Ischemic heart disease

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Definition

- *Ischemia* is defined as inadequate blood supply (circulation) to a local area due to blockage of the blood vessels supplying the area.
- *Ischemic* means that an organ (e.g., the heart) is not getting enough blood and oxygen.
- ***Ischemic heart disease (IHD)***, also called ***coronary heart disease (CHD)*** or coronary artery disease, is the term given to heart problems caused by narrowed heart (coronary) arteries that supply blood to the heart muscle.

Ischemic Heart Disease/IHD (Coronary Heart Disease)

Causes

*The most common cause of IHD is coronary artery atherosclerosis (90% of cases).

*Less commonly it is due to vasospasm and vasculitis.

Ischemic heart disease

Classification

- -Angina pectoris (chest pain).
- -Acute myocardial infarction.
- -Sudden cardiac death.
- -IHD with rhythm and conduction disorders
- -Chronic ischemic heart disease with congestive heart

Ischemic heart disease

Main simptoms

- Chest pain (angina)
- Shortness of breath
- Fatigue
- Sweating
- Palpitations

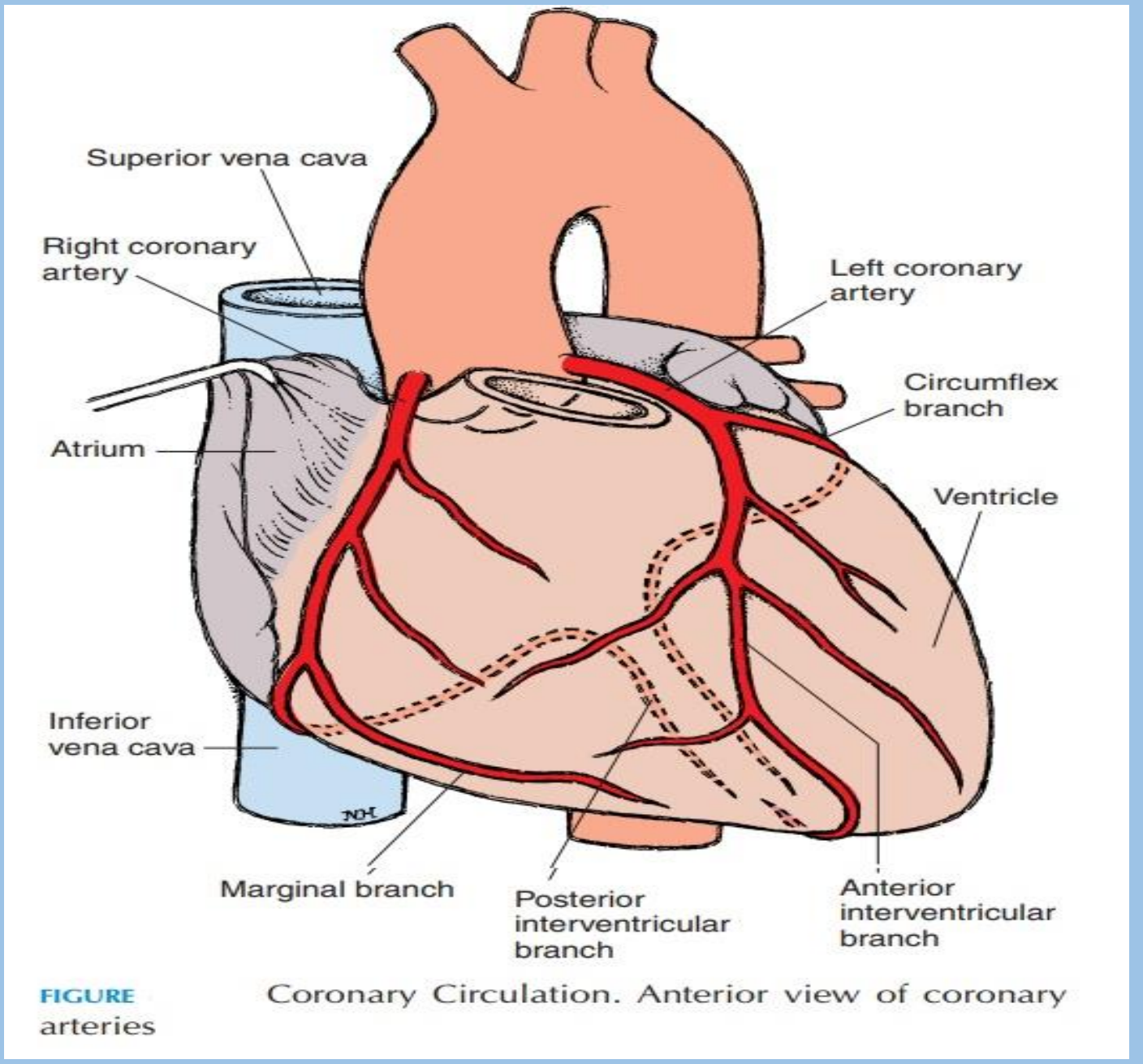
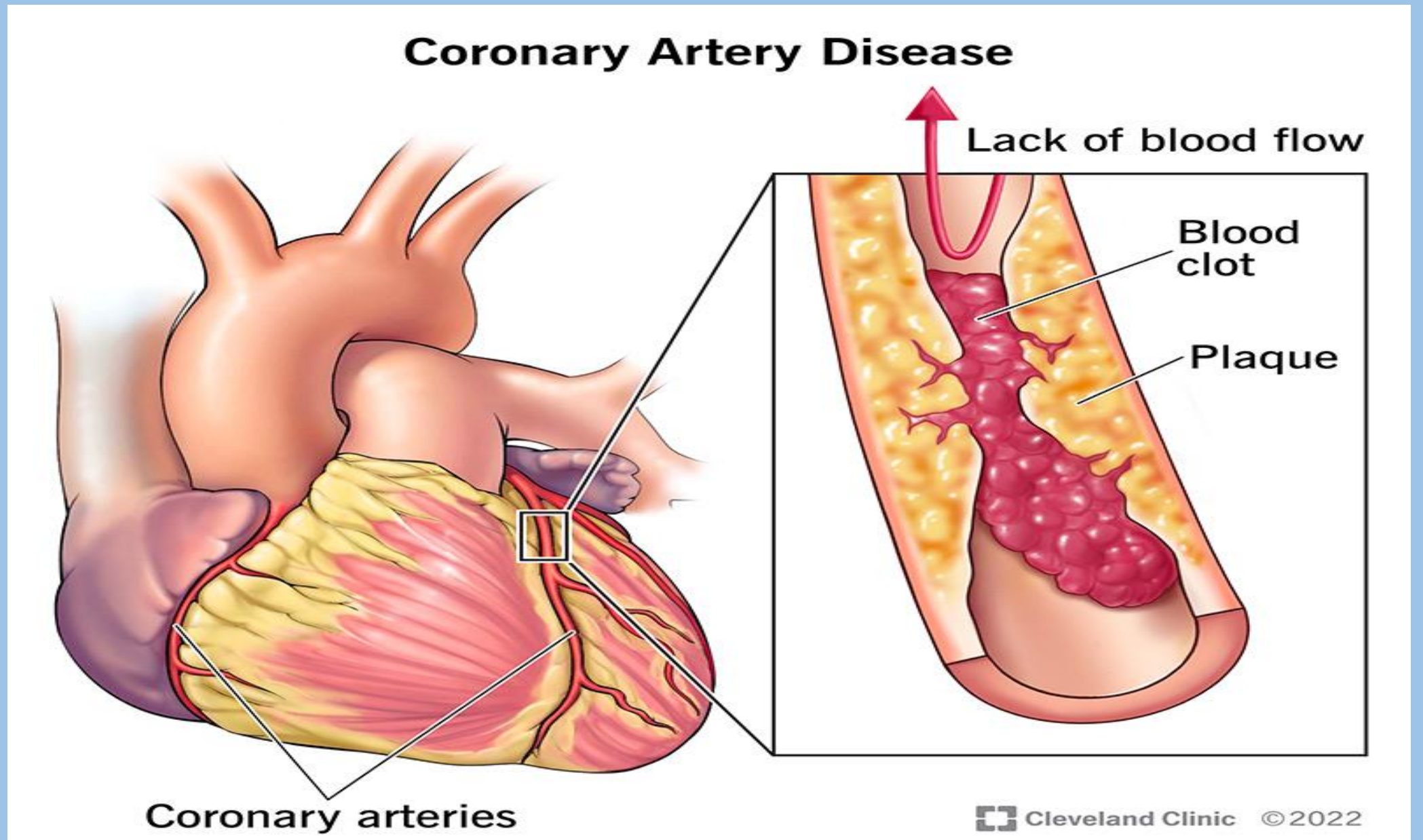
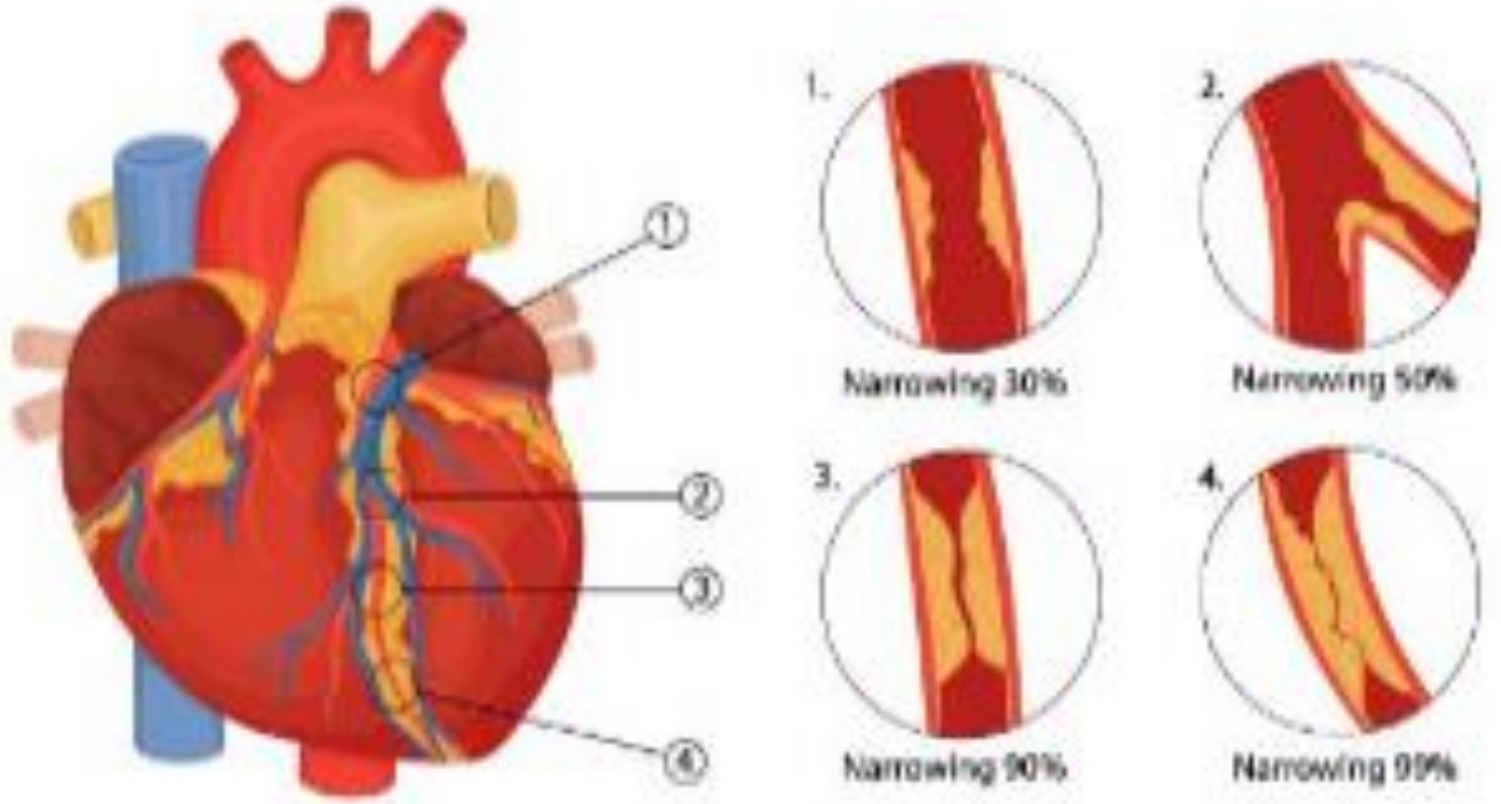


