ISCHEMIC HEART DISEASE

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A 60 YEAR OLD MALE,SMOKER, DM PRESENTED C/O CHEST PAIN OF 6 MONTHS DURATTION

Retrosternal

Heavy

PPT by exertion → excerbated

Relieved by rest

Last about 5 minutes

DIAGNOSIS: stable angina

Ischemic Heart Disease (IHD)

chronic coronary syndrome

acute coronary syndrome

Basic: coronary circulation

Myocardial oxygen supply

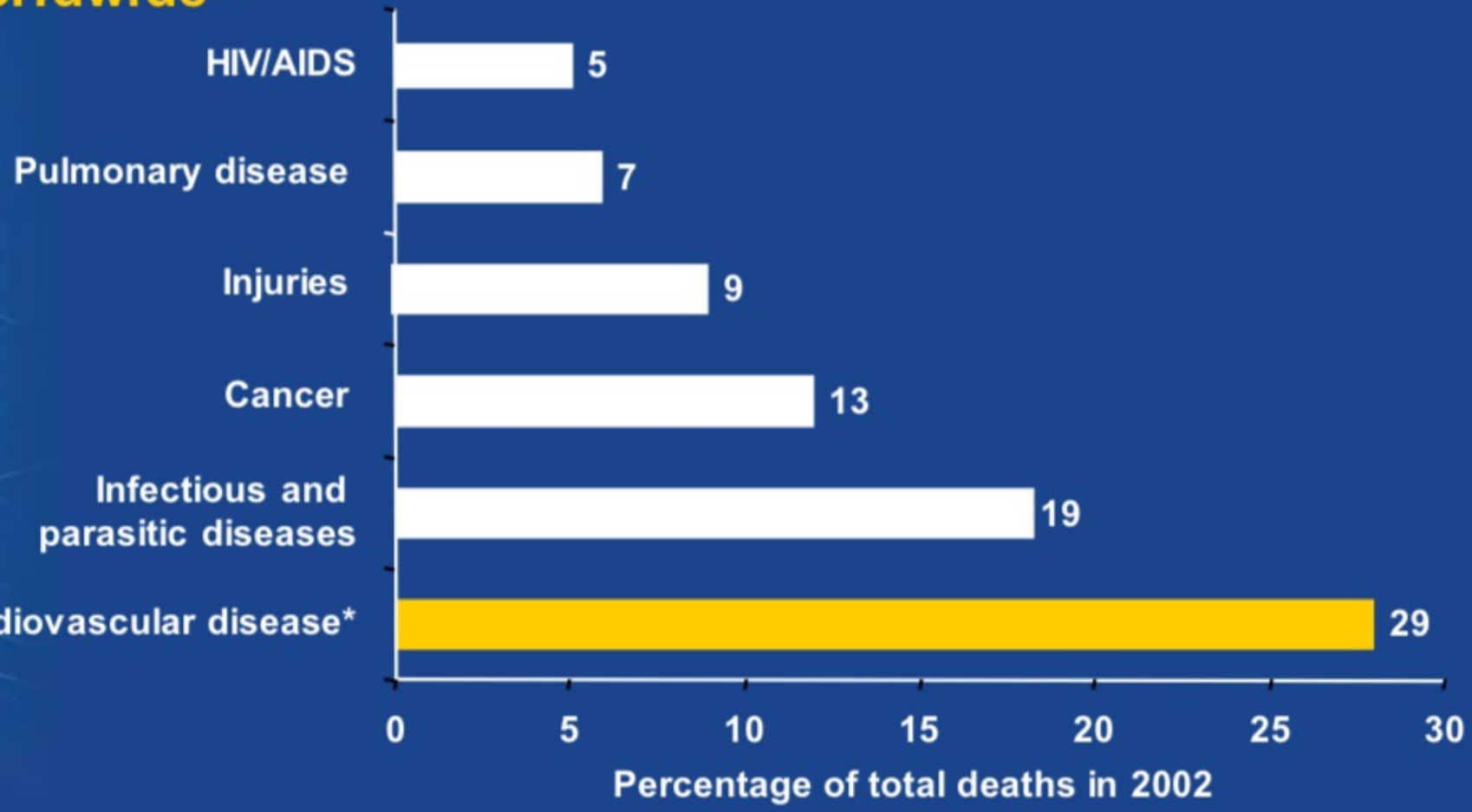
What is IHD

Causes of IHD

Manifestations of IHD

Treatment

Cardiovascular Disease is the Leading Cause of Death Worldwide[1]



*Ischemic heart disease, cerebrovascular disease, hypertensive heart disease, inflammatory heart disease and rheumatic heart disease

1. The World Health Report 2004. WHO Geneva, 2004.
Available at: http://www.who.int/whr/2004/en/. Accessed January 2006.

Heart Anatomy

In the middle mediastinum

- The heart is about the size of a fist and weighs 300-450 gm
- The average beat per minute is 70
- The average adult heart pumps about 6000-7500 liters of blood per day.

↳ Reciprocal changes: ST depressions on the other leads than the MI leads

↳ Tombstone ST elevations are very severe and high mortality

↳ ST elevation → convex ∩ → classic for MI
↳ concave ∪ → classic for pericarditis

Coronary Anatomy

↳1st branch of aorta

there are some collaterals (not functioning)
to function need → transient ischemia

↳Right heart dominance
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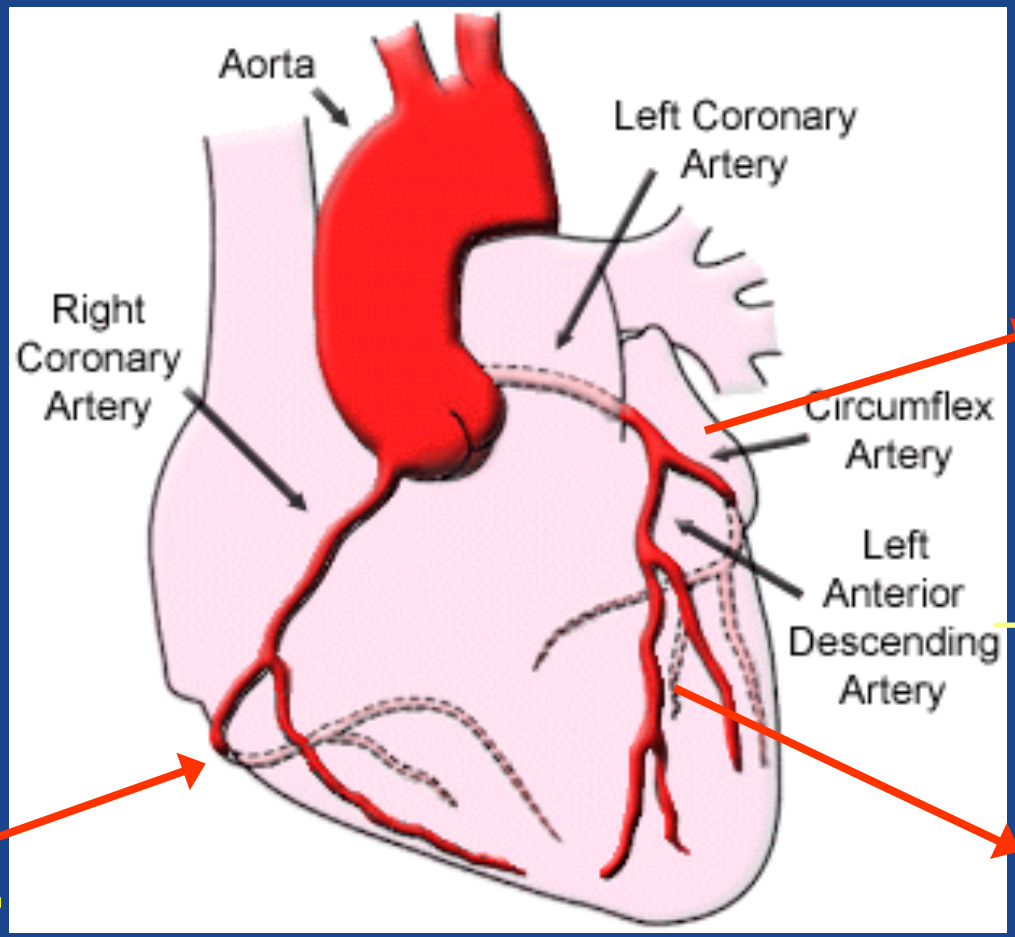
right باد



Lateral
I, AVL, V5-V6 LCX

LAD

Anterior / Septal
V1-V4 LAD

Inferior
II, III, aVF
PDA

Coronary Circulation physiology

1- Flow during basal (at rest) cardiac circulation: 70-80 ml/min/100gm

2- Flow during maximal cardiac work: 300-400ml/min/100gm

3- High oxygen extraction: 65%-75% (fixed)

4- 80% of coronary flow occurs in diastole

need ↑ O_2
need ↑ coronary flow
↳ if not → ischemia

5- Collateral pathways is anatomically present but not functioning

6- Cardiac tissue hypoxia is the potent stimulus to open the collateral

Ischemic Heart Disease

Myocardial O_2 **demand**

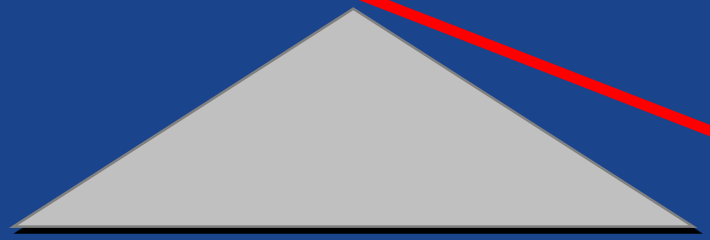
→ to avoid tachycardia : give β-blocker if no contraindication like asthma

1- Heart rate

2- ↑Contractility → ↑ O_2 demand

3- Wall tension

4- Muscle mass (wall thickness)



Myocardial O_2 **supply**

1- Coronary flow (patency of coronary artery) ↳healthy or not

2- Hemoglobuline level

Fixed ← 3- Myocardial oxygen extraction

Lung disease ← 4- Arterial oxygen saturation

Physiology of coronary circulation

Myocardial ischemia: imbalance between oxygen supply and demand

***Myocardial oxygen demand*:**

1- Heart rate

2- Contractility

3- Wall tension

4- Muscle mass (wall thickness)

Myocardial Oxygen supply:

1- Coronary flow (patency of coronary artery)

2- Hemoglobuline level

3- Myocardial oxygen extraction

4- Arterial oxygen saturation

CAUSES OF Myocardial ischemia

Reduced Myocardial O2 Supply

1-Coronary artery disease
(atherosclerosis and non-atherosclerosis)

2-decrease flow of oxygenated blood:

- Sever Anemia
- carboxyhemoglobulinemia
- Hypotension

Increased Myocardial O2 Demand

1-Left Ventricular Hypertrophy:

cause angina due to ↑ in myocardial O_2 demand

- hypertension
- aortic stenosis
- hypertrophic cardiomyopathy

2- Increase cardiac output:

- Thyrotoxicosis
- Rapid Tachyarrhythmias

Causes of coronary artery disease

Atherosclerosis 95%	*Nonatherosclerosis* 5%
Risk factors	*1-Arteritis (SLE,RA,Takayasu ,..)* like: vasculitis *2-Embolism* *3-Coronary mural thickening (amyloidosis,radiation therapy,..)* *4-Coronary luminal narrowing: coronary spasm, aortic dissection* *5-Congenital coronary artery anomalies*

Risk Factors for Cardiovascular Disease

Modifiable

4 major risk factors

- Hypertension
- Smoking → passive & active
- Hyperlipidaemia
  - Raised LDL-C
  - Low HDL-C
  - Raised triglycerides
- Diabetes mellitus
- Dietary factors
- Lack of exercise
- Obesity → truncal
- Homocysteinemia
- Lipoprotein a
- Gout
- Thrombogenic factors: fibrinogen, factors V, VII
- Excess alcohol consumption

Non-modifiable

- Personal history of CVD
- Family history → 1st degree of CVD
  M < 55 F < 65
- Age: M>45, F>55
- Gender M>F (Premenopausal)
- Personality type A
- Genetic factors: ACE gene

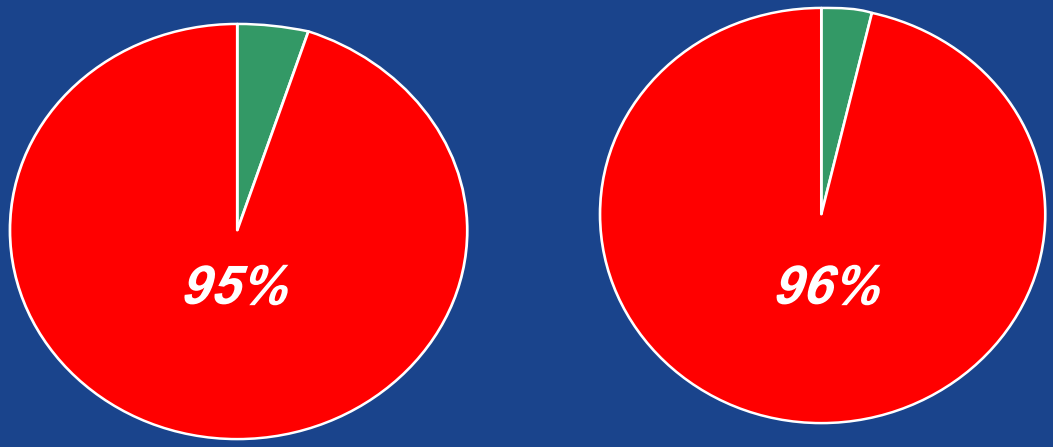
Prevalence of Modifiable Conventional Risk Factors in Patients With CAD in Jordan

