CRONARY ARTERY DISEASE

Internal medicine

إعداد: ريان الرواشدة
مراجعة وتدقيق: محمود بركات

Ischemic Heart Disease

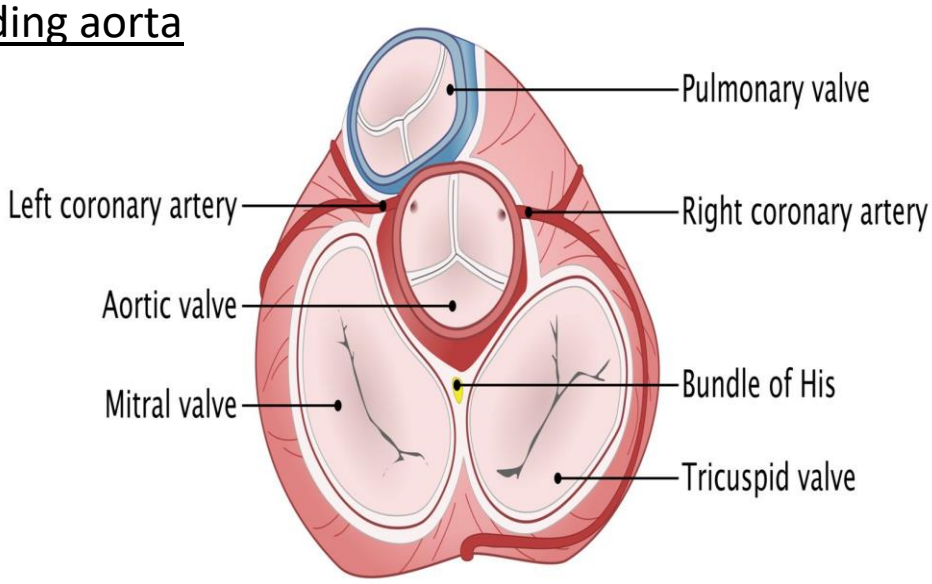
IHD or CAD (coronary artery disease): Disruption of blood flow to heart muscles by narrowing of coronary arteries (blood supply of myocardium) as a result of many disorders (most commonly atherosclerosis), therefore resulting in an imbalance between blood flow and oxygen demand which leads to a group of events (ischemia/necrosis) and ultimately the myocardium won't do its function properly.

First things first, we'll review some important anatomical and physiological concepts about coronary blood flow system.

Anatomy of coronary blood flow

The arterial supply of the heart is provided by the **right** and **left coronary arteries**, which arise from the ascending aorta, immediately above the aortic valve. The coronary arteries and their major branches are distributed over the surface of the heart.

- **The right coronary artery** arises from the right anterior aortic sinus of the ascending aorta
- **The left coronary artery** arises from the left posterior aortic sinus of the ascending aorta



❖ Main branches of each artery

1. Right coronary artery (RCA)

a. Atrial branches
b. Ventricular branches
c. Right marginal branches
d. PDA (posterior descending artery), if right dominant, 80% of people (also can be left dominant, which will arise from the lateral circumflex artery, or codominant, which arise from both RCA and LCX)
e. Nodal branch: to S.A node (60% of people) and to AV node (by the PDA)

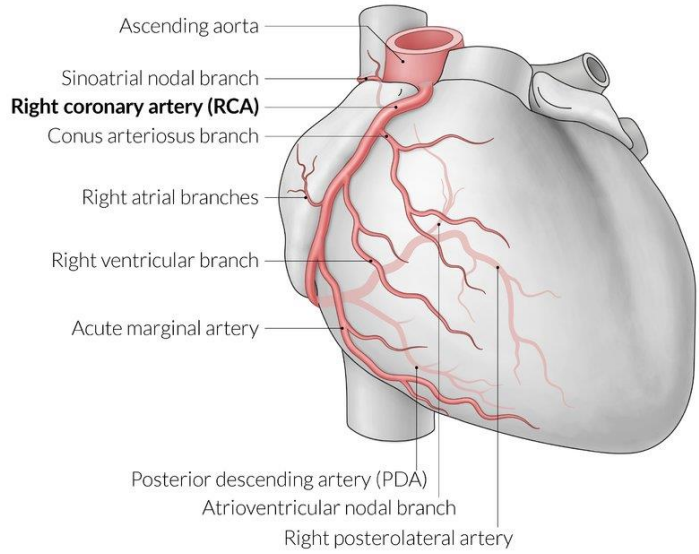
- Branches of the right coronary artery supply the sinoatrial node, the majority of the right atrium and ventricle, the atrioventricular node, approx. ⅓ of the interventricular septum, and a small part of the inferior surface of the left ventricle.
- *In general cover the right side of the heart*

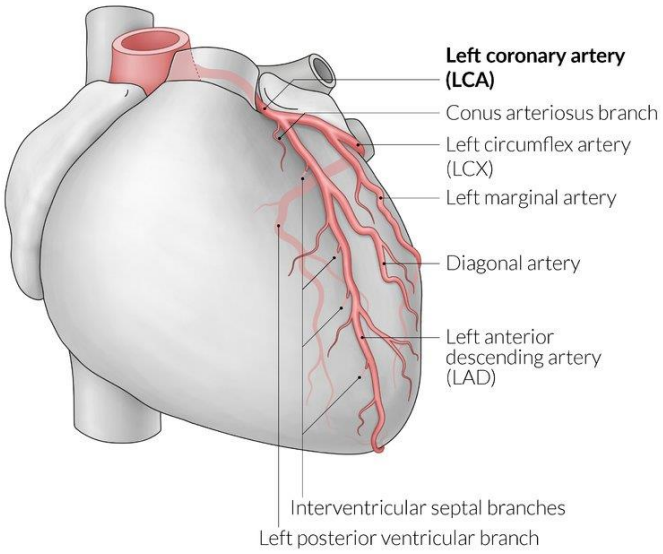
2. Left coronary artery (LCA)

a. Atrial branches
b. Left anterior Descending (LAD) to anterior aspects of right and left ventricles
c. Left circumflex artery (LCX) to posterior aspects of left atrium and ventricle
d. Nodal branches: to SA nodes in (35% of people)

