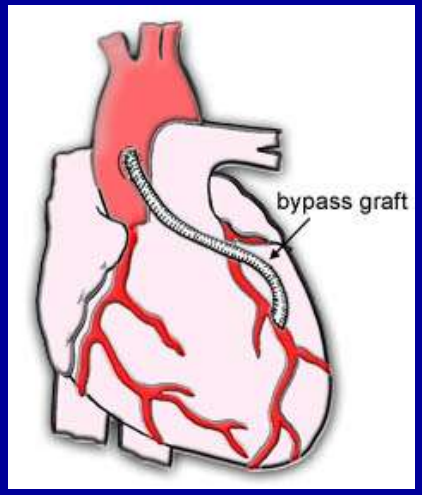
Surgery reroutes or bypasses blood around clogged arteries

Grafts Arterial - Radial artery, internal mammary artery

Venous – Long saphenous vein

Indications

- Triple vessel disease
- Left Main stem disease
- Failed PCI
- Diffuse disease not amenable to PCI
- Severe LV dysfunction or DM

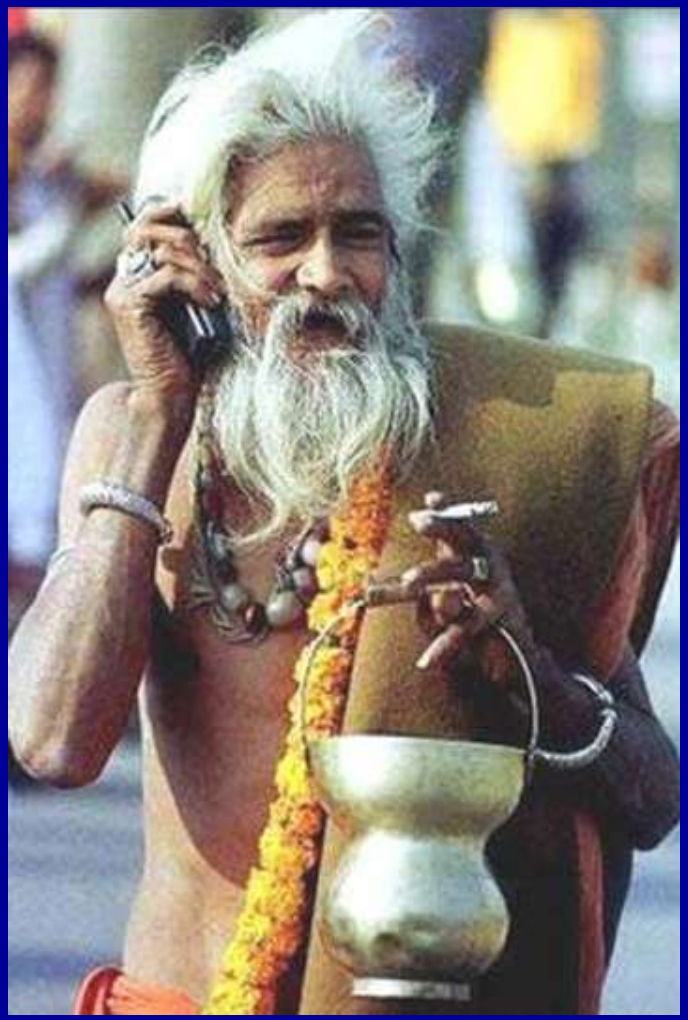


•Patients with ACS (NSTEMI and STEMI) need early invasive strategy

•PCI or CABG depends upon anatomy of vessels and availability of facilities and expertise

PCI vs STK

- STEMI patients should receive either fibrinolysis or primary PCI within 3 hrs of symptom onset
  - PCI: preferred treatment in capable centers/ high risk patients/ failed STK