At least one RF
Diabetes
HTN
Current smoking
C>240
TG>150
HDL<40

1534

Men
1202 (78%)

Women
332 (22%)

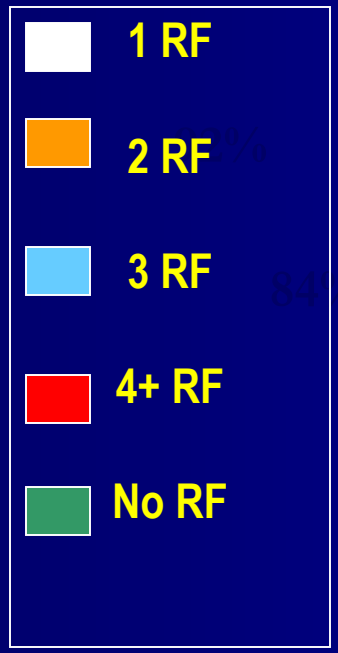


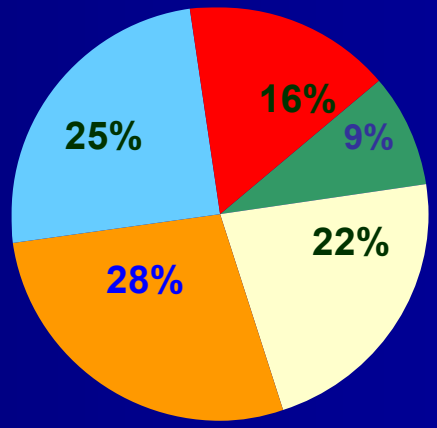
95% of Patients With CAD in Jordan Have at least one of the Modifiable Conventional Risk Factors

Number of conventional risk factors in individuals with CAD in Jordan

Men with CAD

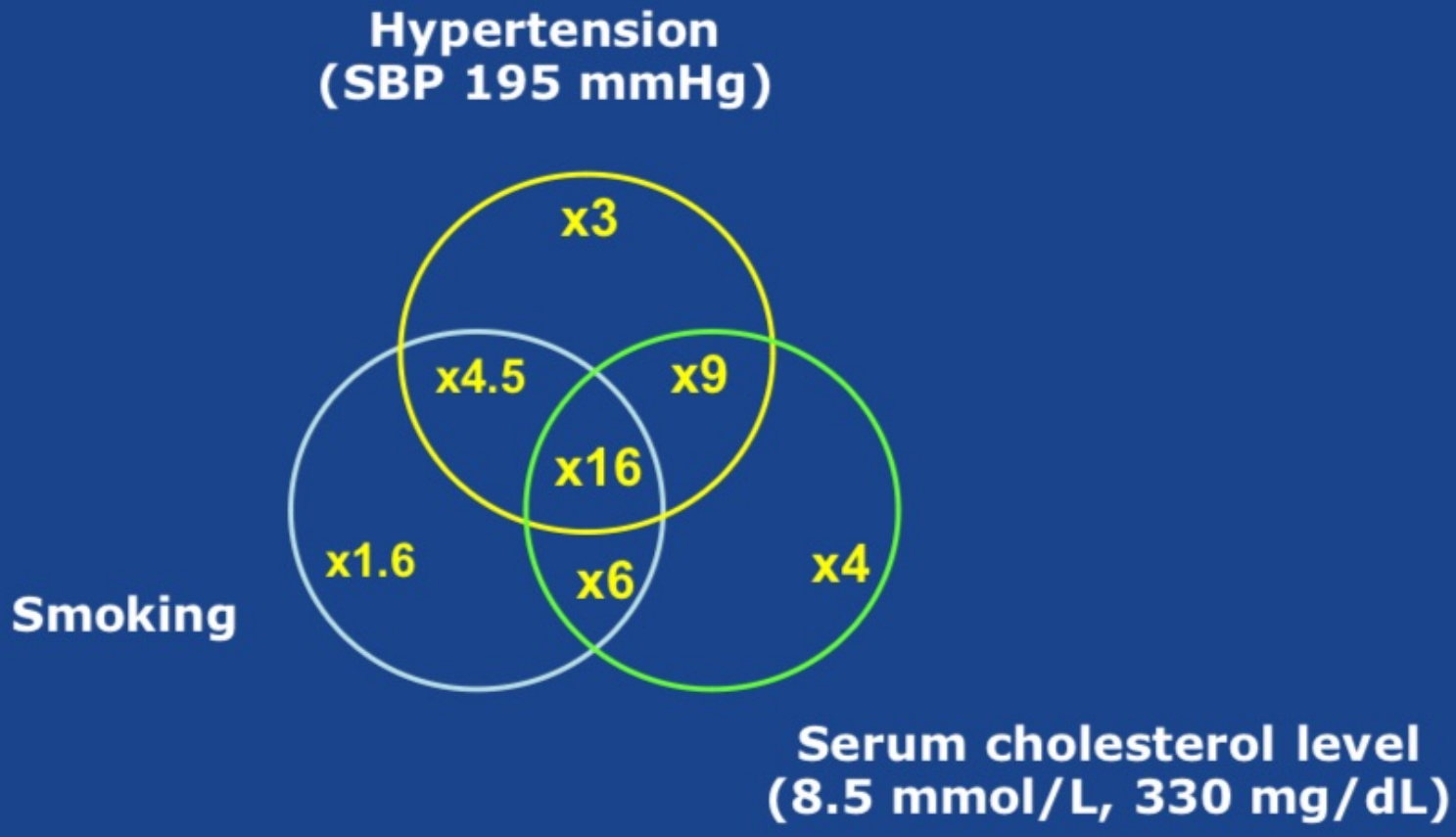
Diabetes
HTN
Smoking
C>240
TG>150
HDL<40





Hammoudeh et al. [JoHARTS 2]
European Heart Journal, September 2005
International Journal of Cardiology, July 2006

Levels of Risk Associated with Smoking, Hypertension and Hypercholesterolaemia. Exponential effect



Pathogenesis of Atherosclerotic Plaques

(mechanical shear stresses, biochemical, immunological, inflammation, genetics abnormalities)

Endothelial damage (Dysfunction)

↓

Protective response results in production of cellular adhesion molecules
(Cytokines, Chemokines, Growth factors)

↓

Monocytes and T lymphocytes attach to 'sticky' surface of endothelial cells

↓

Migrate through arterial wall to subendothelial space

↓

Macrophages take up oxidised LDL-C

↓

Lipid-rich foam cells

↓

Fatty streak and plaque

The 'Activated' Endothelium

'activated' endothelium

- ↑ cytokines (eg. IL-1, TNF-α)
- ↑ chemokines (eg.MCP-1, IL-8)
- ↑ growth factors (eg. PDGF, FGF)

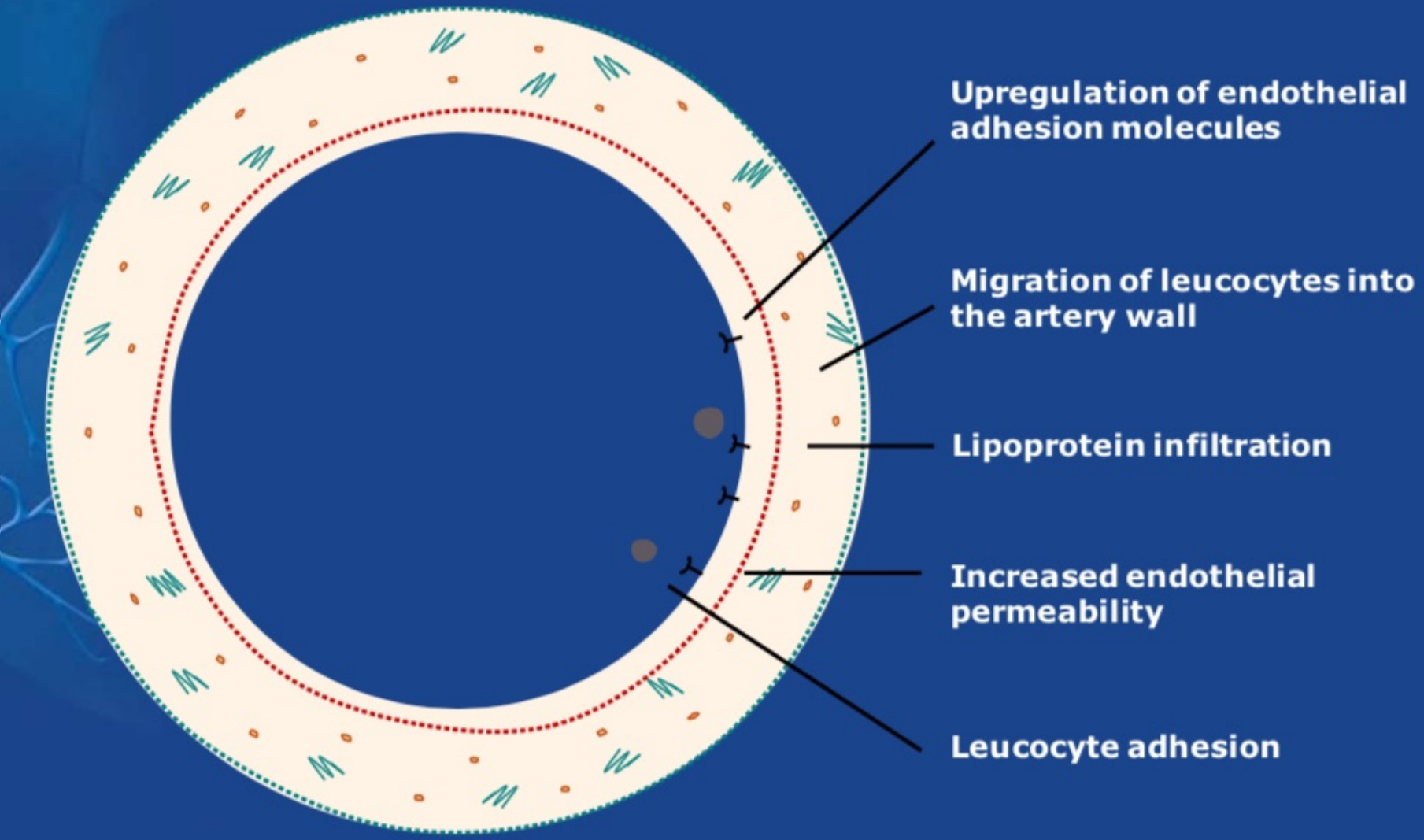
} **CELLULAR ADHESION MOLECULES**

↓ attracts monocytes and T lymphocytes which adhere to endothelial cells

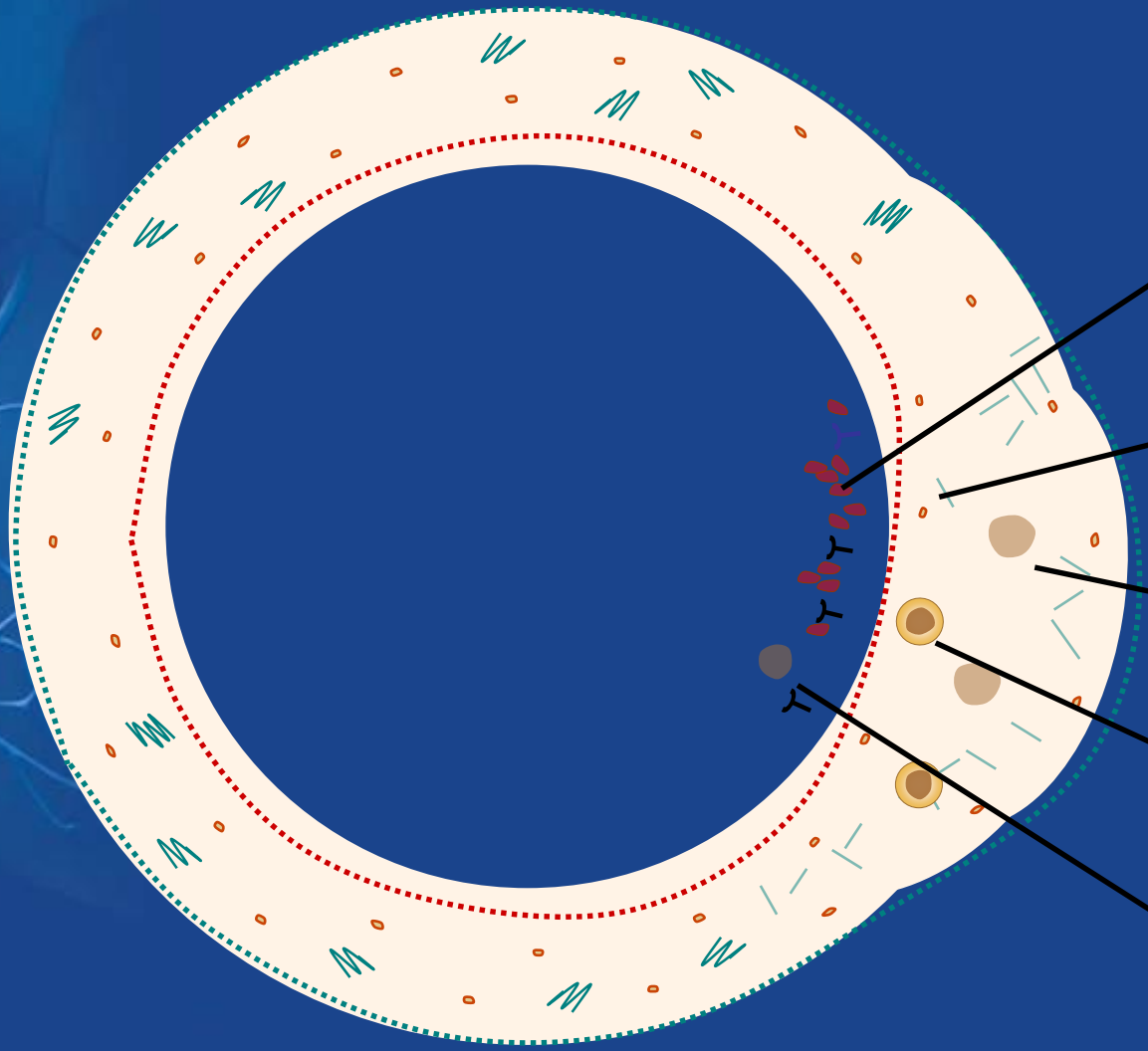
↓ induces cell proliferation and a prothrombic state