FIGURE Coronary Circulation. Anterior view of coronary arteries


Coronary Artery Disease
Lack of blood flow
Blood clot
Plaque
Coronary arteries
Cleveland Clinic ©2022

Cardiac ischemia



Ischemic heart disease

- Although the narrowing can be caused by a blood clot or by constriction of the blood vessel, most often it is caused by buildup of plaque, called atherosclerosis.
- When the blood flow to the heart muscle is completely blocked, the heart muscle cells die, which is termed a heart attack or myocardial infarction (MI).
- Most people with early (less than 50 percent narrowing) CHD do not experience symptoms or limitation of blood flow. However, as the atherosclerosis progresses, especially if left untreated, symptoms may occur. They are most likely to occur during exercise or emotional stress, when the demand for the oxygen carried by the blood increases.

Angina pectoris

- *Angina pectoris* is a type of IHD characterized by paroxysmal and usually recurrent attacks of substernal or precordial chest discomfort, described as constricting, crushing, squeezing, choking, or knifelike pain. May radiate down the left arm or to the left jaw (called as referred pain).

*Angina pectoris is an intermittent chest pain caused by transient, reversible myocardial ischemia.

- is due to inadequate perfusion and is caused by transient (15 seconds to 15 minutes) myocardial ischemia that falls short of inducing the cellular necrosis that defines infarction i.e. duration and severity is not sufficient for infarction.

Angina pectoris

There are three types of angina pectoris:

1.-Stable angina / typical angina pectoris:

- - The most common form of angina.
- - It is caused by atherosclerotic disease with usually ≥70% to 75% narrowing of lumen i.e. (critical stenosis or fixed chronic stable stenosis).
- - This reduction (70 to 75% stenosis) of coronary vessels makes the heart vulnerable, so whenever there is increased demand, e.g. physical activity, emotional excitement, or any other cause of increased cardiac workload, there is angina pain.
- - The chest pain is episodic and associated with exertion or some other form of stress.
- - Is usually relieved by rest (thereby decreasing demand) or with a strong vasodilator like nitroglycerin.