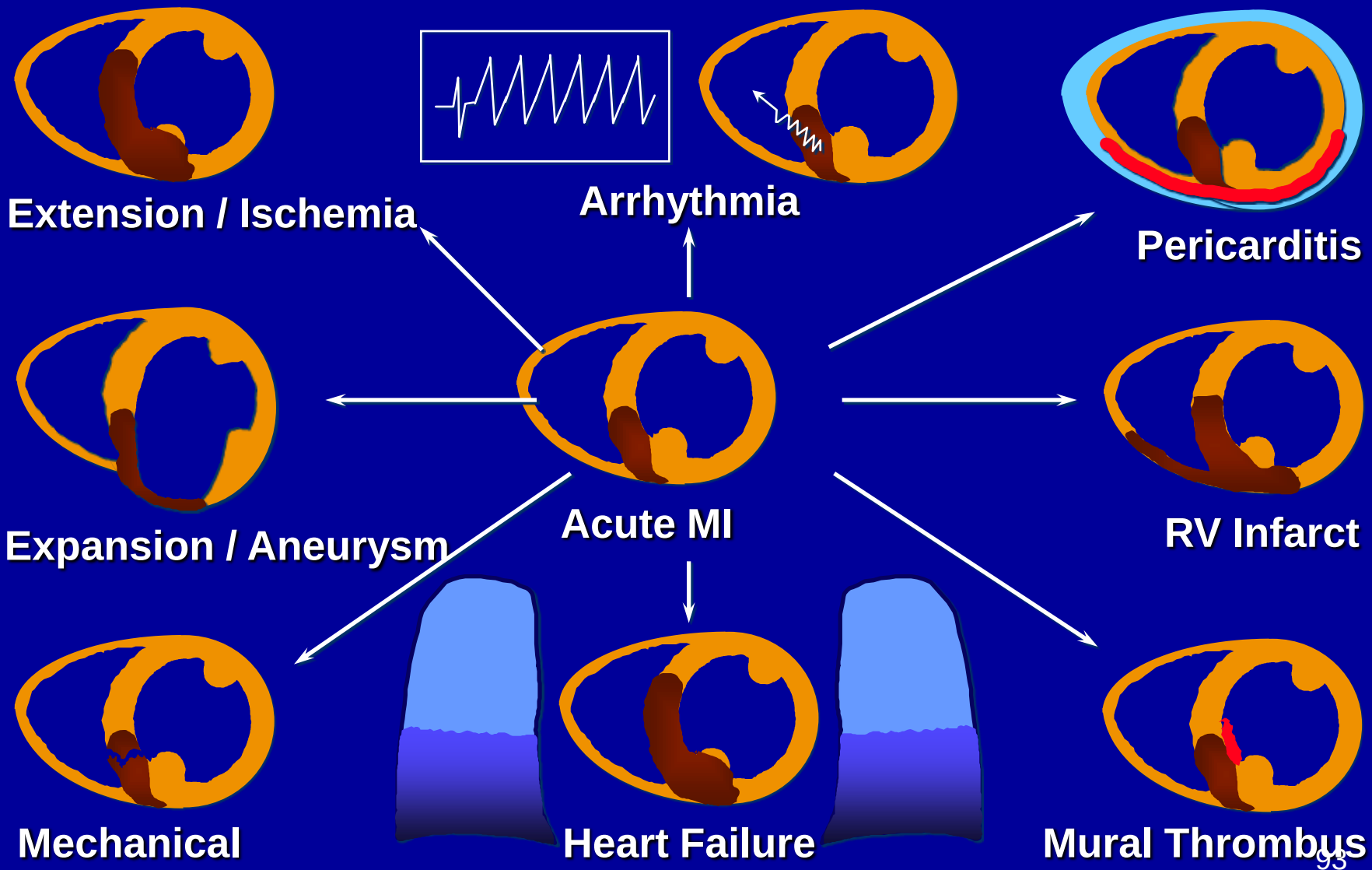


18TH to 20 th century
Balia district to max saket

Post MI Management

- ASA
- Nitrates
- β-blocker
- ACE- inhibitors
- Statins
- Aldosterone antagonists/ diuretics
- Anti-coagulation
  - large infarcts (especially anterior), CHF, LV thrombus, DVT, EF< 35 %, AF

Complications of MI



ARRHYTHMIAS

– Most common complication
– PVCs ~ 90%, SVT ~ 10%, CHB ~ 20% RV infarct
– Bradyarrhythmias – common with inferior MI
– Ventricular fibrillation (2-4%)
resuscitation
defibrillation
– Atrial fibrillation
may not require treatment
If hypotension occurs – DC cardioversion

Indications for Permanent Pacing

- persistent complete (third-degree) AV block
- persistent sinus node dysfunction - symptomatic bradycardia
- intermittent second-degree Mobitz II or third-degree AV block
- second-degree Mobitz II or third-degree AV block with new bundle branch block