Koenig W. *Eur Heart J Suppl* 1999;**1**(Suppl. T):T19–26

Endothelial Dysfunction in Atherosclerosis



Fatty Streak Formation in Atherosclerosis



Adherence and aggregation of platelets

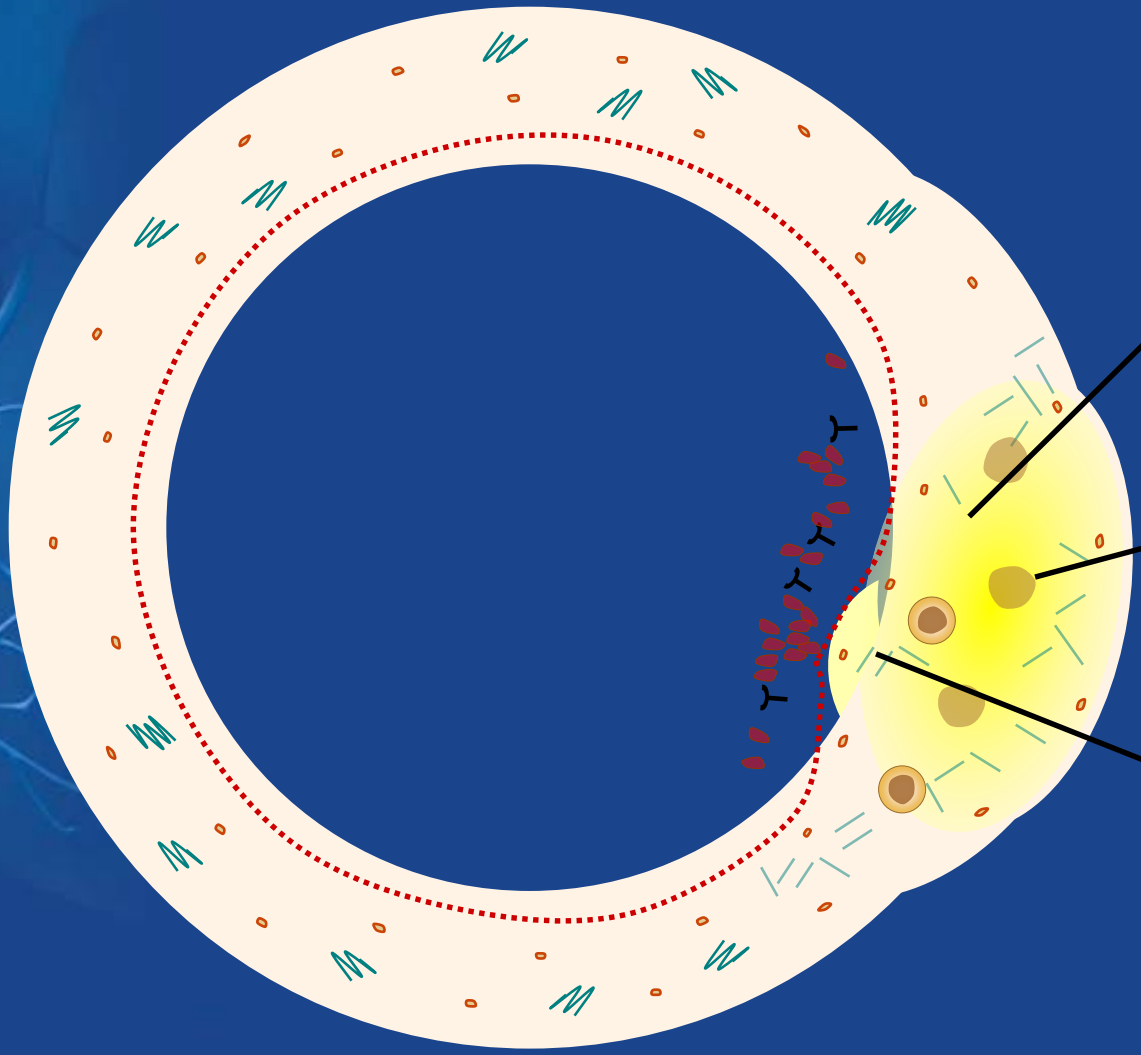
Migration of smooth muscle cells

Formation of foam cells

Activation of T cells

Adherence and entry of leucocytes

Formation of the Complicated Atherosclerotic Plaque

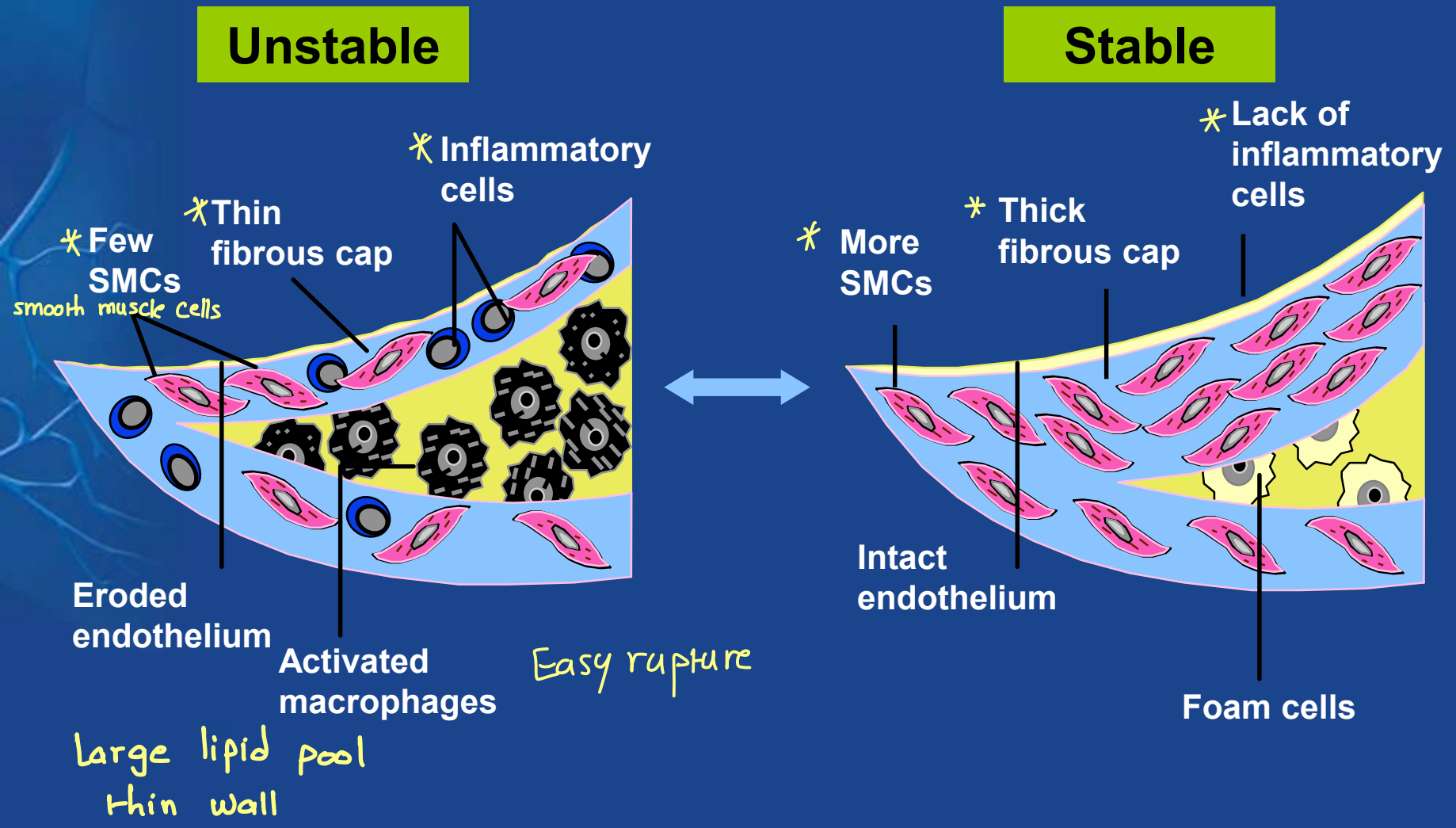


Formation of necrotic core

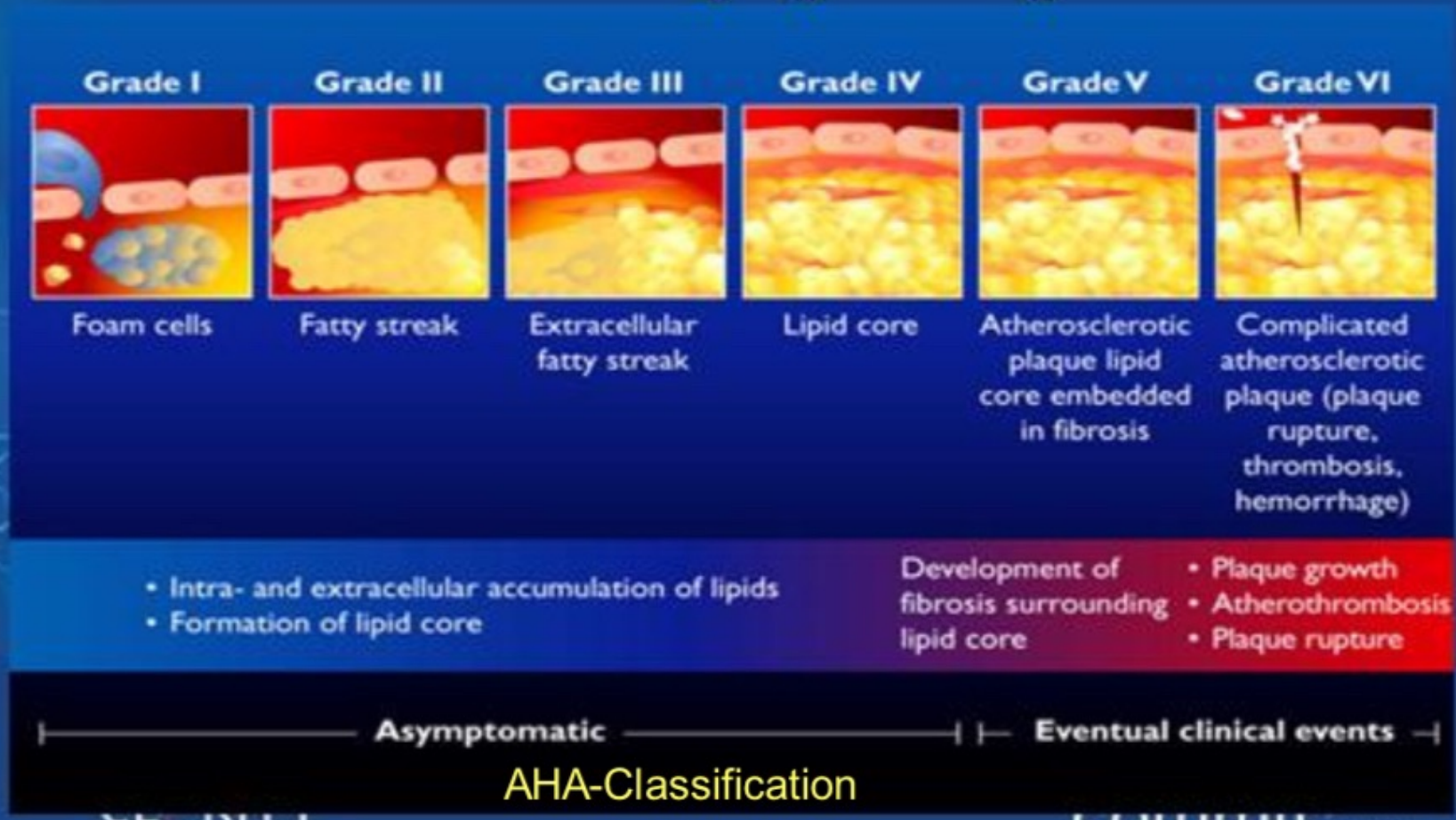
Accumulation of macrophages

Formation of the fibrous cap

Characteristics of Unstable and Stable Plaque

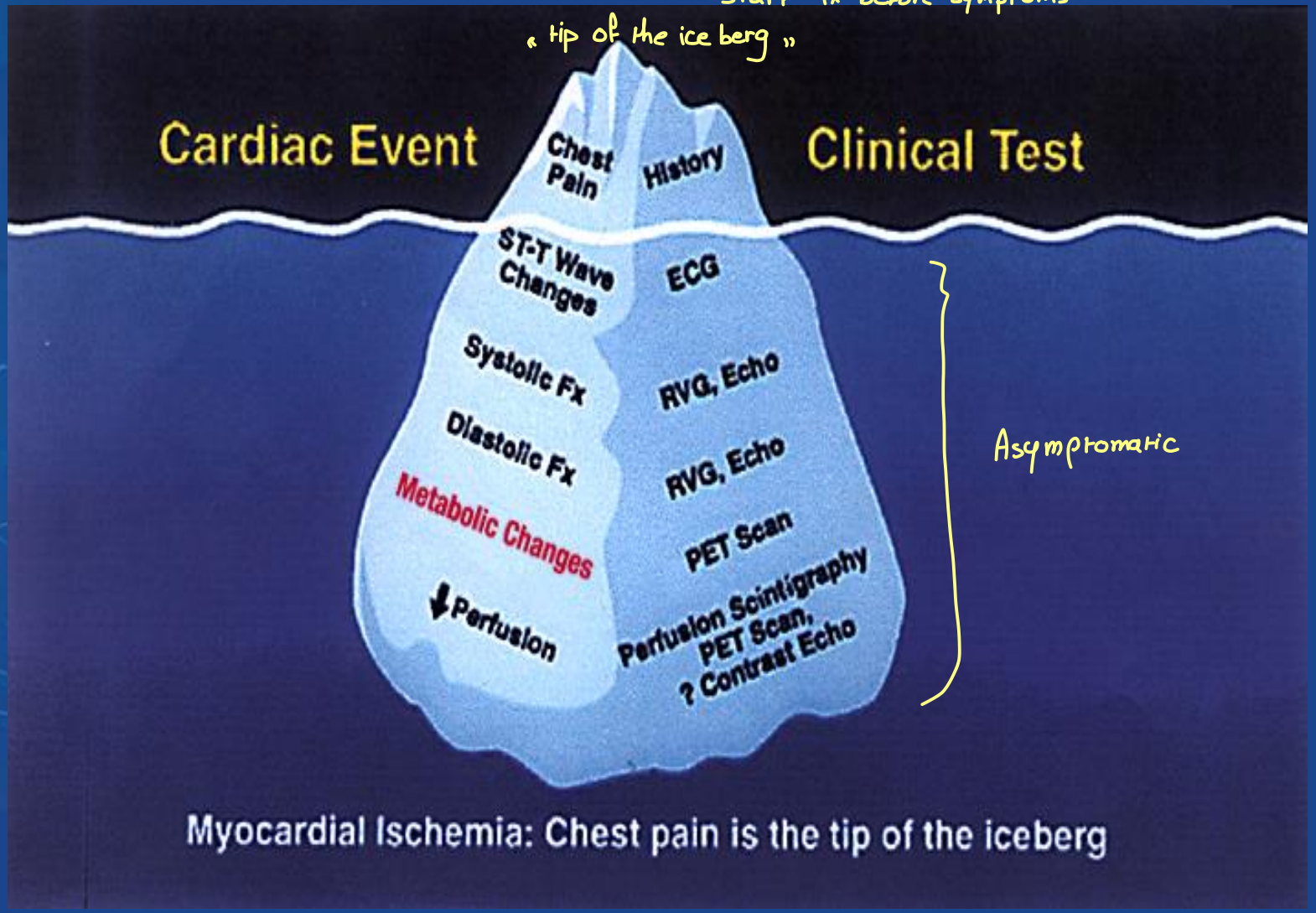


Cardiovascular risk factors and the stages of atherosclerotic plaque development



AHA-Classification

- too late presentation
- start Tx before symptoms

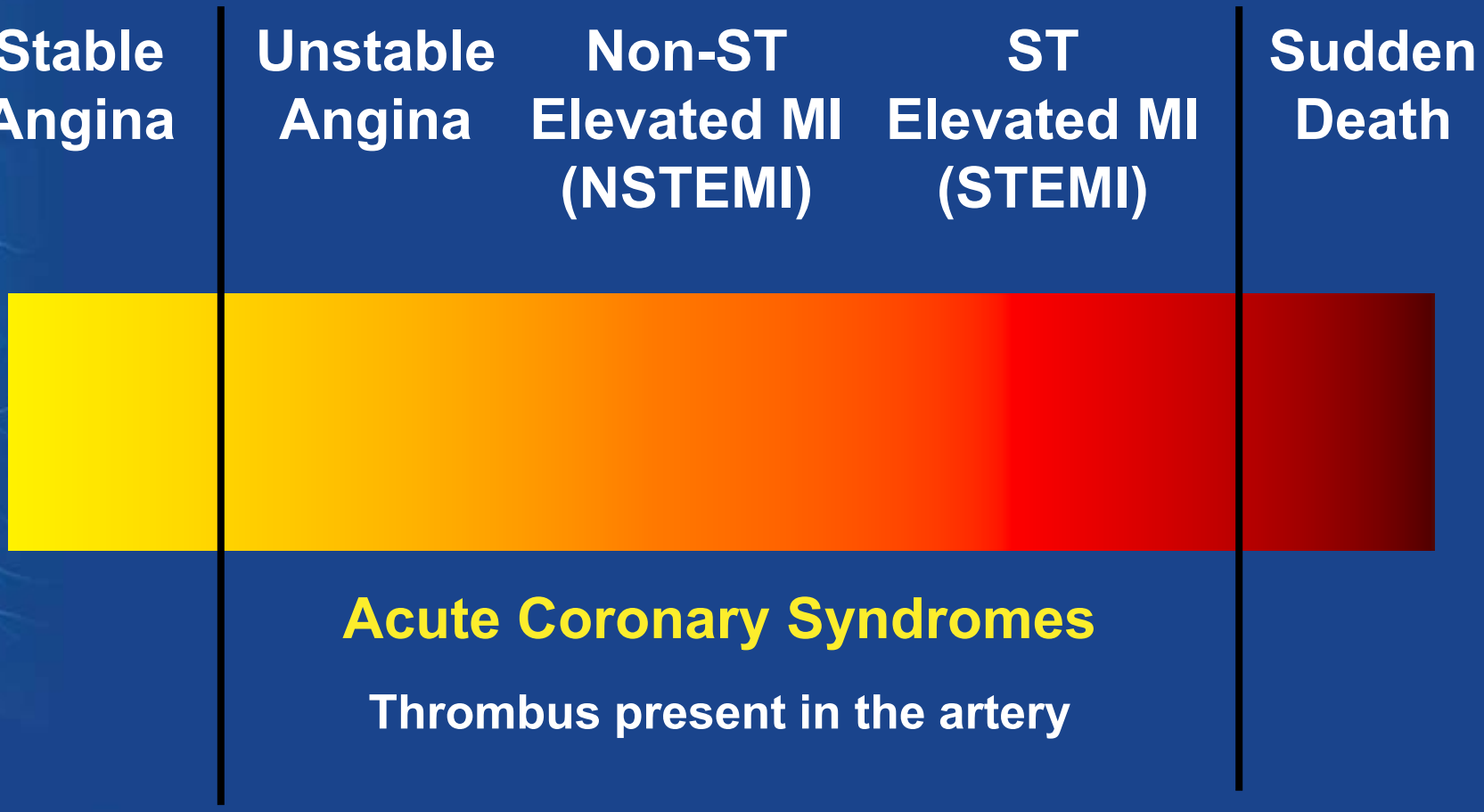


Major Manifestations of Atherothrombosis

- Cerebrovascular disease
- Coronary artery disease
- Renal artery stenosis
- Visceral arterial disease
- Peripheral arterial disease (PAD)

The Spectrum of presentations

Myocardial Ischemia



Adapted from Cannon CP. *Contemporary Diagnosis and Management of Acute Coronary Syndromes.* 2nd ed. Newtown, PA: Handbooks in Health Care Co.; 2008.

Clinical Manifestations of Atherosclerosis

Coronary heart disease

- *Asymptomatic,* **Angina pectoris, variant angina, unstable angina, myocardial infarction,** *congestive heart failure (CHF), arrhythmias, and sudden cardiac death.*

Cerebrovascular disease

- *Transient ischaemic attack, stroke*

Peripheral vascular disease

- *Intermittent claudication, gangrene, cold feet, painful feet, impotence*

IHD-clinicopathological correlation

1- stable angina: stenosis > 70% luminal narrowing

2-variant angina: increase coronay tone
30% normal coronaries

3-unstable angina: rupture plaque
subocclusive thrombus (incomplete occlusion)
progress to myocardial infarction 15-30%

4-myocardial infarction: rupture plaque
occlusive thrombus (complete occlusion)

Stable angina

Commonest form of angina

Causes: imbalance between demand and supply

Symptom: chest pain

Location: central chest (others)

Radiation: arm(s), neck, jaw

Character : squeezing, pressure, heaviness,..

Duration: 2-10 minutes

Precipitating factors: exertion, emotional upset, heavy meal, sexual intercourse, cold weather

Relieving factors: nitrate, rest

Associated symptoms: dyspnea, diaphoresis, nausea

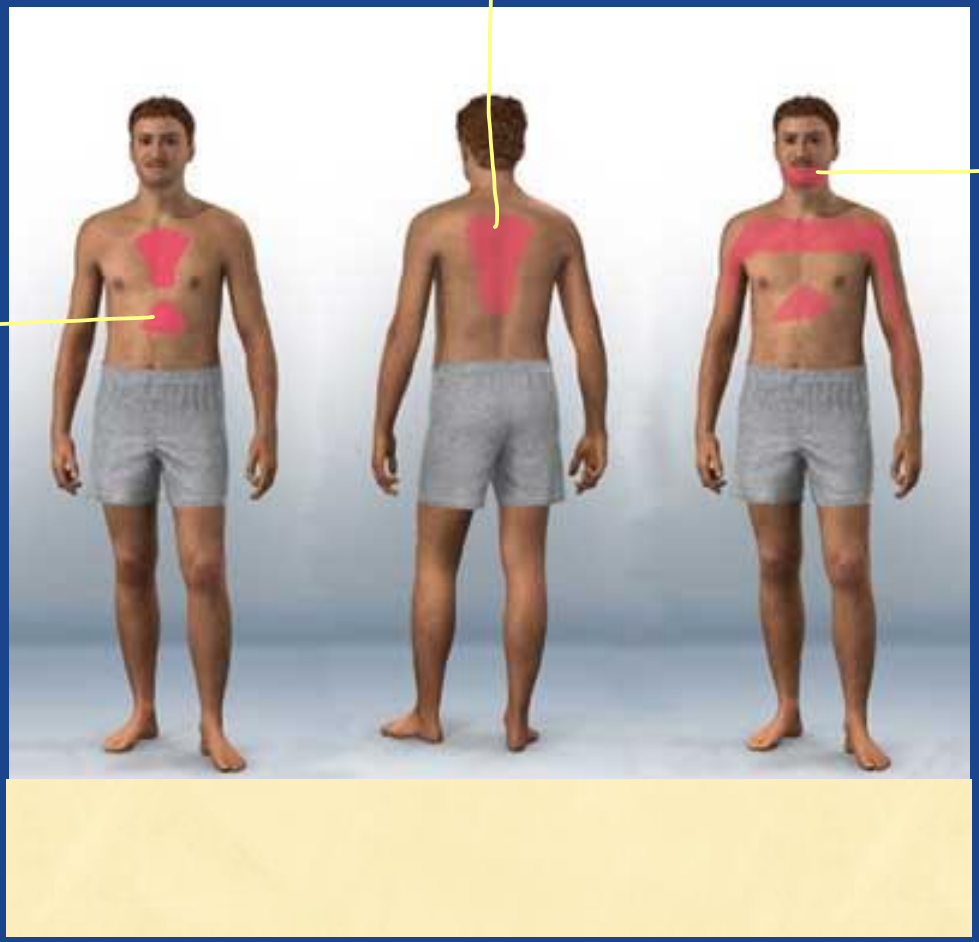
Classes of angina: 1- 4

Physical Examination: normal, sign of risk factors, peripheral vascular disease

Symptoms of Angina

Angina can spread anywhere between the belly button and the jaw, including to the shoulder, arm, elbow or hand- usually on the left side.

typically : chest pain



بس أمشي ظهري بوجعني وبس أرتاح بروح

Angina → clinical diagnosis

ECG may be normal

Angina Chest Pain:

Clinical Diagnosis

CAUSES OF ANGINA

Reduced Myocardial O2 Supply

1-Coronary artery disease

2-Sever Anemia < 9 gm/dl

Increased Myocardial O2 Demand

1-Left Ventricular Hypertrophy:

- hypertension
- aortic stenosis
- hypertrophic cardiomyopathy

2- Rapid Tachyarrhythmias

NYHA Grading of cardiac symptoms (angina / dyspnea)

same classes of dyspneq

Grade 1:

Cardiac disease without resulting limitation of physical activity. Ordinary physical activity does not cause chest pain (dyspnea).

>ordinary

Grade 2:

Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity result in chest pain (dyspea).