Stable angina pectoris

(clinical forms)

- A. Stable exertional angina pectoris:

-Coronary pain on exertion or equivalent to exertion (cold, emotions, postprandial status)

-The pain always occurs at the same threshold of exertion, lasts 3-5 min, promptly gives way to sublingual Nitroglycerin

- B. Spontaneous angina pectoris, stable form:

-Occurs at rest, sometimes at night. Indicates more severe coronary lesions or coronary spasm

-The stable character is indicated by the short duration of the attacks (3-5 min), the average intensity, the prompt response to sublingual Nitroglycerin

- C. Mixed stable angina occurs at rest and during exertion

Angina pectoris

2. -Unstable or crescendo angina:

- - It is an unstable and progressive condition.
- - Pain occurs with progressively increasing frequency, and is precipitated with progressively less exertion, even at rest, and tends to be of more prolonged duration.
- - It is induced by disruption or rupture of an atheroma plaque with superimposed partial thrombosis. Not complete rupture. If it complete=MI.
- !!! Unstable angina is often the precursor of subsequent acute MI. Thus also called as preinfarction angina

Angina pectoris

3. Angina variant Prinzmetal

- - It's an uncommon pattern of episodic angina that occurs at rest and is due to coronary artery spasm.
- - Prinzmetal angina generally responds promptly to vasodilators, such as nitroglycerin and calcium channel blockers.
- - Not related to atherosclerotic disease.
- - Pain at rest.
- - The etiology is not clear.

Classification og Angina pectoris

TABLE 7-1 Canadian Cardiovascular Society Functional Classification of Angina

Class	Definition	Limitations
I	No limitation of ordinary activity. Angina occurs with strenuous, rapid, or prolonged exertion at work or recreation.	Angina may occur with chopping wood, climbing hills, cycling, aerobic ballet, ballroom (fast) or square dancing, jogging a 10-minute mile, rope skipping, skating, skiing, playing tennis or squash, and walking 5 miles per hour.
II	Slight limitation of ordinary activity.	Angina may occur with walking or climbing stairs rapidly, walking uphill, walking or climbing stairs after meals; in cold or in wind; under emotional stress; only during the first few hours after awakening; or with walking more than two blocks on level ground and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions.
III	Marked limitation of ordinary physical activity.	Angina may occur with walking one or two blocks on level ground, and climbing one flight of stairs in normal conditions and at normal pace, playing a musical instrument, performing household chores, gardening, vacuuming, walking a dog, or taking out the trash.
IV	Inability to perform any physical activity without discomfort.	Angina may occur at rest.

SOURCE: Adapted from Goldman et al., 1981.

Angina pectoris

- Angina usually occurs in patients with CHD, but also can occur in individuals with valvular disease, hypertrophic cardiomyopathy, and uncontrolled hypertension.
- Infrequently, patients with normal coronary arteries may experience angina related to coronary spasm or endothelial dysfunction.

DIAGNOSTIC CRITERIA AND METHODS for IHD

- Stress testing is usually performed using an exercise tolerance test (ETT) with a treadmill or, occasionally, with bicycle ergometry.

- Exercise testing can be performed with electrocardiogram (ECG) monitoring alone or combined with a cardiac imaging test: single photon emission computed tomography (SPECT), positron emission tomography (PET), or with echocardiography imaging.

- Each modality has specific criteria for an abnormal test. An abnormal exercise ECG is defined by ST-segment displacement, usually an ST-segment depression greater than or equal to 1 mm, measured 0.08 seconds after the J-point, that is horizontal or downsloping. ST-segment elevation greater than or equal to 1 mm in leads without Q waves occurs infrequently, but this is also considered an abnormal response

DIAGNOSTIC CRITERIA AND METHODS for IHD

- Pharmacologic stress testing using SPECT, PET, or echocardiographic imaging is reserved for patients who are either unable to perform dynamic exercise or unable to achieve at least 85 % of the age-predicted maximal heart rate with exercise, which is the effort level required to achieve adequate sensitivity to detect coronary artery stenosis capable of causing angina. Pharmacologic stress does not consistently cause angina or ECG changes of ischemia, so only the imaging results are diagnostic. Pharmacologic agents are administered intravenously in place of dynamic exercise stress, and the resulting perfusion or wall motion response is compared with the resting state and is interpreted using the same criteria for perfusion defects and wall motion abnormalities listed above for dynamic exercise.

- The most frequently used pharmacologic stress agents for SPECT and PET are the vasodilators dipyridamole, adenosine, and regadenoson, which increase blood flow through the coronary arteries, but only modestly increase heart rate in most patients. Many patients experience chest discomfort during the administration of these agents, which should not be interpreted as angina. The agents create differences in blood flow between coronary arteries that have high-grade blockages and normal arteries, which result in perfusion defects that can be detected using radioactive imaging.