Extension / Ischemia

Post infarct angina – 50%

Inc of CK-MB > 50% than previous nadir

Management same as unstable angina

In distribution of infarct vessel:

- IRA reperfusion, then reocclusion
- thrombus propogation, distal embolization

At a distance:

- reduced collateral flow from IRA
- new coronary thrombus
- reduced systemic perfusion pressure
- increased myocardial oxygen consumption

Treatment:

- Pharmacologic (beta blockers, nitrates)
- Urgent revascularization
- Repeat lytics (antibodies to SK)

Pericarditis

- Commonly occurs on 2nd -3rd day of infarction
- Results from infarction extending to epicardial surface of heart, with associated inflammatory response\

Post MI Syndrome (Dressler's)

- Fever
- Pericarditis
- Pleurisy
- Weeks to month after infarction
- High dose aspirin or corticosteroids

Right Ventricular Infarction

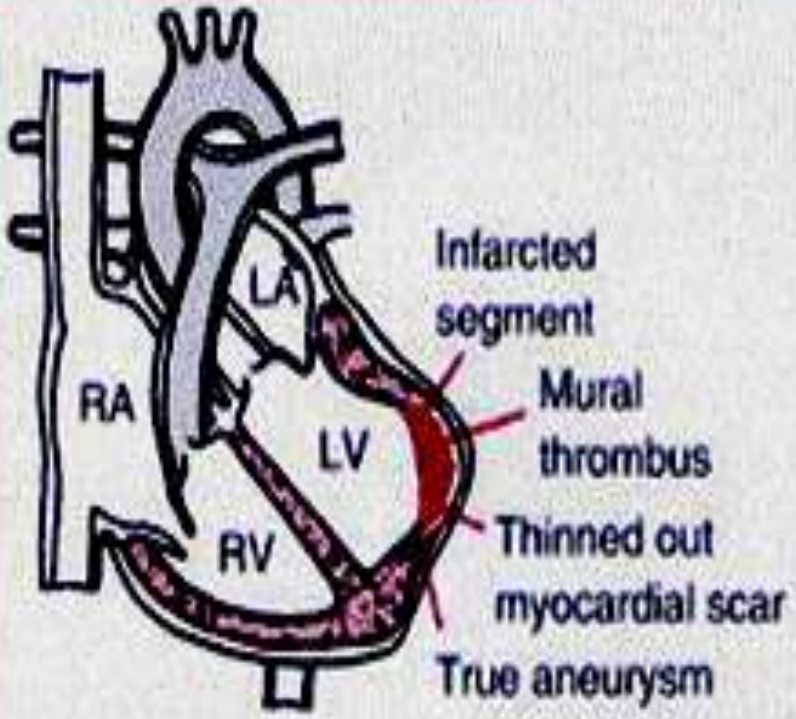
- Associated with occlusion of proximal RCA
- Classic triad by hypotension, ↑JVP, clear lungs
- ECG: ST↑ in RV4
- Echo: RV dilation and hypokinesia