Ordinary

Grade 3:

moderate limitation in physical activity. Comfortable at rest. Less than ordinary activity causes symptoms

<ordinary

Grade 4:

sever limitation: symptoms at rest. unstable

Rest

normal ECG will not rule out any cardiac disease

↳ no specific signs, normal exam

Stable angina- Diagnosis

↳ auscultate coronary for bruits

- History : angina pectoris is clinical diagnosis
- Physical exam normal
- Electrocardiogram: 12 ECG, 24 ECG
- Stress ECG : diagnostic and prognostic information ← 60-70% specific
- Radioactive studies: thalium scan,..
- Echocardiography
- CT Coronary angiography
- Serum lipid(LDL, HDL, TG), FBG,CBC → anemia
- Coronary angiography

Types of stress test

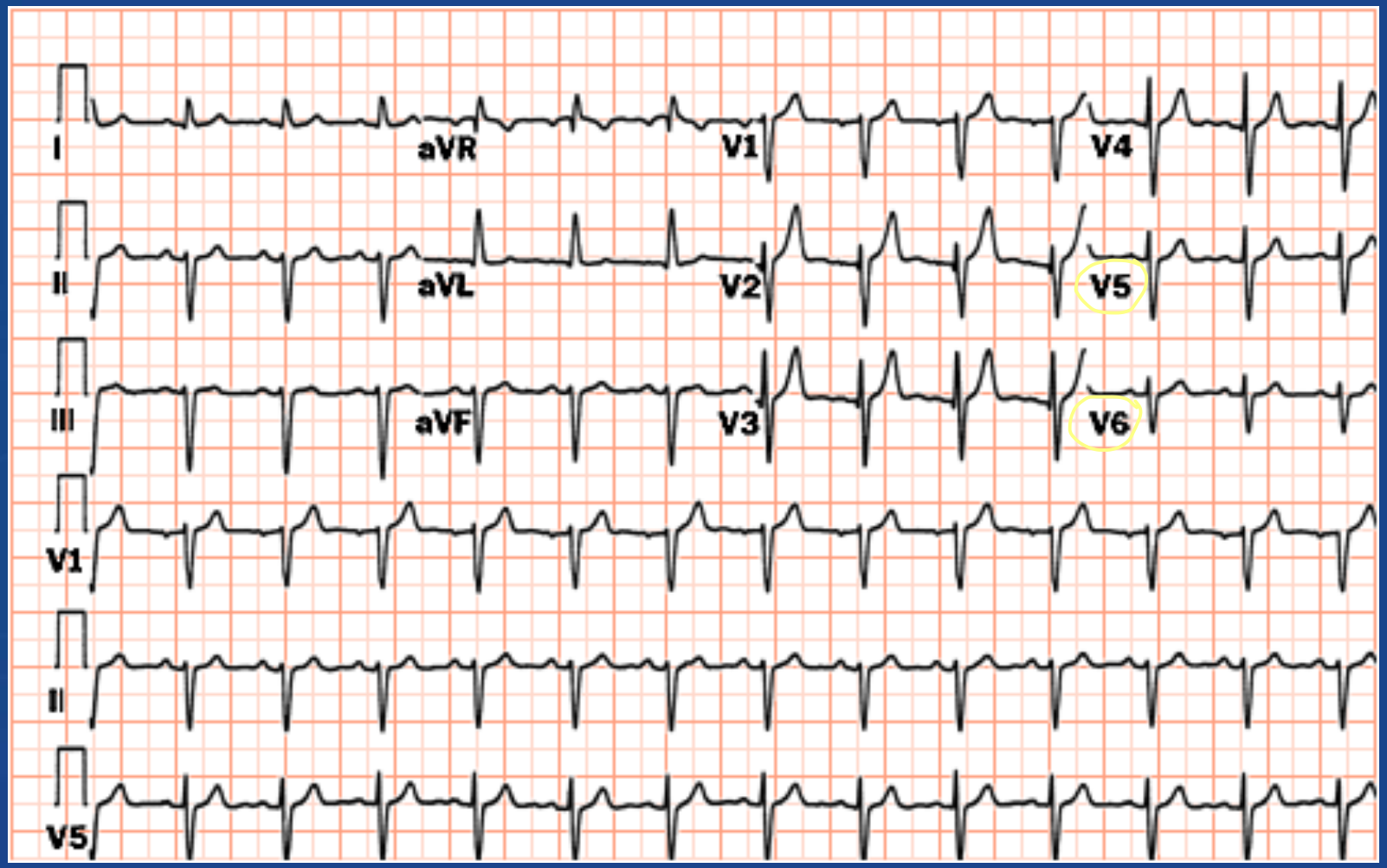
- *Exercise tolerance test: ST segment depression*
- *Exercise or dobutamine Echocardiogram: Wall motion abnormalities*
- *Exercise or dipyridamole Thallium: Decrease uptake of the nuclear isotope during exercise*

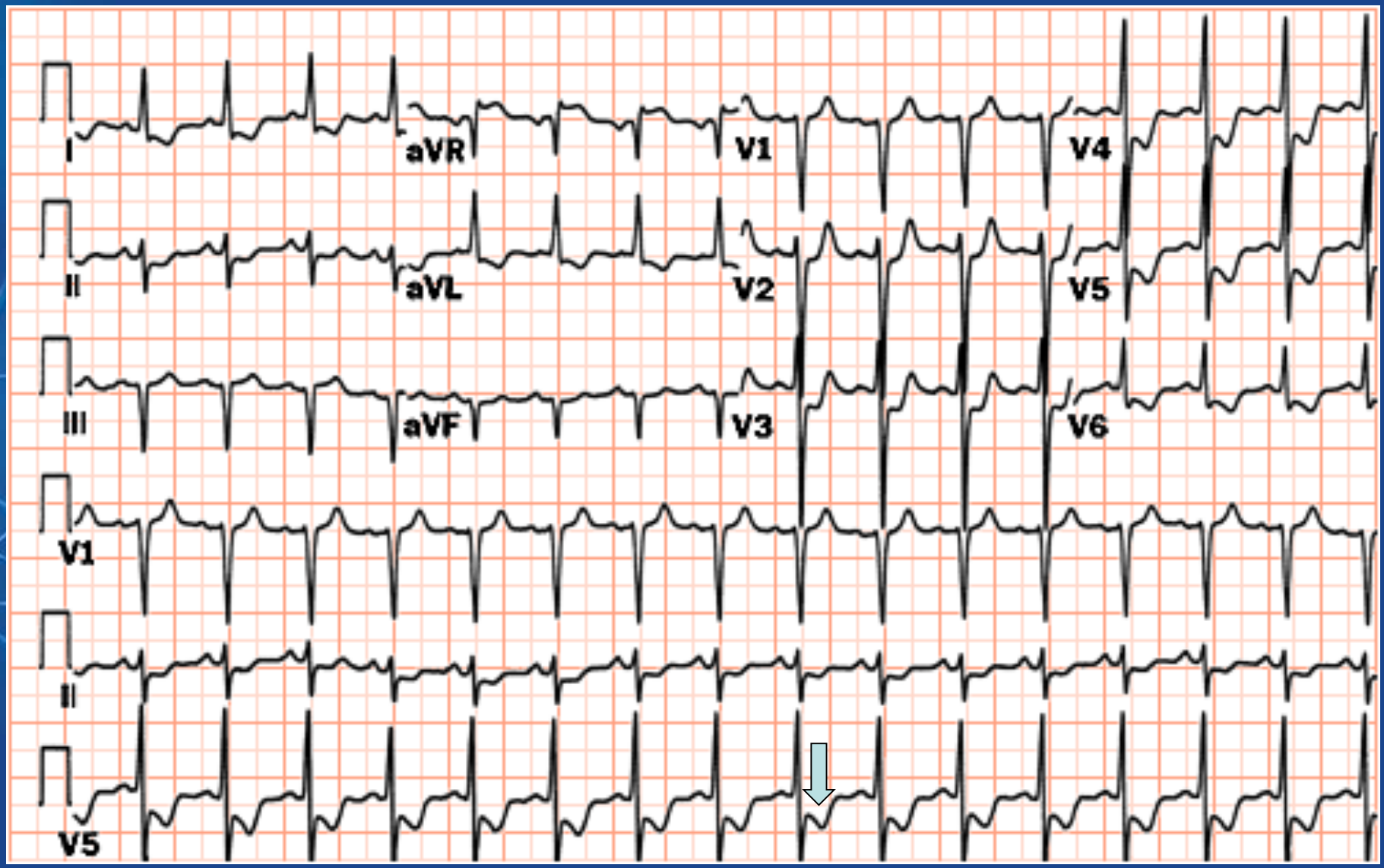
Bruce Protocol for Treadmill Testing

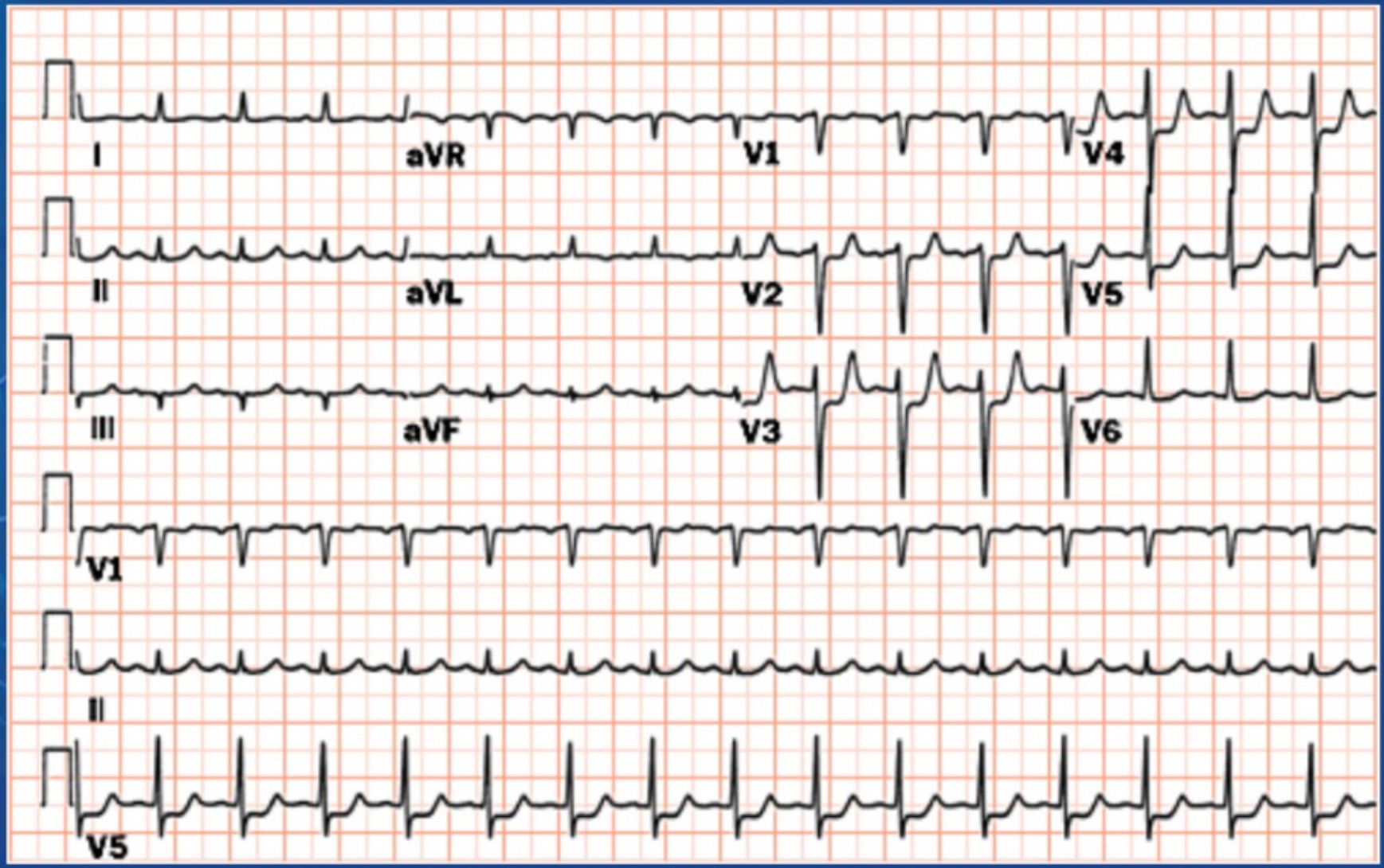
STAGE	TIME	SPEED (mph)	GRADE (%)	METS
REST	00.00	0.0	0.0	1.0
1	03.00	1.7	10.0	4.6
2	03.00	2.5	12.0	7.0
3	03.00	3.4	14.0	10.1
4	03.00	4.2	16.0	12.9
5	03.00	5.0	18.0	15.1
6	03.00	5.5	20.0	16.9
7	03.00	6.8	22.0	19.2