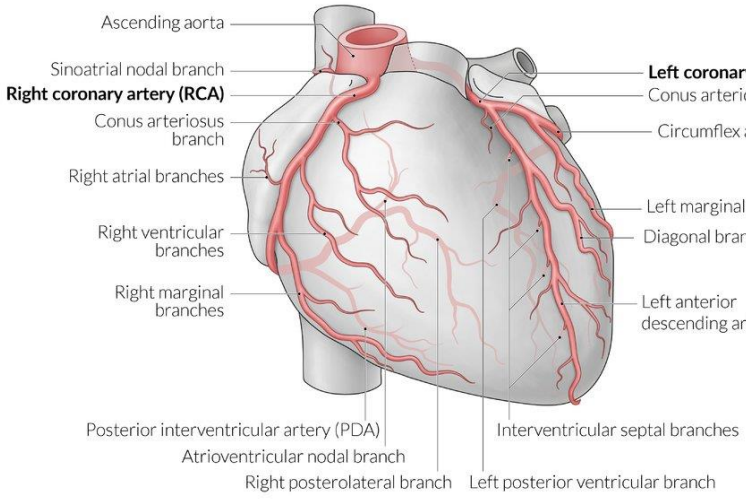
- Branches of the left coronary artery supply **the majority of the left atrium and ventricle**, the anterior aspects of both ventricles, the cardiac apex, and ⅔ of the interventricular septum.

Main branches of coronary arteries

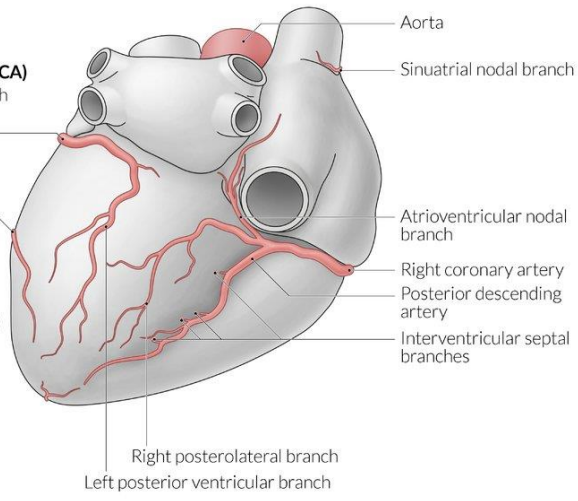




Anterior surface of the heart (ventral view)



Diaphragmatic surface of the heart (dorsocaudal view)



Important physiological concepts of coronary blood flow

- Coronary blood flow is not constant; it peaks in early diastole; sinuses are open and intramural arteries are not constricted (thus the duration of diastole is the major limiting factor for coronary blood supply to myocardium), note that this does not mean there is no blood flow during other phases, it just peaks during this time.
- During systole, blood flow decreases in the **left ventricle**'s coronary arteries; this is because of intramural coronary vessels are **perpendicular to myocardium so in systole when myocardium contracts the coronary vessels are compressed by high ventricular wall pressure and the aortic sinuses are closed.**
- Whereas in the **right ventricle** there is relatively constant blood flow to the right ventricular myocardium (**Pressure in RV is much lower than LV (~25 vs 120), thus coronary perfusion pressure is able to overcome right ventricular wall pressure throughout the cardiac cycle**)
- Coronary blood flow is regulated by many factors:
  1. **Oxygen demand**: most important factor (Increased demand means increased blood flow)
  2. **Metabolites** (CO^2, H^+, Adenosine, Lactate, K^+, mnemonic: CHALK): when increased from myocardial activity cause the coronary arteries to dilate.
     - Endothelium derived vasodilator substance (EDVS) is increased in atherosclerotic patients, it causes the coronary vessels to dilate more and more.
  3. **Heart rate**, when increased that means the heart is pumping fast, the duration of systole and diastole is decreased so the diastolic filling time is decreased, when decreased the blood flow to coronary vessels is decreased also. Generally, when sympathetic NS is activated, β_2 receptors cause vasodilation and β_1 receptors increase H.R
  4. **Aortic driving pressure** that's increased when there's stenosis in coronary arteries.