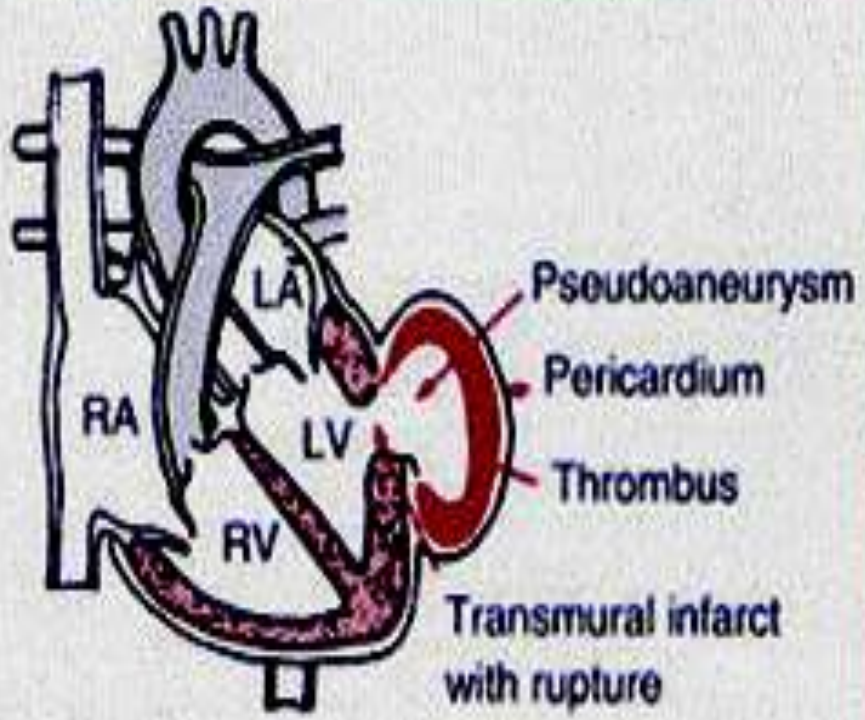
Management

- Usually transient ischemic dysfunction with long-term recovery common
- Marked sensitivity to preload reduction (nitrates)
- Fluid volume infusion for hypotension and low cardiac output

Infarct Expansion " Aneurysm "

- Circumscribed non-contractile outpouching of LV
- Usually composed of fibrous tissue + necrotic muscle +/- viable myocardium .
- Develops in 8 – 15% of patients post MI
- Common after AWMI with totally occluded poorly collateralized LAD
- Rarely seen with multivessel disease
- Potential consequences:
  - Mural thrombus +/- embolization
  - Adverse LV remodeling and CHF
  - ventricular rupture
  - ventricular arrhythmias