ST deletion
subendocardial
ischemia

Resting Electrocardiogram




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


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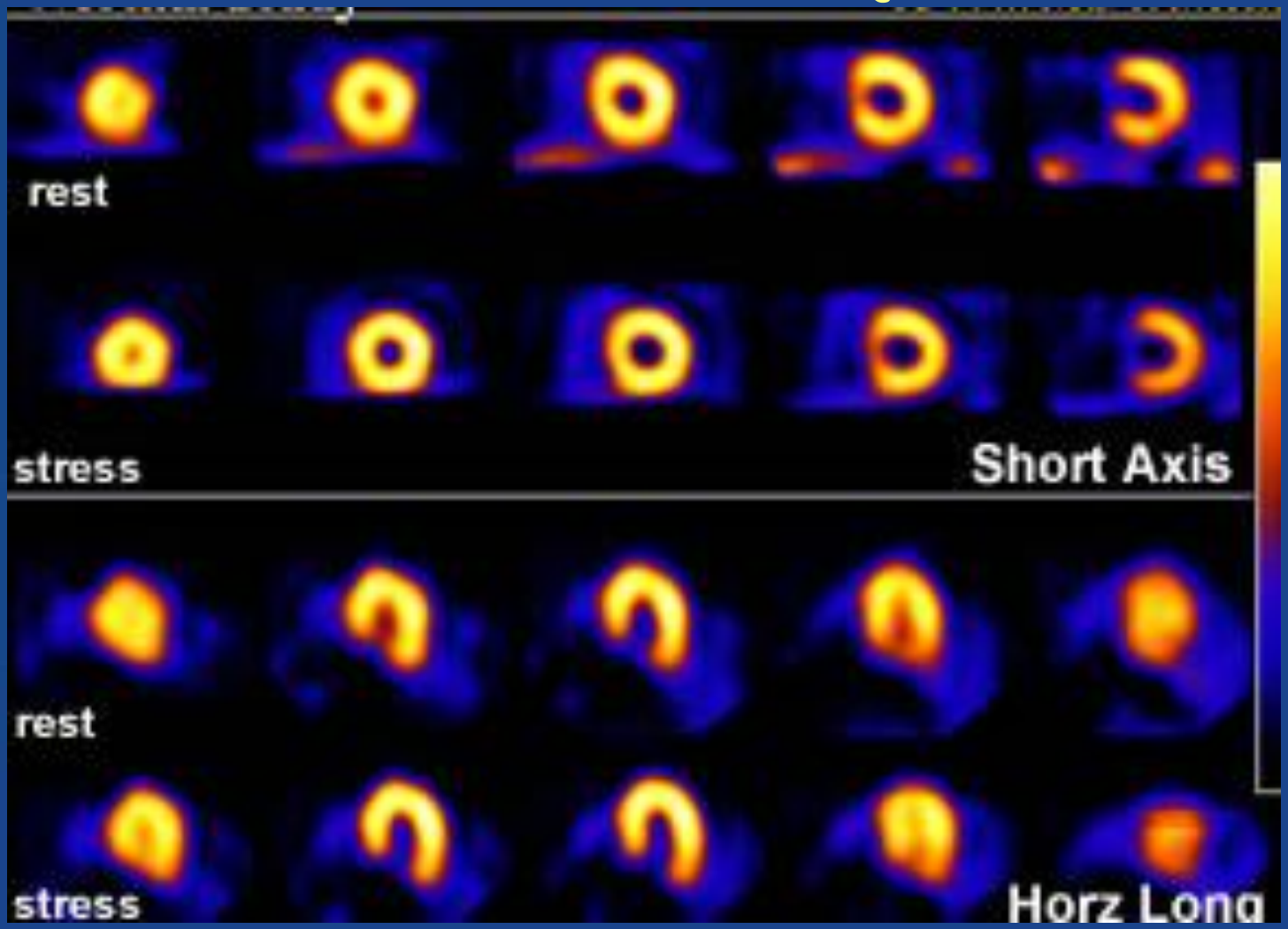
V2
V5
III
aVF
V3
V6
V1
II
V5

Types of stress test

- *Exercise or dipyridamole Thallium: Decrease uptake of the nuclear isotope during exercise*