DIAGNOSTIC CRITERIA AND METHODS for IHD

Coronary CT Angiography is an imaging technique during which an iodinated contrast dye is injected through a peripheral vein and images of the coronary arteries are taken using a CT system. It provides images of the coronary arteries similar to those obtained using coronary angiography, during which the dye is injected directly into the coronary arteries using an arterial catheter. It is most useful in patients with an intermediate risk of coronary heart disease. In patients with extensive calcium deposits or prior coronary artery stents, detection of stenosis is difficult

TREATMENT of IHD

Comprehensive management of angina and stable CHD entails multiple therapeutic approaches, including the following:

- Identification and treatment of associated diseases that can precipitate or worsen angina and ischemia;
- Cardiac risk factor identification and intervention;
- Application of pharmacological and nonpharmacological interventions for secondary prevention;
- Pharmacological and symptomatic management of angina and ischemia; and
- Myocardial revascularization with PCI or CABG surgery, when indicated.

Treatment of IHD

- **lifestyle changes** – quitting smoking, losing weight, exercising, getting 7-9 hours sleep, eating a healthy diet etc.
- **medications** - aspirin and other anti-platelet agents (‘blood thinners’), statins, beta blockers, nitrates, ACE inhibitors, calcium channel blockers etc.
- **other therapies to address risk factors and ischaemic heart disease progression** – such as taking medications to control blood pressure, diabetes, and cholesterol
- **angioplasty** – where a small balloon is inserted into the artery and inflated to improve blood flow
- **stenting** – where a small wire mesh coil (stent) is inserted into the artery to improve blood flow
- **coronary artery bypass surgery** – where a blood vessel from another area of the body is used to create a graft that can bypass the narrowed artery to improve blood flow
- **heart transplant**, in select but severe cases

Minimally invasive and surgical treatments for IHD

- **Implantable cardioverter defibrillator (ICD)**
- **Cardiac resynchronization therapy (CRT)** by biventricular pacemaker.
- **Percutaneous coronary intervention (PCI)** - a catheter-based procedure to open a blocked artery..
- **Atherectomy**
- **Coronary artery bypass graft (CABG)** surgery (a blood vessel from another part of your body to create a new path around one or more blocked arteries).

MIOCARDIAL INFARCTION

Myocardial infarction (MI), colloquially known as "heart attack," is caused by decreased or complete cessation of blood flow to a portion of the myocardium.

*MI may be"*silent*," and go *undetected*, or it could be a *catastrophic event* leading to hemodynamic deterioration and *sudden death*.

*Most MIs are due to underlying coronary artery disease, the leading cause of death in the world.

*With coronary artery occlusion, the myocardium is deprived of oxygen.

*Prolonged deprivation of oxygen supply to the myocardium can lead to myocardial cell death and necrosis.[

Clinical forms of AMI

Typical forms of AMI:

- anginous form (pain lasts more than 30 min)

Atypical forms of AMI:

- Asthmatic form – like an hit of bronchial asthma
- Abdominal form – like an acute abdomen, with a dyspeptic syndrome (often in old people)
- Cerebral form – like the symptoms in stroke
- Arrhythmic form – it begins with arrhythmias.

MI –etiology

- Smoking
- Abnormal lipid profile/blood apolipoprotein (raised ApoB/ApoA1)
- Hypertension
- Diabetes mellitus
- Abdominal obesity (waist/hip ratio) (greater than 0.90 for males and greater than 0.85 for females)
- Psychosocial factors such as depression, loss of the locus of control, global stress, financial stress, and life events including marital separation, job loss, and family conflicts
- Lack of daily consumption of fruits or vegetables
- Lack of physical activity
- Alcohol consumption (weaker association, protective)

MI –etiology

- a moderately high level of plasma *homocysteine*, which is an independent risk factor of MI.
- Some *non-modifiable* risk factors for myocardial infarction include:

- advanced age,
- male gender (males tend to have myocardial infarction earlier in life),
- -genetics (there is an increased risk of MI if a first-degree relative has a history of cardiovascular events before the age of 50.

MI - Pathophysiology

- The acute occlusion of one or multiple large epicardial coronary arteries for more than 20 to 40 minutes can lead to acute MI.
- The occlusion is usually thrombotic and due to the rupture of a plaque formed in the coronary arteries.
- The occlusion leads to a lack of oxygen in the myocardium, which results in sarcolemmal disruption and myofibril relaxation. These changes are one of the first ultrastructural changes in the process of MI, which are followed by mitochondrial alterations.
- The prolonged ischemia ultimately results in liquefactive necrosis of myocardial tissue.
- The necrosis spreads from sub-endocardium to sub-epicardium. The subepicardium is believed to have increased collateral circulation, which delays its death.
- Depending on the territory affected by the infarction, the cardiac function is compromised.
- Due to the negligible regeneration capacity of the myocardium, the infarcted area heals by scar formation, and often, the heart is remodeled characterized by dilation, segmental hypertrophy of remaining viable tissue, and cardiac dysfunction.