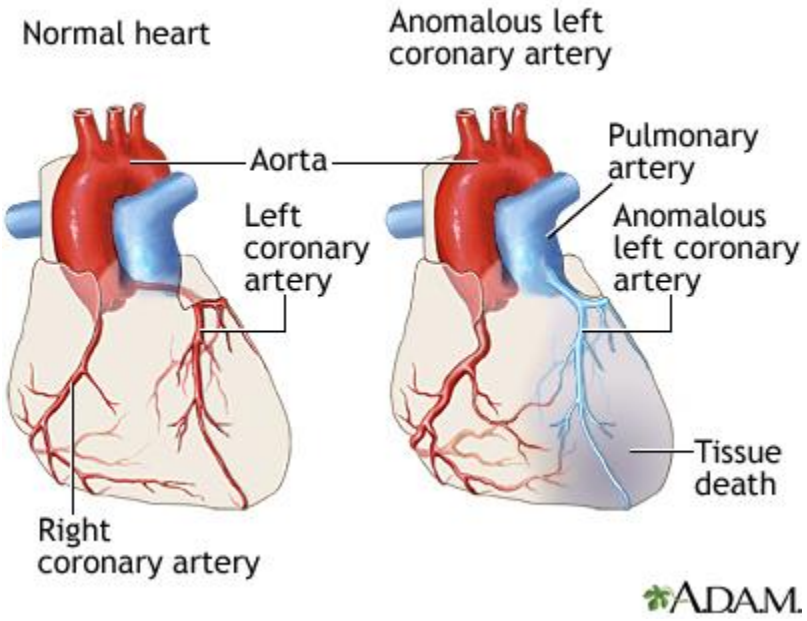
Types of CAD

Let's say that there's two types of Ischemic disease that occurs to myocardium one is congenital, and the other is acquired.

1- **Congenital type**

a- Anomalous origin from pulmonary artery **(Bland white garland syndrome)**:

- A rare congenital cardiac anomaly in which the left coronary artery arises abnormally from the pulmonary artery.
- Infants with this condition typically present with pallor, irritability, and diaphoresis after feeding.
- Can progress to congestive heart failure.
- Require immediate surgical reimplantation of the left coronary artery to the aorta



b- Anomalous origin from other coronary arteries:

- As like RCA and LCA originate from same sinus which is right aortic sinus and **split right coronary artery (RCA)** is a congenital anomaly of the coronary arteries where either two separate arteries arise from the aortic trunk or RCA bifurcates into two major arteries immediately after its origin from the right sinus, A split right coronary artery is considered benign and not associated with any symptoms and can be incidentally found on imaging studies

c- Myocardial bridging

- Coronary artery goes through myocardium instead of lying on its surface
- It may be asymptomatic, but if symptomatic it causes angina symptoms

Notes

- Coronary arteries anomalies have a variety of presentation ranging from asymptomatic to mimicking angina
- Some patients may stay asymptomatic until sudden cardiac death
- the congenital type of CAD is rare and is not common

2- **Acquired type**

- Most common cases of CAD are acquired, and most commonly due to atherosclerosis
- The acquired type of CAD is classified into atherosclerotic and non-atherosclerotic causes

a- Non atherosclerotic

Some conditions that are not related to atherosclerosis can lead to coronary ischemia:

1. **embolus**:
    a. From atrial fibrillation or endocarditis
    b. Or post cardiac valve replacement
    - Embolus that formed in these situations can also cause ischemia anywhere in body like brain(stroke), lung, gut and coronary vessels as well could be affected
2. **Drug induced** ex. Cocaine, methamphetamine can cause angina and MI
3. **Vasculitis**: as Kawasaki disease causes inflammation of the coronary arteries and can cause damage to vessels which disrupt blood flow
4. **Aortic dissection**: as we know that coronary arteries are originate from ascending aorta, so when A.A dissect, it may continue to coronary arteries and dissect also
5. **Iatrogenic**: in invasive procedures like angiography or catheterization may cause injury to coronary vessels
6. **Severe anemia**: it causes to reduce oxygen that's delivered to tissues and may cause ischemia
7. **Thyrotoxicosis**: it causes high increase in B.P and H.R and increased myocardial oxygen needs especially in exertion.

- Generally anything that may cause decreased blood flow or decrease oxygen delivery or increase oxygen demand, it may cause non atherosclerotic CAD.

b- Atherosclerotic type:

- It's caused by atherosclerosis (the most common cause of CAD)
- Atherosclerosis: arterial wall thickening (hardening) and elasticity loss caused by a buildup of fatty plaque in the intima (inner layer of vessel)
- Causes and risk factors:
  a. Smoking
  b. Diabetes mellitus. **worst risk factor**.
  c. Age: males ≥ 45 years, females ≥ 55 years (post menopause)
  d. Arterial hypertension. **most common risk factor**.
  e. Dyslipidemia (increased LDL, Decreased HDL)
  f. High homocysteine levels (homocystinuria)
  g. Obesity
  h. Physical inactivity
  i. Increased alcohol consumption
  j. Family history: cardiovascular events in first-degree relatives below the age of 55 (male)/65 (female); **if it not a first-degree relative or a first-degree but age above 55 in males and 65 in females it not considered a risk factor**
  k. Age: males ≥ 45 years, females ≥ 55 years (post menopause)
  l. Chronic Kidney Disease

Pathogenesis of atherosclerosis and plaque formation:

1. **Chronic stress** on the endothelium
2. Endothelial dysfunction, (endothelial injury) which leads to
    a. Invasion of **inflammatory cells mainly monocytes** through the disrupted endothelial barrier
    b. Adhesion of **platelets to the damaged vessel wall** → platelets release inflammatory mediators (e.g., cytokines) and platelet-derived growth factor (PDGF)
    c. PDGF stimulates migration and proliferation of smooth muscle cells (SMC) in the tunica intima
3. Inflammation of the vessel wall
4. Macrophages and SMCs (smooth muscle cells) ingest cholesterol from oxidized LDL and transform into **foam cells**.
5. Foam cells accumulate to form fatty streaks (early atherosclerotic lesions).
6. Lipid-laden macrophage (foam cells) and SMCs produce extracellular matrix (e.g., collagen) → development of a fibrous plaque (atheroma)

- Which result in narrowing of arteries and decrease blood supply
- Severity of ischemia is affected by the degree of arterial lumen occlusion by formed atheroma.