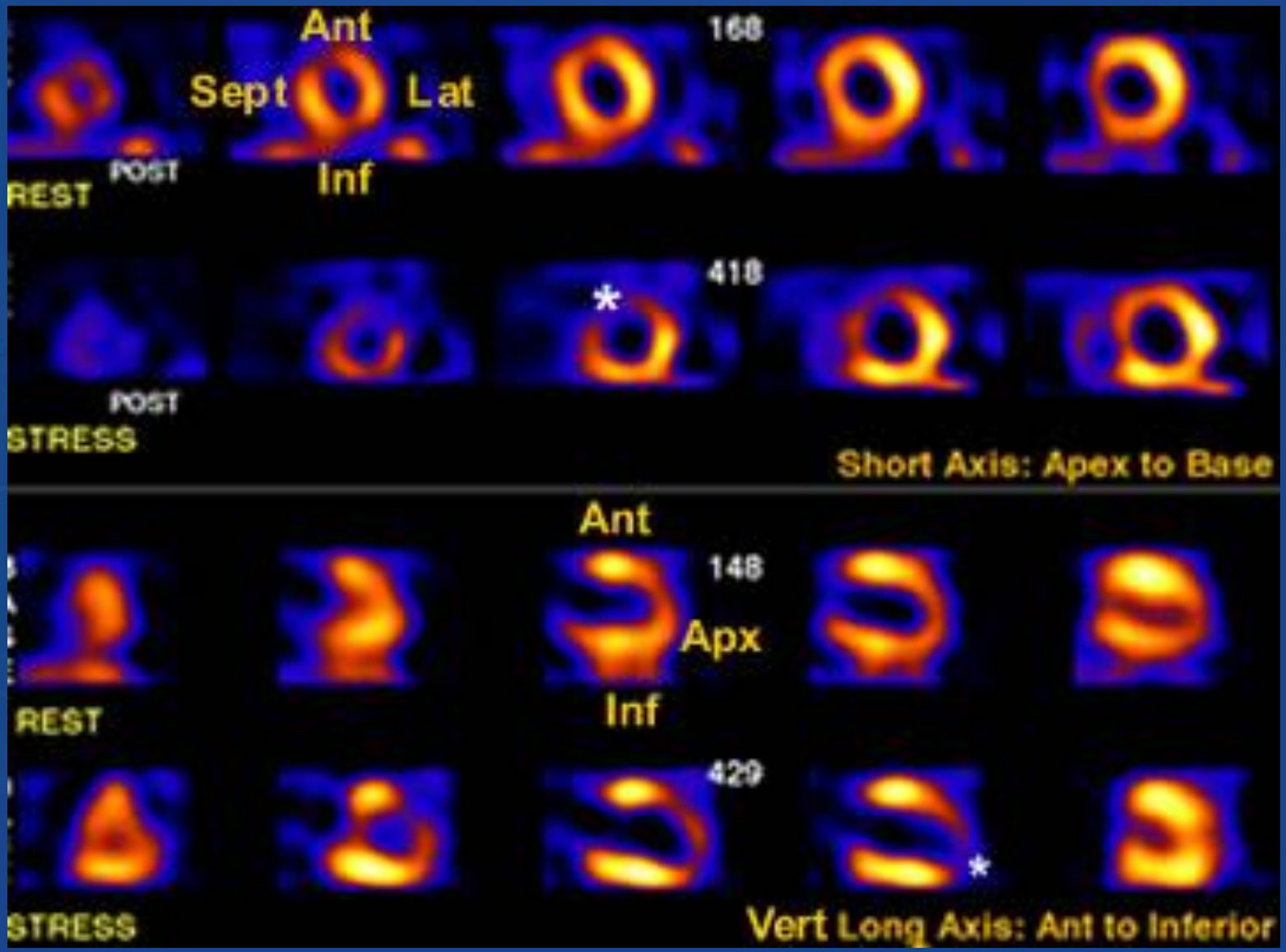
Normal Myocardial Perfusion

Reversable ischemia = stable anging



Infarct = constant ischemia

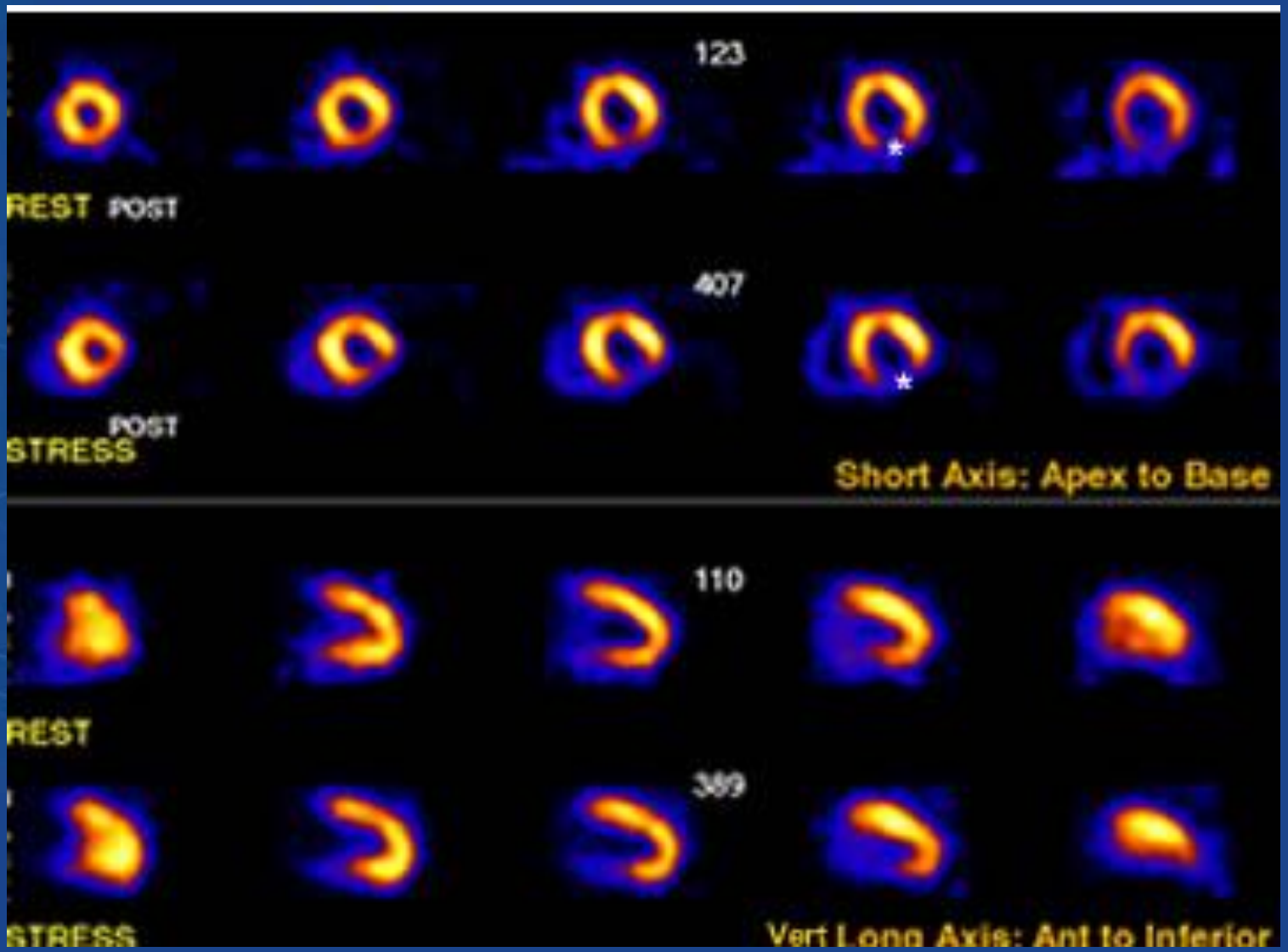
Myocardial Ischemia



ischemic → hyperkinetic

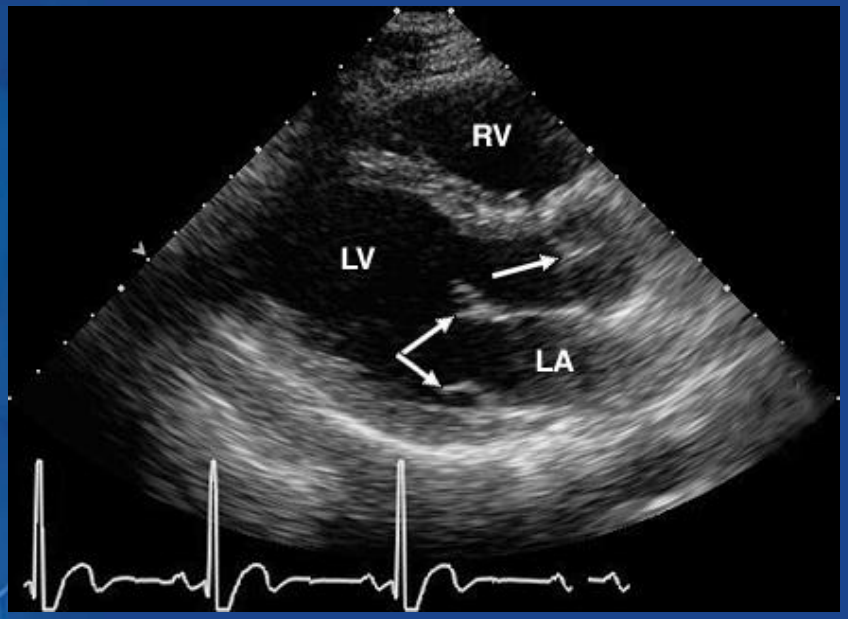
الطبيعي إنها تكون مشدودة و ما بتتحرك

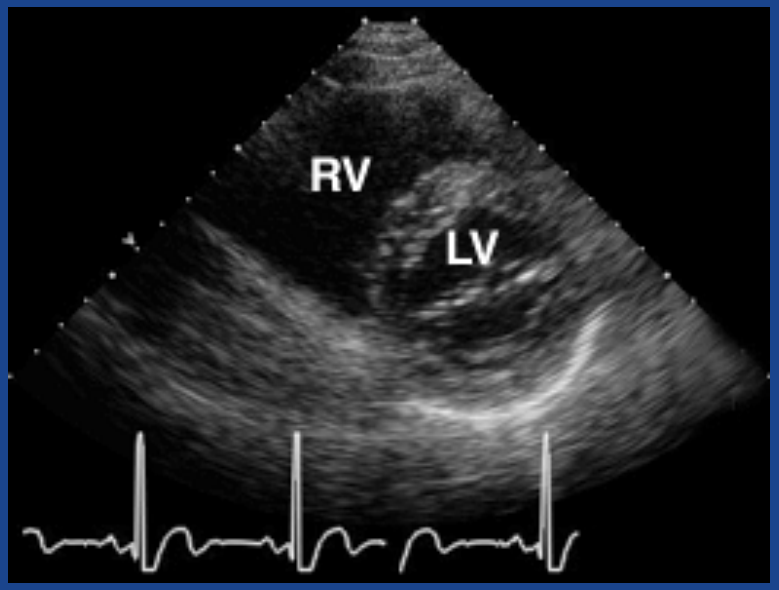
Myocardial Infarction



Types of stress test

- *Exercise or dobutamine Echocardiogram: Wall motion abnormalities*


RV
LV
LA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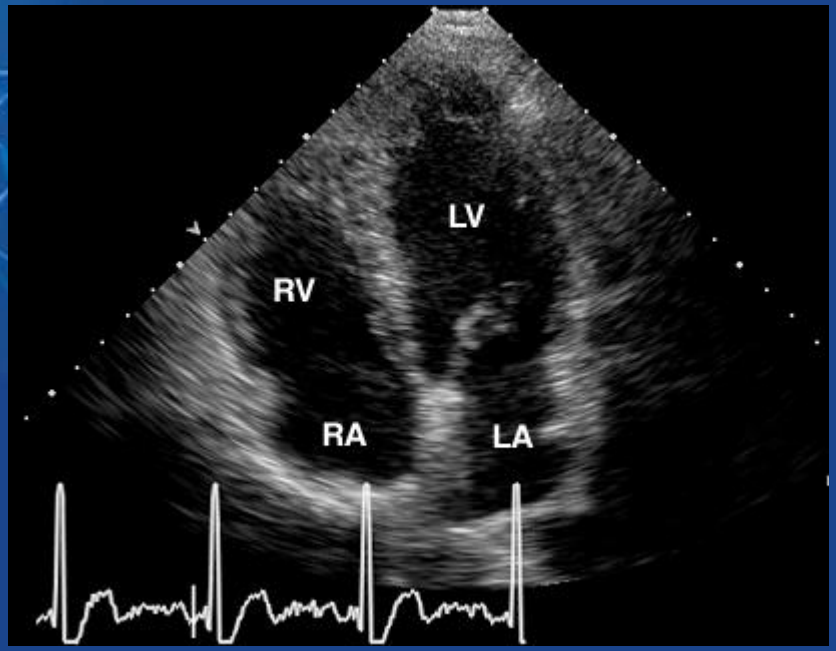