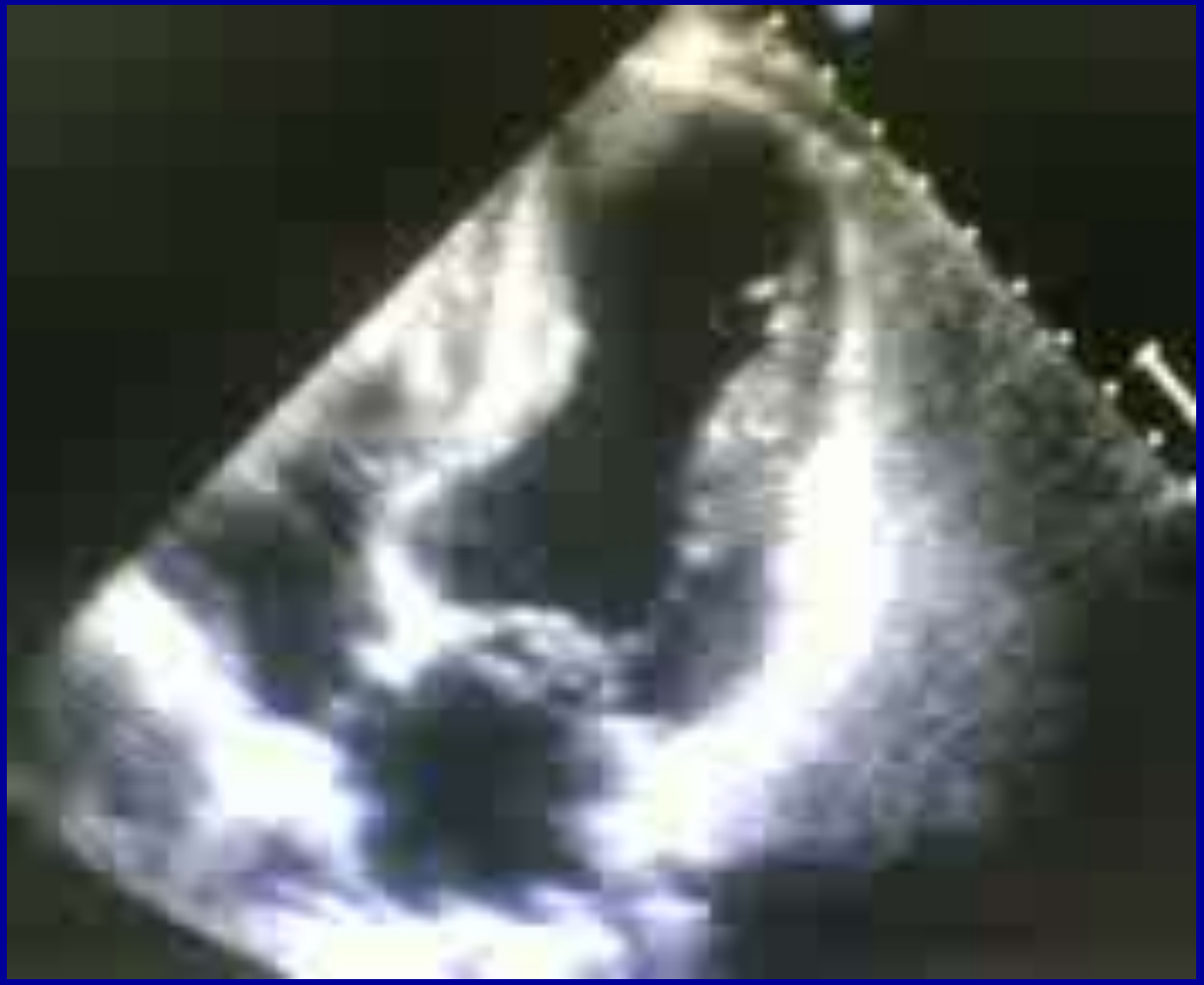


True aneurysm
1. Wide base
2. Walls composed of myocardium
3. Low risk of free rupture

Pseudoaneurysm
1. Narrow base
2. Walls composed of thrombus and pericardium
3. High risk of free rupture

Differences between a pseudoaneurysm and true aneurysm

Infarct Expansion “ Aneurysm ”



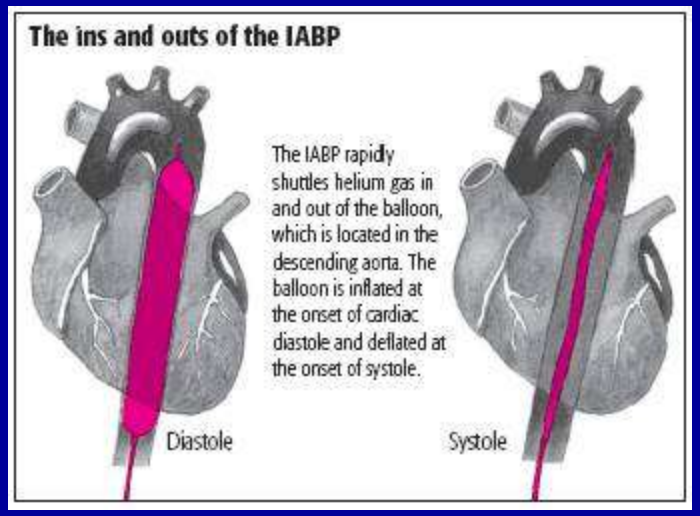
Heart (LV) failure & shock

- Cardiogenic shock:
  - 6 % of STEMI, 2 % of NSTE ACS
  - Mortality rate: 60%
  - LV wall > 40% infarcted
- Diastolic or systolic dysfunction may predominate

- Extensive LV infarctions
- Impaired relaxation, compliance
- Extensive RV infarction or ischemia
- VSD or acute severe MR
- Tamponade (with or without free wall rupture)
- Others - sepsis, beta- or Ca^{+2}-blocker overdose, pulmonary embolism

Heart (LV) failure & shock

- Correction of hypoxemia, acidosis, bradycardia, AV block, new onset AF
- Mechanical circulatory support + inotropic support
- Correction of hypoxemia, acidosis, bradycardia, AV block, new onset AF
- Mechanical circulatory support + inotropic support
- Intraaortic Balloon Counterpulsation
  - Extremely effective in supporting patients undergoing coronary angiography, PTCA, and CABG in cardiogenic shock.
  - provides bridging support until an LV assistance device can be implanted or cardiac transplantation can be performed



- LV and biventricular assistance devices
- Percutaneous cardiopulmonary bypass support

LV thrombus and emboli

- Incidence of clinically evident systemic embolism after MI < 2%.
- Increases in patients with AWMI
- Incidence of mural thrombus after MI - 20%
- Systemic embolism -10% of LV thrombus
- Risk is highest in the first 10 days but persists at least 3 months