- *هذه العملية بتضل مستمرة وبتراكم دهون أكثر مع مرور الوقت و(atheroma) بتضلها تكبر وتسبب انسداد أكثر.*

7. Inflammatory cells in the atheroma (e.g., macrophages) secrete matrix metalloproteinases → weakening of the fibrous cap of the plaque due to the breakdown of extracellular matrix → minor stress ruptures the fibrous cap
8. Calcification of the intima
9. Plaque rupture → exposure of thrombogenic material (e.g., collagen) → thrombus formation with vascular occlusion or spreading of thrombogenic material

نقطه رقم 6 7 بتحكي انه ممكن يصير في (rupture of fibrous cap) وبصير ال(Collagen) ال تكون من ال(foam cells) معرض لل(platelets) وبالتالي بصير في (thrombus) يسبب في (increase occlusion of vascular wall and increase severity of ischemia) وطبعا لا تنسوا الصور الموجودة في المحاضرة على هذه العملية

What arteries are most commonly affected by atherosclerosis?

- Lower abdominal aorta > coronary > popliteal > carotid artery > circle of Willis

Thus, atherosclerosis affects mainly:

1. **Coronary artery**
2. Brain
3. Arteries of lower limb

- Remember to examine these areas in all atherosclerotic patients

Complications of atherosclerosis:

1. Ischemia due to occlusive effect of atheroma narrowing of artery
2. Thrombosis or thromboembolism
3. Weakening of vessel wall: arterial aneurysm or dissection
4. Renovascular hypertension: atherosclerosis of the renal artery → activation of the renin-angiotensin-aldosterone system leads to water an Na retention which leads to Hypertension

---

Clinical Presentation / Diagnosis and management of CAD

Patients with CAD have following clinical presentation:

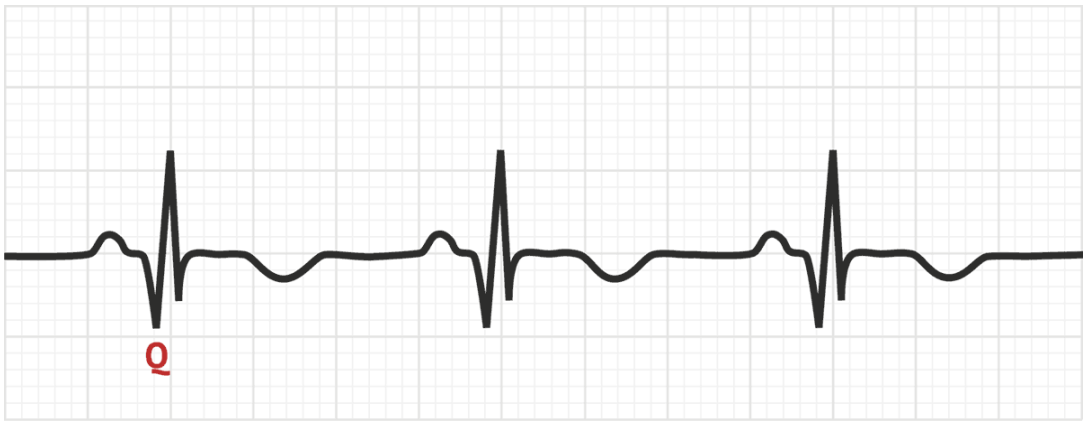
Asymptomatic → Stable Angina → Unstable Angina → Myocardial Infarction → Sudden cardiac death

Notes:

- Symptoms appear when there's imbalance between B.F and O_2 demand
- The imbalance is either due to increased O_2 demand or decreased B.F.
- All the causes of CAD result in the same symptoms, for sake of simplicity we will focus on atherosclerosis as it is the most common cause of CAD

Stable Angina

- Atherosclerotic lesion still Asymptomatic until complications occur especially ischemia (narrowing of arteries) and thrombus formation
- Symptoms begins when **70%** of Coronary Artery are occluded by atheroma or at percent of occlusion which imbalance between blood supply and oxygen demand occur.
- In case of stable angina symptoms begins <u>when there's increased O2 demand by myocardium</u> due to exertion or increased work on heart.
- Physiologically the blood flow must be increased but because of presence of stenosis by atheroma BF will not be increased well
- **Symptoms of Stable Angina**: "**Typical** chest pain"
  1- Chest pain (retrosternal or substernal)
  2- Character: heaviness or pressure
  3- Gradual onset
  4- Lasts less than 10 or 15 minutes
  5- Exaggerated with exertion or stress or some drugs like cocaine
  6- Relieved with rest or nitrates
  7- Pain <u>doesn't</u> change with **breathing** or **body position** and <u>there's not</u> **chest tenderness**.
- **Findings of stable angina**:
  1. **<u>Rest</u> ECG**: Normal or presence of Q-waves that indicate that there's previous MI.



2. **Biomarkers**: negative
3. **CBC**: for probable anemia.
4. **Hemodynamics for B.P**: to rule out hypertension
5. **Sugar level**: for probable diabetes
6. **Lipid and cholesterol (LDL, HDL) levels**: for probable hyperlipidemia
7. **Stress test, pharmacologic stress test**, and **coronary angiography:**

Stress test

- A noninvasive diagnostic procedure that uses treadmill bicycle exercise, blood pressure monitoring, and electrocardiography (ECG) and Echocardiography to evaluate the heart response to exercise.
- **For stress ECG**: ischemia is detected by the presence of ST-depression. (ST depression means sub-endocardial ischemia)