MI - evaluation

- *The three components in the evaluation of the MI are:*

- clinical features (history and physical)
- ECG findings
- cardiac biomarkers

MI - History and Physical

- MI can present as chest pain, upper extremity pain, mandibular, or epigastric discomfort that occurs during exertion or at rest; dyspnea or fatigue, which are known to be ischemic equivalents.
- The chest pain is usually retrosternal and is sometimes described as the sensation of pressure or heaviness. The pain often radiates to the left shoulder, neck, or arms with no obvious precipitating factors, and it may be intermittent or persistent. The pain usually lasts for more than 20 minutes. It is usually not affected by positional changes or active movement of the region.
- Additional symptoms, such as sweating, nausea, abdominal pain, dyspnea, and syncope, may also be present.
- The MI can also present atypically with subtle findings such as palpitations, or more dramatic manifestations, such as cardiac arrest.
- The MI can sometimes present with no symptoms.

MI -ECG

- The resting 12 lead ECG is the first-line diagnostic tool for the diagnosis of acute coronary syndrome (ACS).
- It should be obtained within 10 minutes of the patient's arrival in the emergency department.
- Acute MI is often associated with dynamic changes in the ECG waveform.
- Serial ECG monitoring can provide important clues to the diagnosis if the initial EKG is non-diagnostic at initial presentation.
- Serial or continuous ECG recordings may help determine reperfusion or re-occlusion status.
- A large and prompt reduction in ST-segment elevation is usually seen in reperfusion

MI -ECG

- ECG findings suggestive of ongoing coronary artery occlusion (in the absence of left ventricular hypertrophy and bundle branch block):

**ST-segment elevation in two contiguous lead (measured at J-point) of*

-Greater than 5 mm in men younger than 40 years, greater than 2 mm in men older than 40 years, or greater than 1.5 mm in women in leads V2-V3 and/or

-Greater than 1 mm in all other leads

**ST-segment depression and T-wave changes*

-New horizontal or down-sloping ST-segment depression greater than 5 mm in 2 contiguous leads and/or T inversion greater than 1 mm in two contiguous leads with prominent R waves or R/S ratio of greater than 1

MI -ECG

- The hyperacute T-wave amplitude, with prominent symmetrical T waves in two contiguous leads, maybe an early sign of acute MI that may precede the ST-segment elevation.

- Other ECG findings associated with myocardial ischemia include cardiac arrhythmias, intraventricular blocks, atrioventricular conduction delays, and loss of precordial R-wave amplitude (less specific finding).

MI -ECG

- ECG changes associated with prior MI (in the absence of left ventricular hypertrophy and left bundle branch block):

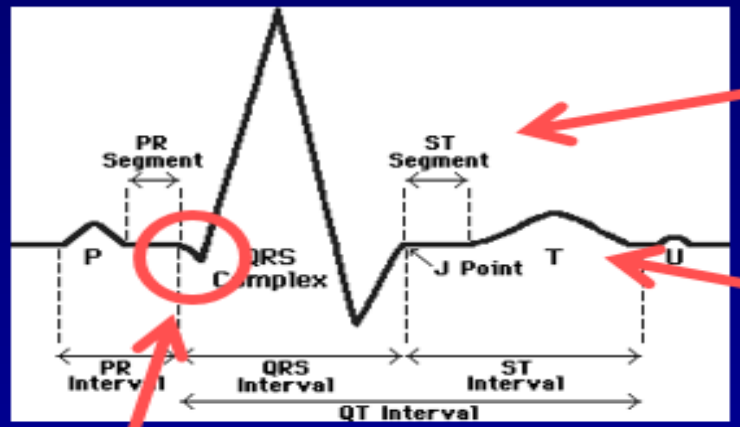
-Any Q wave in lead V2-V3 greater than 0.02 s or QS complex in leads V2-V3

-Q wave > 03 s and greater than 1 mm deep or QS complex in leads I, II, aVL, aVF or V4-V6 in any two leads of contiguous lead grouping (I, aVL; V1-V6; II, III, aVF)

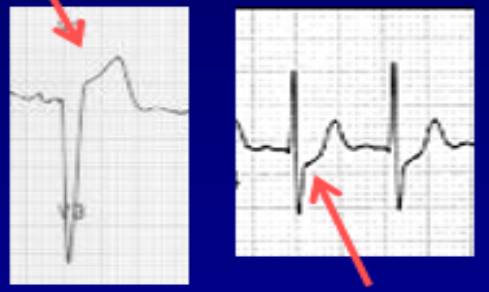
-R wave > 0.04 s in V1-V2 and R/S greater than 1 with a concordant positive T wave in the absence of conduction defect.