Mechanical complications

Ventricular septal rupture

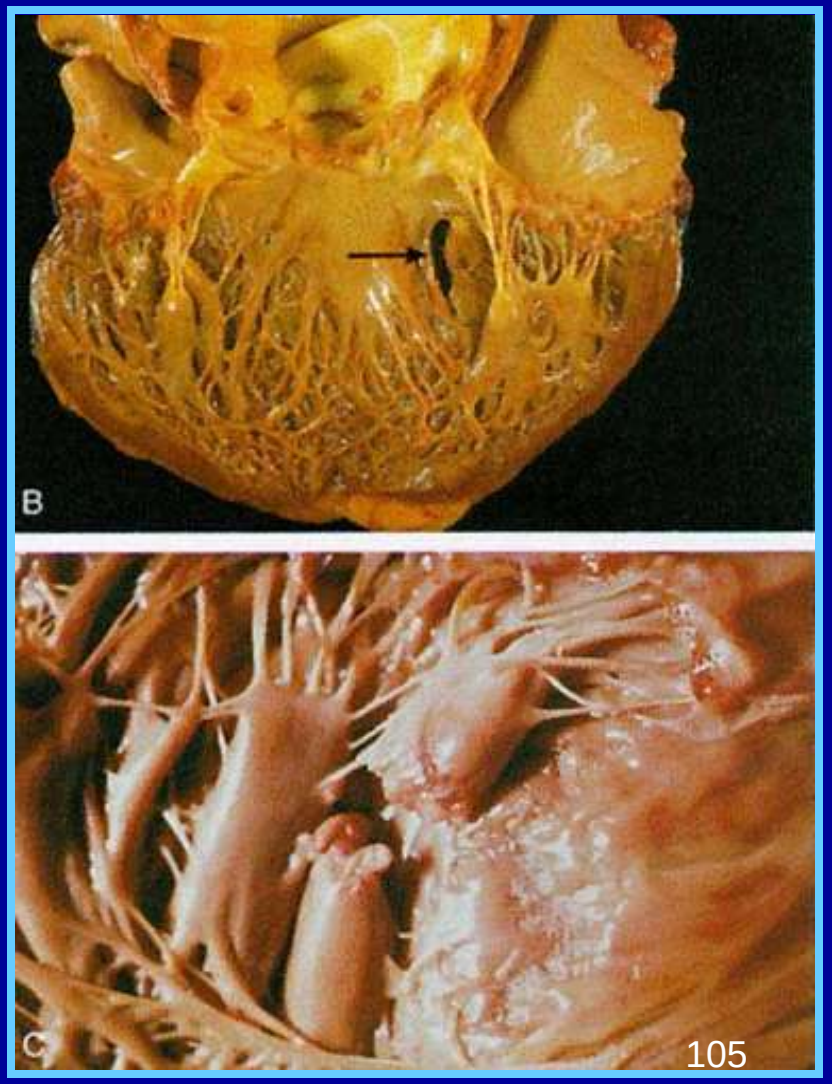
2-5 days post MI

Free wall rupture

within 2 weeks post MI

Ischemic MR

13 hours post MI



Ventricular Septal Rupture

- Incidence - 1-3% of transmural Mis
- Medical stabilization and IABP
- Early surgical repair for decompensated pts
- Small asymptomatic VSDs may not require repair
- Sudden appearance of loud systolic murmur and thrill medial to the apex along the left sternal border in the 3rd or 4th intercostal space, accompanied by hypotension with or without signs of LV failure, is characteristic

Acute Mitral Regurgitation

- Transient MR common in early MI (20-40%)
- Persistent MR, even mild, associated with increased long-term mortality post-MI
- Due to papillary muscle or chordal rupture or dilation of ventricle and annulus
- Most common with inferior MI
- Early transient late apical systolic murmur thought to represent papillary muscle ischemia
- Sudden hemodynamic deterioration common
- Stabilize medically, IABP, then surgical repair

Free Wall Rupture

- Less frequent (1-3.4%), but earlier with STK
- Uncontained $\rightarrow$ sudden death or asystole
- Pseudoaneurysm $\rightarrow$ transient hypotension, bradycardia, repetitive emesis, restlessness
- Echocardiogram usually diagnostic
- Surgical repair - may require pericardiocentesis for uncontained rupture

Post MI Complications
"ACT RAPID"