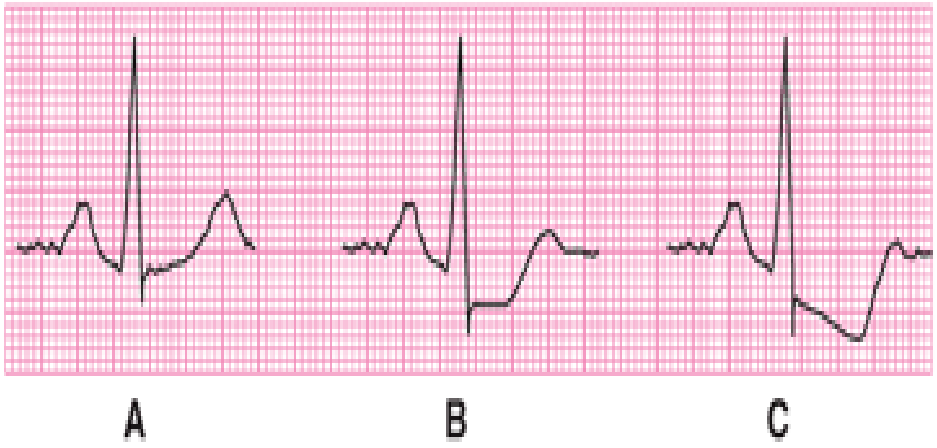


FIGURE 10.3. Single ECG waveforms at rest (A) and two variations of the abnormal condition of exercise-induced subendocardial ischemia (B and C).

- **For stress Echo**: Exercise-induced ischemia is evidenced by **wall motion abnormalities (e.g., akinesis or hypokinesis**) not present at rest. This study is less reliable in patients with existing wall motion abnormalities or lowered EF. Stress Echo is indicated if heart murmur is present; to diagnose Valvular heart diseases

- **Note**: Hypotension and ventricular arrhythmias may be seen in stress test in patients with CAD.
- Information gained from a stress test can be enhanced by **stress myocardial perfusion imaging** after IV administration of a radioisotope.
  - **Concept**: Viable myocardial cells extract the radioisotope from the blood. No radioisotope uptake means no blood flow to an area of the myocardium.
  - Helpful in detecting the Affected region and to determine if ischemia is reversible or not; if the perfusion improve with rest after the exercise, then the ischemia is reversible otherwise the tissue is infarcted

---

Pharmacological stress test

- If the patient cannot exercise, perform a pharmacologic stress test.
- Pharmacological stress test is a stress test in which a drug such as dobutamine or another chronotropic or vasodilating drug is used to increase the heart rate during assessments of myocardial perfusion or function, thus help us to detect ischemia on ECG, Echo, and nuclear detecting scan

A. **IV adenosine and dipyridamole**
   - Cause generalized coronary vasodilation.
   - **Concept**: Since diseased coronary arteries are already maximally dilated at rest to increase blood flow, they receive relatively less blood flow when the entire coronary system is pharmacologically vasodilated. (Coronary steal phenomenon)
   - **Note**: Drugs that cause Coronary steal phenomenon are contraindicated in airway disease

B. **Dobutamine**

- Increases myocardial oxygen demand by increasing heart rate, blood pressure, and cardiac contractility. So, we can detect ischemia.
- Can cause arrythmia as side effect

Note: negative stress test doesn't exclude MI or Angina risk in future because stress test is performed to detect if there's flow-limiting stenosis or not. That is small atheroma that are not large enough to obstruct the artery to a level enough to show any symptoms (asymptomatic atheroma)

Coronary angiography

- Definitive and golden test for CAD.
- Contrast is injected into coronary vessels to visualize any stenotic lesions**.**
- Defines the location and extent of coronary disease.
- Although it is considered gold standard it is rarely used as diagnostic test, commonly used when preforming cardia catheterization

---

Management

- Before deciding management plan, we should assess the prognosis of the patient.
- **Bad prognosis indicators:**
  1. 2 or more vessels are occluded
  2. Ejection fraction less than 50%.
     (EF = Stroke volume / End Diastolic volume)
  3. Occlusion of left main coronary artery
  4. Severity of symptoms (degree of occlusion).
  5. Associated with Dyspnea
  6. Prior MI or known CAD

As constant rule of management of stable angina patients:

1. **Modification of risk factors**
   A. Smoking cessation
   B. HTN control
   C. Sugar control in diabetes patients
   D. Hyperlipidemia: reduction in serum cholesterol with lifestyle modifications and HMG-CoA reductase inhibitors (statins) reduce CAD risk.
   E. Weight loss, exercise, low- cholesterol diet
2. **Medical therapy**
   A. Aspirin (low-dose 325 mg/day)
      - Lipid-lowering therapy (HMG-CoA reductase inhibitors (statins) or PCSK9 inhibitors): Lower LDL level
      - These drugs and risk factor modification are constant and continues interventions in any patient with stable angina because they improve mortality and morbidity in long-term life.

   B. β-Blockers (Metoprolol, Atenolol):
      - Block sympathetic stimulation of the heart, Reduce HR, BP, and contractility, thereby decreasing cardiac work (i.e., **β- blockers lower myocardial oxygen consumption**)

   C. Nitrates (nitroglycerine):
      - Cause coronary vasodilation (improve blood flow) and systemic venodilation (pooling blood in veins so reduce preload and myocardial oxygen demand)

   D. Calcium channel blockers (Diltiazem, Verapamil):
      - Add to antianginal therapy when β-blockers and nitrates are not fully effective, cause coronary vasodilation (increased blood flow) and afterload reduction, reduce contractility and O2 demand.