ECG Changes

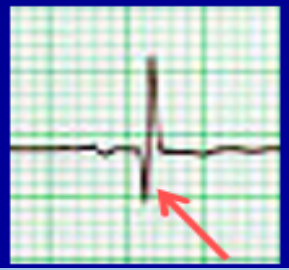
Ways the ECG can change include:



ST elevation & depression

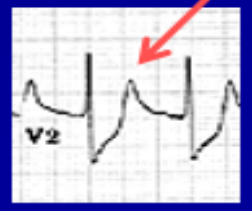


Appearance of pathologic Q-waves

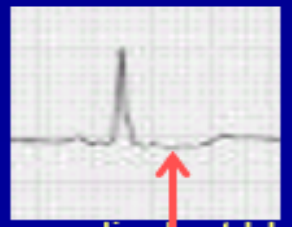


T-waves

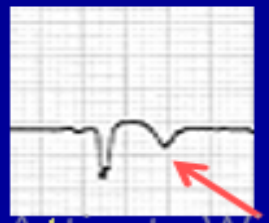
peaked



flattened

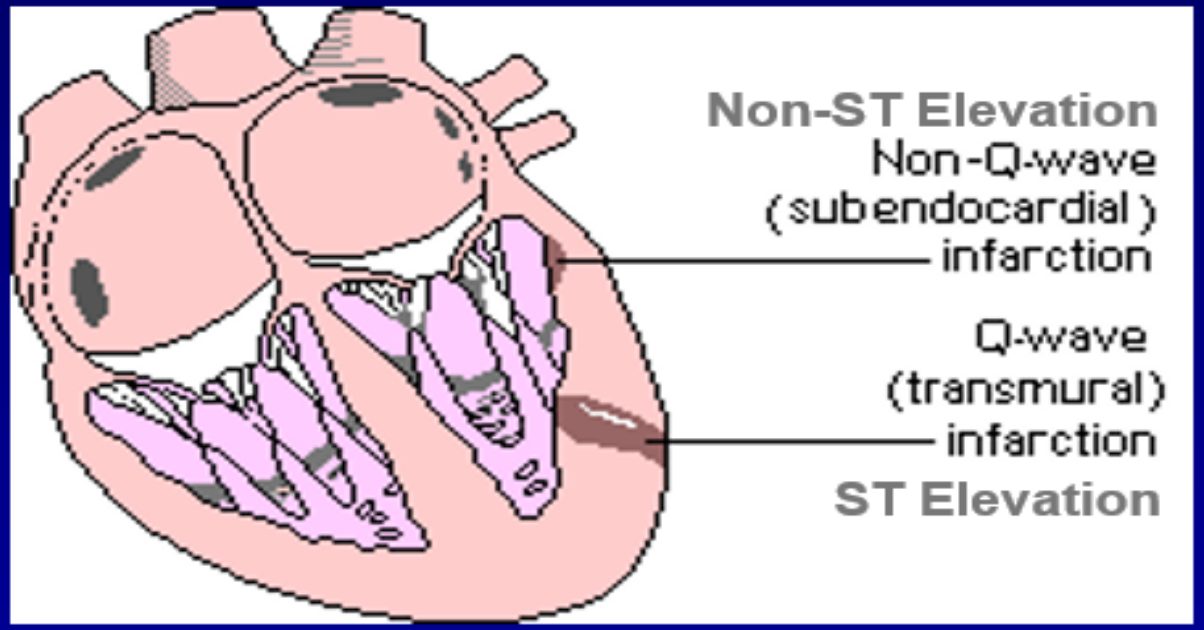


inverted



ECG Changes & the Evolving MI

There are two distinct patterns of ECG change depending if the infarction is:



–ST Elevation (Transmural or Q-wave), or

–Non-ST Elevation (Subendocardial or non-Q-wave)

ST Elevation Infarction

The ECG changes seen with a ST elevation infarction are:

Before injury	**Normal ECG**
↓	
Ischemia	**ST depression, peaked T-waves, then T-wave inversion**
↓	
Infarction	**ST elevation & appearance of Q-waves**
↓	
Fibrosis	**ST segments and T-waves return to normal, but Q-waves persist**

ST Elevation Infarction

Here's a diagram depicting an evolving infarction:

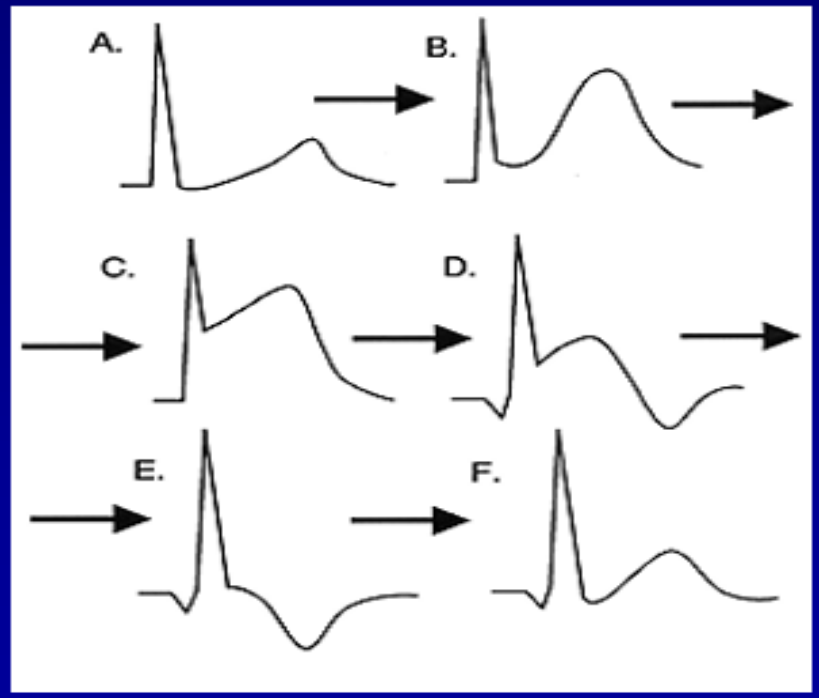
A. Normal ECG prior to MI

B. Ischemia from coronary artery occlusion results in ST depression (not shown) and peaked T-waves

C. Infarction from ongoing ischemia results in marked ST elevation

D/E. Ongoing infarction with appearance of pathologic Q-waves and T-wave inversion

F. Fibrosis (months later) with persistent Q-waves, but normal ST segment and T-



Non-ST Elevation Infarction

The ECG changes seen with a non-ST elevation infarction are:

Before injury	**Normal ECG**
↓	
Ischemia	**ST depression & T-wave inversion**
↓	
Infarction	**ST depression & T-wave inversion**
↓	
Fibrosis	**ST returns to baseline, but T-wave inversion persists**

Biomarker Detection of MI

- Cardiac troponins (I and T) are components of the contractile apparatus of myocardial cells and expressed almost exclusively in the heart.
- The rising and/or falling pattern of cardiac troponins (cTn) values with at least one value above the 99 percentile of upper reference limit (URL) associated with symptoms of myocardial ischemia would indicate an acute MI.
- Serial testing of cTn values at 0 hours, 3 hours, and 6 hours would give a better perspective on the severity and time course of the myocardial injury. Depending on the baseline cTn value, the rising/falling pattern is interpreted. If the cTn baseline value is markedly elevated, a minimum change of greater than 20% in follow up testing is significant for myocardial ischemia.
- Creatine kinase MB isoform can also be used in the diagnosis of MI, but it is less sensitive and specific than cTn level.

MI - Imaging

Different imaging techniques are used to assess myocardial perfusion, myocardial viability, myocardial thickness, thickening and motion, and the effect of myocyte loss on the kinetics of para-magnetic or radio-opaque contrast agents indicating myocardial fibrosis or scars:

- Echocardiography (regional wall motion abnormalities induced by ischemia can be detected by echocardiography almost immediately after the onset of ischemia when greater than 20% transmural myocardial thickness is affected)
- radionuclide imaging,
- cardiac magnetic resonance imaging (cardiac MRI) - provides an accurate assessment of myocardial structure and function.

MI - Acute Management

*Reperfusion therapy is indicated in all patients with symptoms of ischemia of less than 12-hours duration and persistent ST-segment elevation.

-Primary percutaneous coronary intervention (is preferred to fibrinolysis if the procedure can be performed <120 minutes of ECG diagnosis.)

-Fibrinolysis

*Relief of pain, breathlessness, and anxiety:

-Intravenous opioids (e.g., morphine)

-A mild anxiolytic (usually a benzodiazepine) may be considered in very anxious patients

-Supplemental oxygen is indicated in patients with hypoxemia (SaO2 <90% or PaO2 <60mm Hg)

*Nitrates

*Beta-blockers

*Platelet inhibition

MI -Long-Term Management

- *Lipid-lowering treatment (statins)*
- *Antithrombotic therapy:*
- *ACE inhibitors* are recommended in patients with systolic left ventricular dysfunction, or heart failure, hypertension, or diabetes.
- *Beta-blockers* are recommended in patients with LVEF less than 40% if no other contraindications are present.
- *Antihypertensive therapy* can maintain a blood pressure goal of less than 140/90 mm Hg.
- *Mineralocorticoid receptor antagonist* therapy is recommended in a patient with left ventricular dysfunction (LVEF less than 40%).
- *Glucose lowering* therapy in people with diabetes to achieve current blood sugar goals.

MI - Lifestyle Modifications

- *Smoking cessation* is the most cost-effective secondary measure to prevent MI. Smoking has a pro-thrombotic effect, which has a strong association with atherosclerosis and myocardial infarction.

- *Diet, alcohol, and weight control:* A diet low in saturated fat with a focus on whole grain products, vegetables, fruits, and the fish is considered cardioprotective. The target level for bodyweight is body mass index of 20 to 25 kg/m2 and waist circumference of <94 cm for the men and <80 cm for the female.


Thank you