- **A**rrhythmias
- **C**ongestive Heart Failure
- **T**amponade / **T**hromboembolic disorder

- **R**upture (Ventricle, septum, papillary muscle)
- **A**neurysm (Ventricle)
- **P**ericarditis
- **I**nfection
- **D**eath / **D**ressler's Syndrome

PREVENTION

PRIMORDIAL

- Preventing spread of CHD risk factors and life styles that have not yet appeared or become endemic by Mass education

PRIMARY

- Aims at reversing risk factors that have established themselves
- Population strategy, High risk strategy
- It includes :- TLS modifications

2˚MI Prevention Goals

- Control modifiable CAD risk factors
- Prevent development of systolic HF
- Prevent recurrent MI, stroke
- Prevent death, including SCD

2˚MI Prevention Drugs

- ASA
- β-blocker
- ACE- inhibitors
- Statins

Exercise

- Walking, jogging or bike riding
- Intensity – 70 – 85% of Heart Rate max
- Duration – 30 minutes with warm up 5 minutes and cool down 5 minutes
- Frequency - 5 times a week

Dietary goals

-Fat limited to 20-30% of total daily intake

-Saturated fats upto 10%

-PUFA upto 10%

-Refined carbohydrate

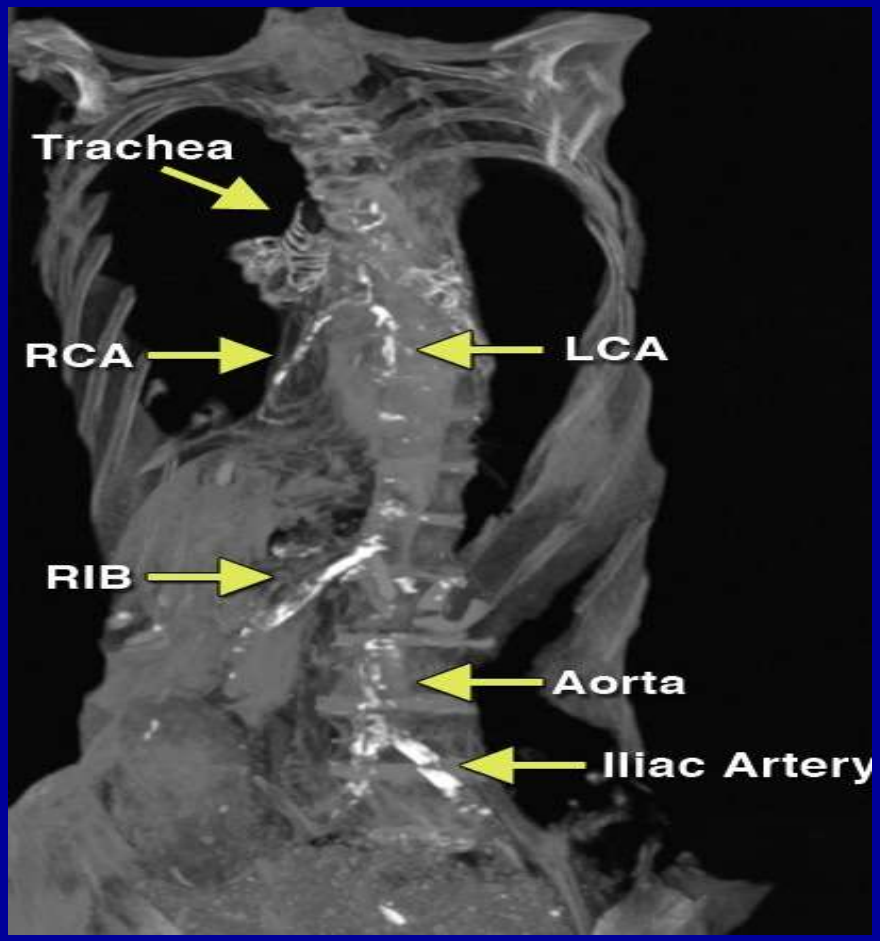
-Protien upto15%

-Fibre upto 20-30mg%

-Cholesterol <200mg%

17th dynasty princess, “Ahmose-Meryet-Amon" 1550-1580 BCE

Horus Study of Ancient Egyptian Mummies



♂ 33M 40±10.2 y ♀17F 37.6±12 y

Thank you

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