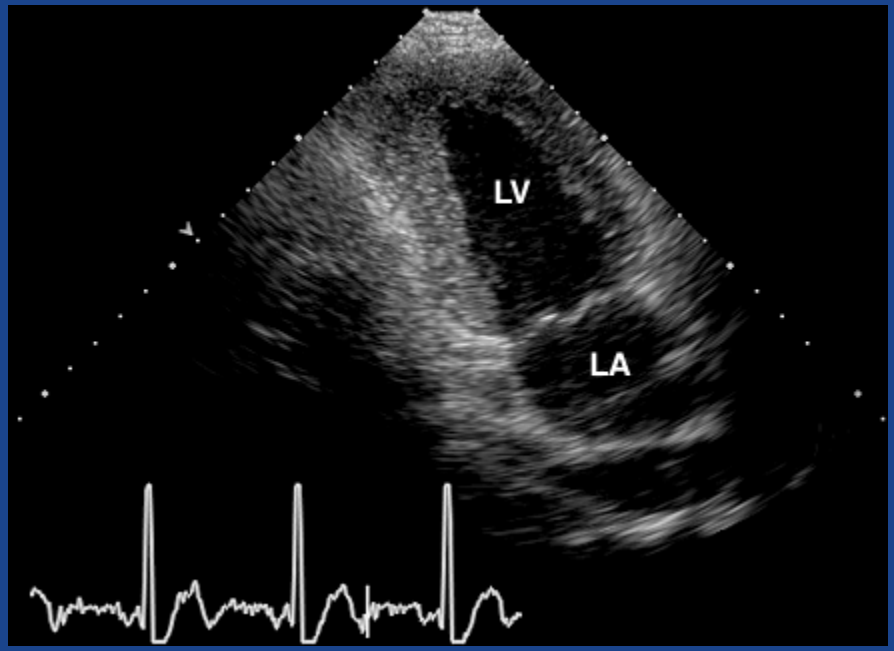

LV
RV
RA
LA


LV
LA

Imaging Techniques Used to Assess Atherosclerosis

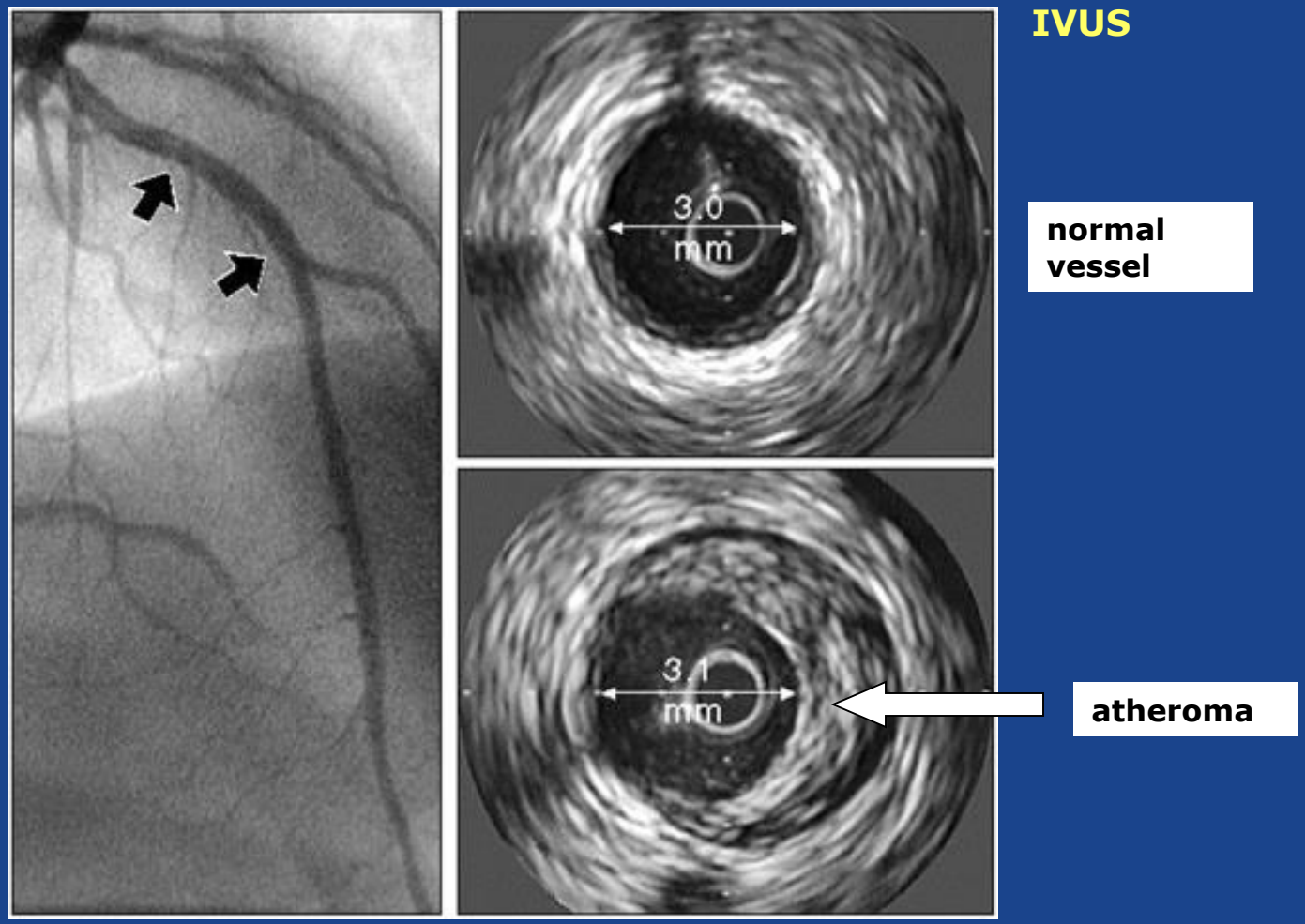
Invasive techniques

- *Coronary angiography*
- *Intravascular ultrasound (IVUS)*

Non-invasive techniques

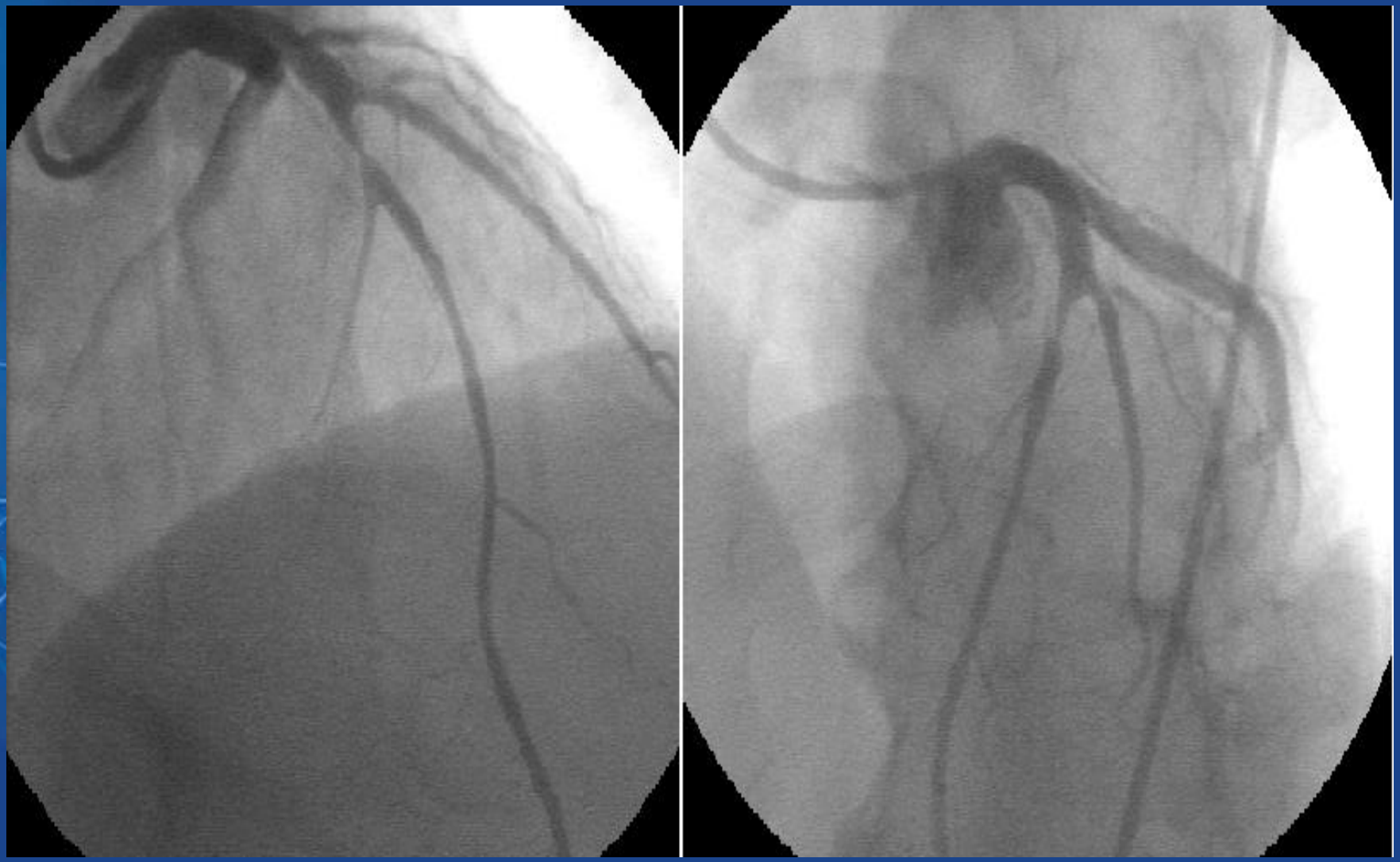
- *Magnetic resonance imaging (MRI)*
- *Computed tomography (CT)*
- *Ultrasound (B-mode)*

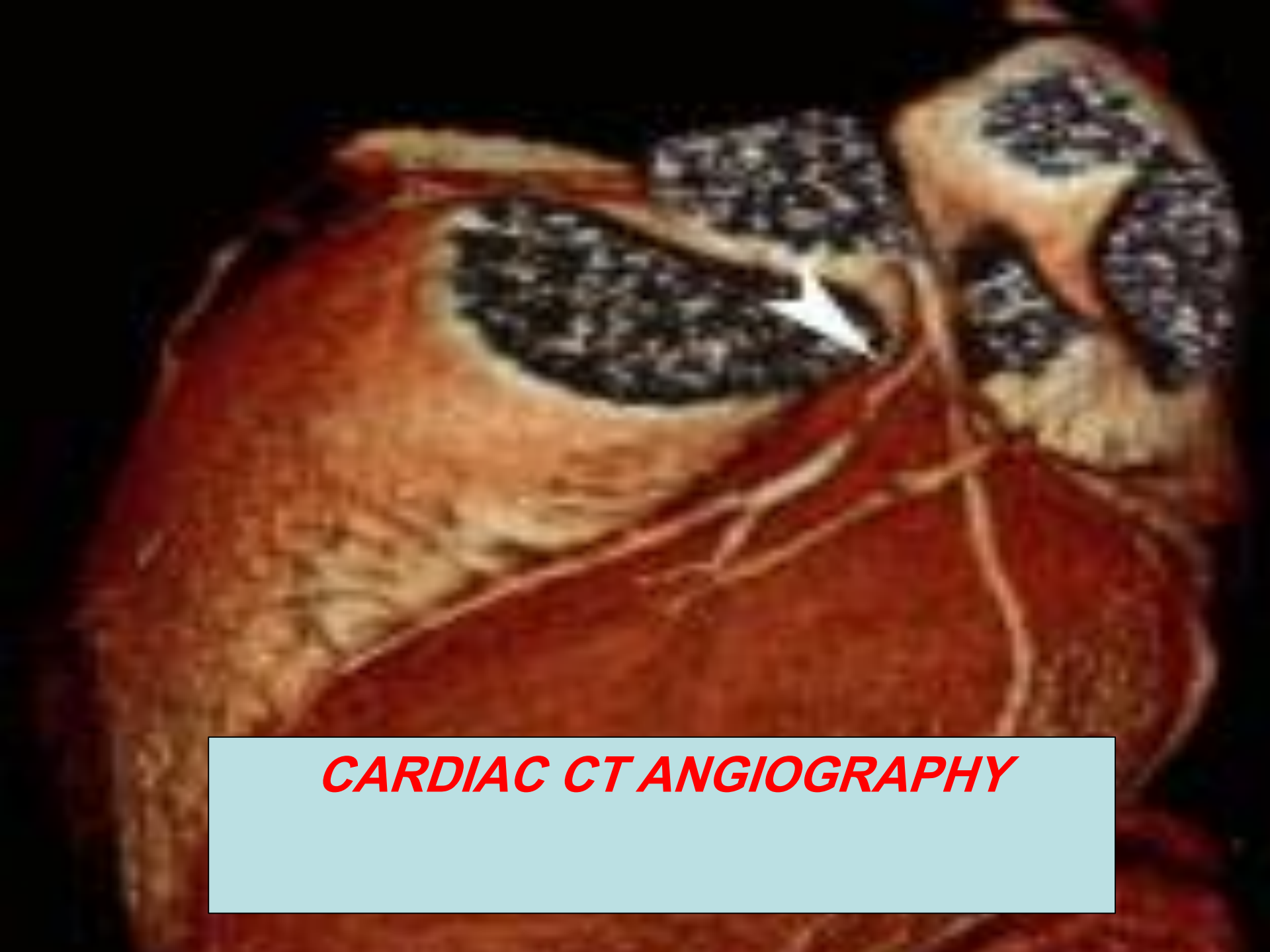
Intravascular Ultrasound (IVUS) Showing Atheromatous Plaque



Reproduced from *Circulation* 2001;**103**:604–616, with permission from Lippincott William & Wilkins.