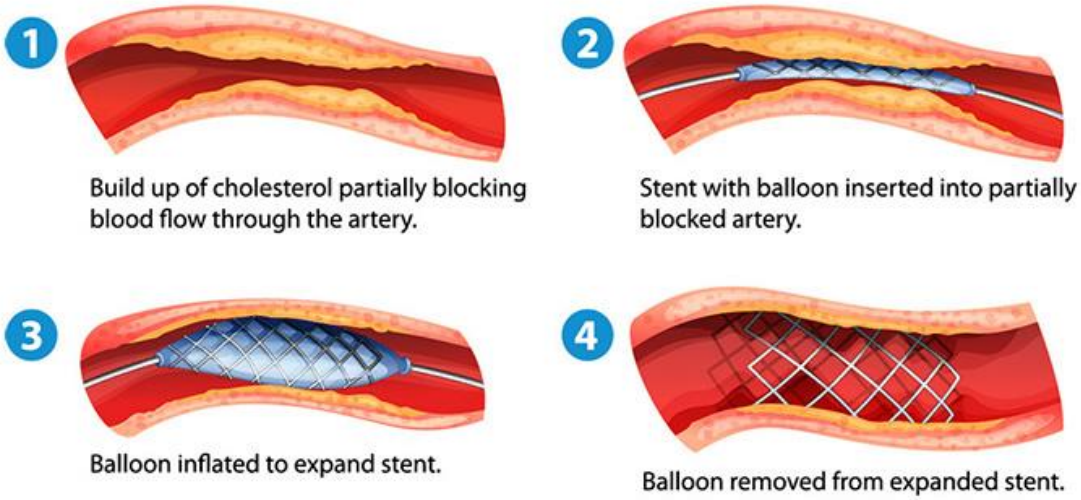
- Notes:
  - β-blockers, nitrates, and CCBs are used to relieve and improve symptoms of angina (symptom control) when appear, whereas aspirin and statin are prophylactic.
  - β-blockers and CCBs combination is contraindicated; can result in heart block
  - β-blockers are contraindicated in some cases:
    a. Cardiogenic shock and hypotension
    b. Decompensated heart failure
    c. Asthma and COPD
  - CCBs are only used in cases of stable angina and Prinz-metal (variant) angina. and are contraindicated in severe reduced ejection fraction.

3. Surgical or invasive therapy (revascularization)

- According to studies both medical and surgical therapies yield approximately the same result in improving life and decreasing morbidity in treating CAD.
- That's why surgical therapy is ONLY indicated in case of failure of other therapies, absolute contraindications of the medications or if the patient has bad prognostic indicator.
- The two methods of surgical therapy are **PCI** and **CABG**

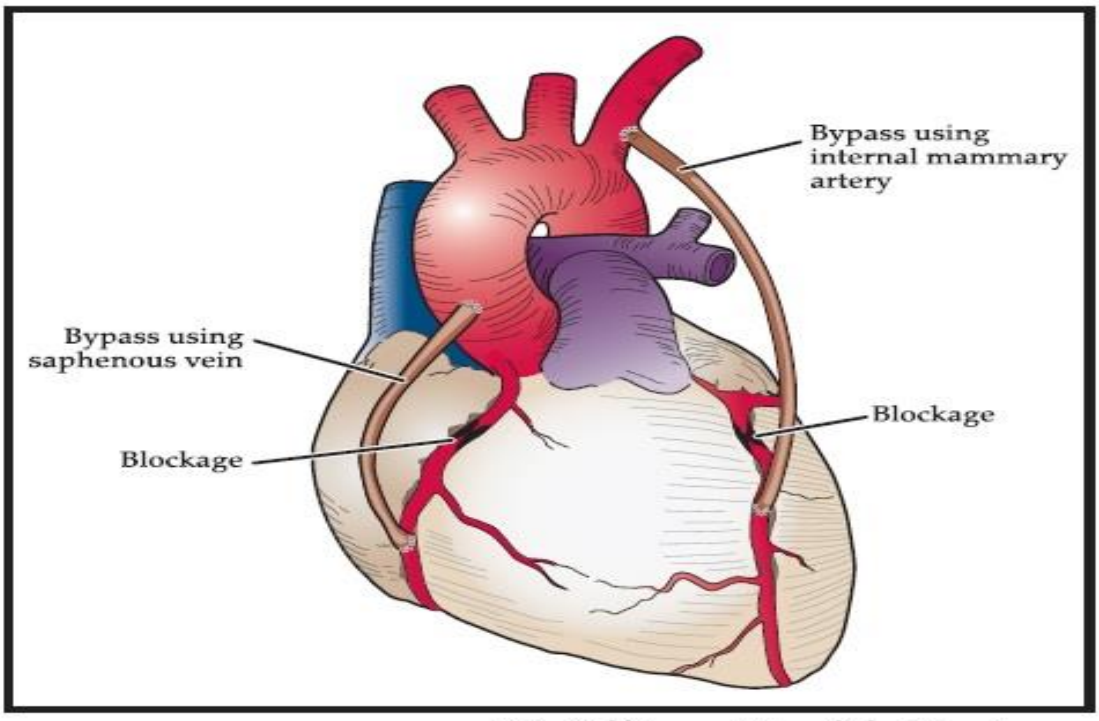
A. PCI (Percutaneous Coronary Intervention):

- Consists of both coronary angioplasty with a balloon and stenting.
- The stent which left in coronary artery may cause complications:
  a. <u>Restenosis:</u> caused by growth of scar tissue on stent so it causes re-occlusion, this problem is reduced by using drug-eluting stents
  b. <u>Thrombosis:</u> thrombus formation on stents prevented by dual anti-platelet therapy



B. CABG (Coronary Artery Bypass Grafting):

- Taking graft from left internal mammary artery or saphenous vein and bypass the occluded artery.