Cardiac CATH




CARDIAC CT ANGIOGRAPHY

Management goals of stable angina

Main goals:

- **To improve prognosis (mortality reduction)**
  - *Modification of risk factors*
  - *Aspirin*
  - *Lipid-lowering therapy*
  - *ACE-Inhibitor*
  - *Revascularization procedures (PTCA, CABG)*

- **To decrease anginal symptoms**
  - *Medical treatment*

Treatment of stable angina

1- General measures

2- Medical therapy: Increase O2 supply
Decrease O2 demand

3-Revasularization: PCI (percutaneous coronary intervension)
CABG (coronary artery bypass grafting)

TREATMENT OF STABLE ANGINA
General Measures

- Correction of established risk factors(reversible)
- weight reduction (ideal body weight)
- Areobic exercise:
  improve functional capacity, well-being sensation
- Treatment of: anemia, thyrotoxicosis, arrhythmias,..

MEDICAL THERAPY OF STABLE ANGINA

Prognostic: Aspirin, Statines, ACEI

anti-platelet

B-B → ↓ HR
↑ time for flow
↑ diastole time

Symptomatic: Nitrate,B-,CA-blocker, (nicorandil, ranolazine, ivabradine)

CA B

INCREASE O2 Supply

1-Increase diastolic time: B-blocker

2-Decrease coronary tone: nitrate, ca-blocker

Dilation

3-Decrease LV diastolic pressure: nitrate

4-Correct coronary stenosis: PCI, CABG

5-Increase O2 capacity of blood: transfusion if anemia

DECREASE O2 Demand

1-Decrease heart rate: B-blocker, ca-blocker

2-Decrease contractility: B-blocker, ca-blocker

3- Decrease wall tension (LV pressure and cavity radius): nitrate

4- metabolic: trimetazidine

β-blocker
↑ diastole time
↓ HR
↓ contractility

Ca-blocker
↓ coronary tone
↓ HR
↓ contractility

nitrate
↓ coronary tone
↓ LV diastolic pressure
↓ LV wall tension

Treatment in practice

1-General measures

2-Aspirin → according to chest pain frequency
↳ sublingual

3-Nitrate: S/L, Oral, dermal

3-B-blocker

4-Statins: LDL>100 mg/dl(70mg/dl)

5-Ca-blocker

6-Angio :PTCA,CABG

New medical and invasive therapies for refractory angina

Inhibition of fatty acid metabolism: trimetazidine

Potassium channel activators: Nicorandil.

Ranolazine: interact with sodium channel

Ivabradine: SA inhibitor

Endothelin Receptor Blockers: bosentan

Testosteron: improve endoth dysfunction.

Enhanced external balloon counterpulsation

Spinal cord stimulation.

Laser revascularization, angiogenesis.

Prognostic Indicators of Coronary Artery Disease:

1- Left Ventricular Function:

Normal: 50-75%

< 50% associated with increased mortality

2- Vessel(s) involved: severity and extent of ischemia

mortality/year

2% single vessel-------12% left main stem

Differential diagnosis of angina

1- Neuromuscular disorder
2- Respiratory disorders
3-Upper GI disorder
4- Psychological
5- Syndrome X

Cardiac Syndrome X

↓ vasodilation on exersize

Typical, exertional angina with positive exercise stress test

normal coronary

Anatomically normal coronary arteries
Reduced capacity of vasodilation in microvasculature

F>M
Young > Elderly
Excellent prognosis
Antianginal therapy is rarely effective

Long term prognosis very good

Case History

A 45 Year old male, presented with recurrent attcaks of chest pain last few mintes, during attacks of chest pain the ECG abnormalities as attached ECG. After pain subside the ECG back to normal.

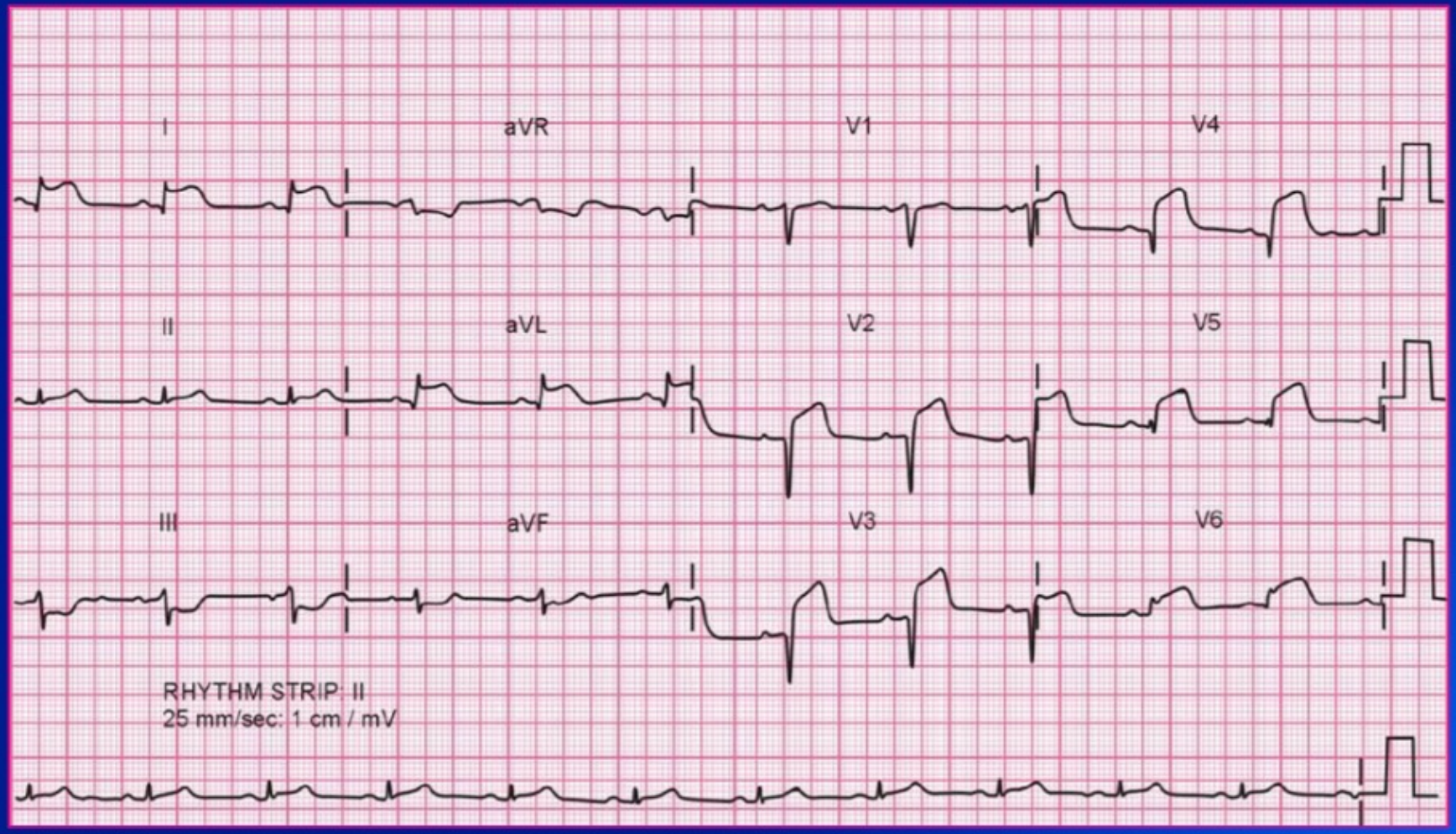
What is the diagnosis?

Intermediate ----

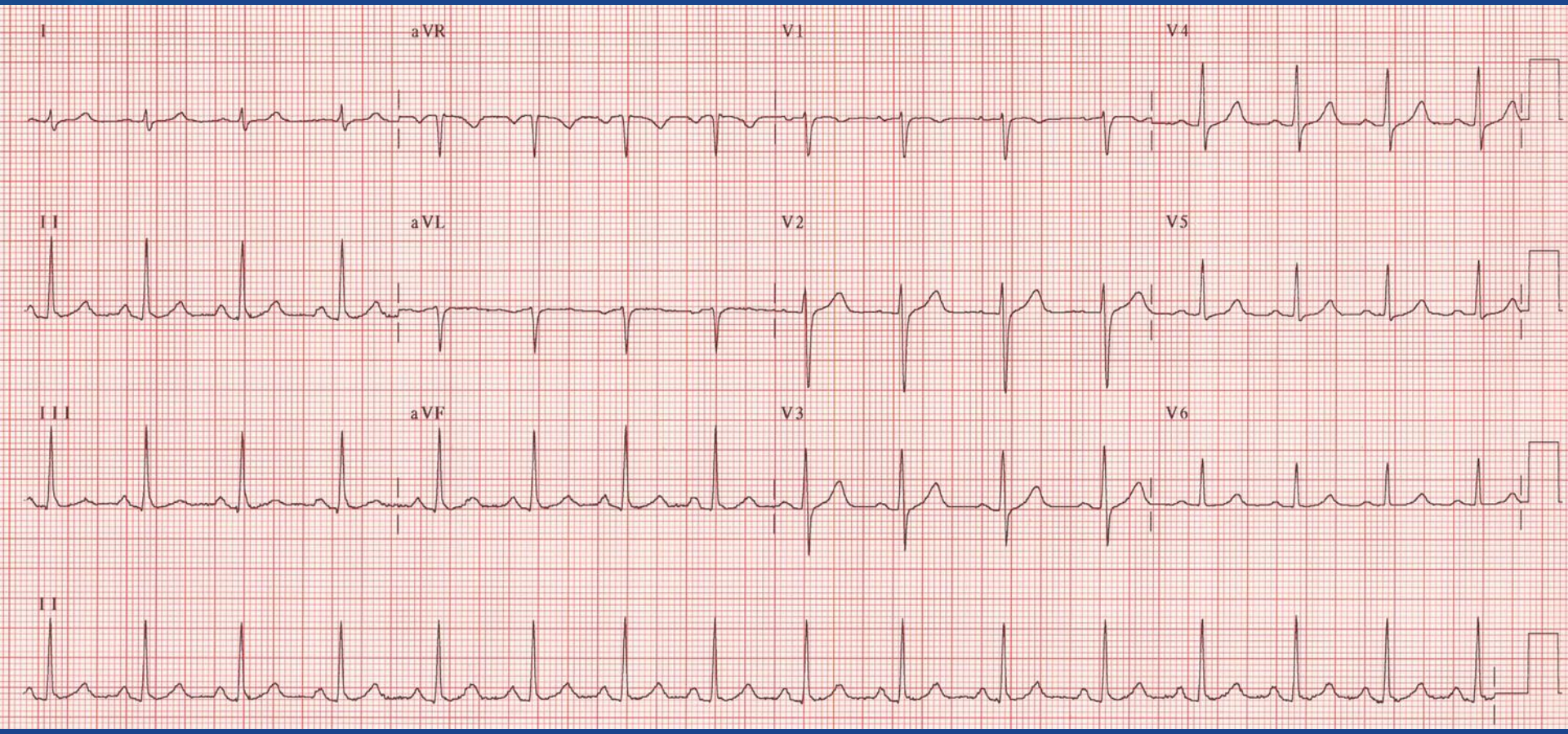
ST elevation
V1, V5, V6

after nitrate → relcived

During chest pain



After sublingual isosorbid dinitrate tablet



VARIANT ANGINA-PRINZMETAL ANGINA

Chest pain with ST-Segment elevation
Usually at rest, at night
Troponin: negative
Female > male

Spasm of large epicardial coronary vessel during the attack
transmural ischemia
70% on top of atherosclerosis
Vasospastic symptpms in other organs: Migraine, rhynauds

Can cause arrhythmias and death

Treatment: CA-blocker, Nitrate → BB blocks β-receptors & leave α-receptors + cause vasoconstriction

B-blocker is contraindicated

Prognosis: 5 year mortality < 5%

Thank you