Prinzmetal angina

- Also known as variant angina and vasospastic angina
- Due to transient coronary vasospasm that usually is accompanied by a fixed atherosclerotic lesion or in normal coronary arteries**.**
- **Clinical presentation**: like stable angina but symptoms occur at rest and timing of episodes from midnight to early morning, symptoms relieved by exertion
- Associated with smoking
- **ECG finding**: transient ST elevation
- Stress test is not effective in this type of angina, diagnosed by angiography
- Treatment:
  1. Avoid smoking
  2. Vasodilators: Nitrates
  3. CCBs can also be used
- **Note:** β-blockers are C/I in Prinzmetal angina: because of vasoconstrictive effect of this drug

---

Remember: Untreated atheroma can result in 1 of 2 outcomes:

1. Keep growing causing more and more narrowing over the time (i.e., Chronic angina)
2. Rupture and the collagen inside get exposed to platelets which leads to thrombus formation and rapid narrowing and occlusion of artery (i.e., Acute coronary syndrome “ACS”)

Acute coronary syndrome “ACS”

- **ACS:** The clinical manifestations of atherosclerotic plaque rupture and coronary occlusion.
- Term generally refers to:
  1. Unstable angina, NSTEMI
  2. STEMI (myocardial infarction)

Unstable Angina & NSTEMI

- **Unstable angina/NSTEMI (Non-elevating ST element myocardial infarction):** subtotal occlusion 90% of artery caused by thrombus formation or enlarged atheroma, so the blood flow is severely decreased when O2 demand is unchanged, so the main cause of imbalance and ischemia here is decreased blood flow more than O2 demand.
- The difference between unstable angina and NSTEMI that in NSTEMI the cardiac markers (CPK and Troponin) are positive indicating subendocardial ischemia but for sake of simplicity we will discuss both together.
- **The symptoms** of unstable angina differ from stable angina in the following features:
  1. Symptoms occurs at rest (whereas stable angina at exertion only)
  2. Increasing frequency, duration and intensity of chest pain
- **Findings of unstable angina/NSTEMI:**
  1. **Resting ECG:** ST-depression and inverted T waves
  2. **Biomarkers**: negative at unstable angina, positive at NSTEMI
  3. **Stress testing** is not preferred in Acute coronary syndrome patients.
  4. **Echocardiography** is used when the diagnosis is not clear

- **Notes**:
  - Biomarkers: enzymes that increased when there's myocardial necrosis by ischemia (infarction), the most important enzymes for myocardial infarction:
    a. Troponin T or I (Troponin I is more important): elevated in 2-4 hours and returns to normal in 2 weeks.
    b. CK-MB: elevated 4-6 hours and returns to normal in 2-3 days, so it's useful in detecting reinfarction.

Management

- Unstable angina patients they must be admitted to hospital.
- **Acute management:**
  1. Supportive therapy:
     a. nitrates for pain or morphine but it's controversial because it's masking worsening symptoms.
     b. Oxygen if patient hypoxic.
  2. Anti-platelet agents:
     a. Aspirin (325mg/day)
     b. P2Y12 inhibitor (clopidogrel, ticagrelor)
     c. Abciximab
  3. β-blockers if there's no contraindications
  4. Low-molecular-weight heparin (LMWH)
  5. Cardiac catheterization (Revascularization): PCI or CABG.
     Cardiac catheterization is non-emergent procedure we can do it after 1 or 2 days of admission. Almost all patients improve by medical therapy.

TIMI score

- Estimates the risk of mortality, new or recurrent myocardial infarction, or the need for urgent revascularization in patients with NSTE-ACS
- Can help determine the therapeutic regimen and timing for revascularization

TIMI score for NSTE-ACS [16]	
Characteristics	**Points**
Age ≥ 65 years	1
≥ 3 CAD risk factors (e.g., family history of CAD, DM, smoking, HTN, hypercholesterolemia)	1
Known CAD (stenosis > 50%)	1
≥ 2 episodes of severe angina in the last 24 hours	1
ASA use in the past 7 days	1
ST deviation (≥ 0.5 mm)	1
Elevated cardiac biomarkers	1
Interpretation • Score of 0–1 (low-risk): favors an ischemia-guided strategy [15] • Score ≥ 2 (non-low-risk): favors an invasive strategy	

- Angiography still the golden test for CAD diagnosis.
- **ST-depression** means sub-endocardial ischemia (seen in U.A/NSTEMI and stable angina under stress) it's seen also in non-ischemic cases like hypokalemia and aortic stenosis.
- **ST-elevation** means Transmural ischemia (seen in MI and prinzmetal angina) also seen in non-ischemic cases like pericarditis and left bundle branch block.

- **Long term management**:
  1. Risk factor modification
  2. Aspirin or other anti-platelet therapy.
  3. B blockers
  4. Nitrates
  5. Statin
  6. Anti-arrhythmic drugs (in case of arrhythmia)

MI(STEMI)

- It's part of ACS and manifested by Total occlusion of coronary artery which caused mainly by thrombus formation on ruptured atheroma.
- **Clinical features**:
  1. Chest pain
     a. substernal diffuse pressure sensation
     b. Radiation to neck, jaw, arms, or back, commonly to the left side
     c. More severe than angina
     d. Lasts longer than angina (more than 20 mins to 30 mins)
     e. Not relieved by rest or nitrates.
  2. GI symptoms may present (N/V and epigastric pain)
  3. Dyspnea
  4. Diaphoresis
  5. Weakness, dizziness, fatigue
  6. Pulse rate may be normal, but often bradycardia is present in inferior infarctions. Tachycardia is often seen with large infarctions.
  7. Blood pressure is often elevated.
  8. Cardiac exam is usually normal

- **Findings**:
  1. ECG:
     a. ST-segment elevation
     b. peaked T-wave: very early
     c. pathologic Q waves: seen later on ECG
     2. Cardiac enzymes: positive
  - The most common coronary artery occluded is LAD. (**Widow-maker**)
    (The widow-maker is a massive heart attack that occurs when the left anterior descending artery (LAD) is totally or almost completely blocked)

- **Types of MI:**
  - There are types of MI according to which vessel is occluded and it's important to know these types to know the special complications of each type and appropriate management.
  - We can detect these types by ECG leads

1- Anterior MI

- **ST elevation and Q waves (late)** in *V1-V4* leads
- **Artery affected**: LAD
- If large it causes Cardiogenic shock and Heart failure because of large necrosis of anterior wall left ventricle.
- **Associated with** formation of ventricular aneurysm.

2- Lateral MI

- **ST elevation and Q waves (late)** in lead ***I, aVL***, *V4, V5,* and *V6*
- **Artery Affected**: Left anterior descending or circumflex artery.

3- Posterior MI

- **ST elevation** in (*V7-V9*) with *V1-V4* **ST depressions**
- **Artery affected**: Posterior descending artery
- **Usually associated with** lateral or inferior MI

4- Inferior MI

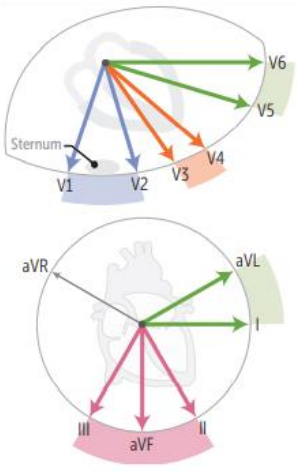
- **ST elevation and Q waves (late)** in leads *II, III* and *aVF*
- **Artery affected**: Right coronary artery
- It causes Rt. ventricular infarction, so **Rt. Heart failure** is seen with elevated jugular venous pressure.
  - Nitrates are C/I in inferior MI, because it decreases preload and make the case worse and worse.
- Inferior MI causes also **sinus bradycardia** and heart block because SA, AV nodes may be infarcted in Inferior MI.
- So, if bradycardia or heart block is present, B blockers are C/I because it blocks nodes and make the case worse and worse.
- **Associated with** formation of ventricular pseudoaneurysm.

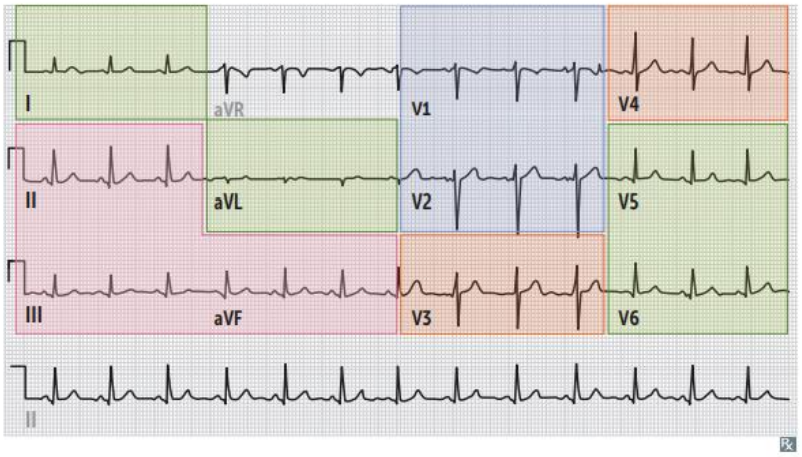
5- Left main artery occlusion

- ST elevation only in *aVR* lead.

ECG localization of STEMI

INFARCT LOCATION	LEADS WITH ST-SEGMENT ELEVATIONS OR Q WAVES
Anteroseptal (LAD)	V_1–V_2
Anteroapical (distal LAD)	V_3–V_4
Anterolateral (LAD or LCX)	V_5–V_6
Lateral (LCX)	I, aVL
InFerior (RCA)	II, III, aVF
Posterior (PDA)	V_7–V_9, ST depression in V_1–V_3 with tall R waves





- Complications of MI:

1) 0-24 hours post –MI:

a) **Arrhythmia**:

- **Ventricular arrhythmias**
  - If patient is hemodynamically unstable, electrical cardioversion is indicated.
  - If patient is hemodynamically stable, start antiarrhythmic therapy)
- **AV block**
  - According to type of block if first and second'type1' therapy is not required if second'type2' IV Atropine and pacemaker
- **Sinus bradycardia**
- **Asystole**
- **Atrial fibrillation**

b) **Sudden Cardiac Death**:

- Death result from **Fatal ventricular arrhythmia** is considered to be the underlying mechanism of SCD.
- *Most common cause of death in MI patient in first days*

c) **Pump failure**:

- *Most common cause of death in-hospital*
- it's caused by tissue necrosis and decrease contractility of myocardium so it's not able to maintain C.O and pump blood to systemic circulation. So, **CHF** and **cardiogenic shock** occur.
- A fourth heart sound (S4) is common due to a stiffened Ventricle
- The second heart sound may be **paradoxically split** as the left ventricular contraction time increases due to LBBB and weakened left ventricle

2) 1-3 days post-MI:

- **Fibrinous pericarditis**: caused by extension of inflammation in necrotic tissue (to form scar tissue) that's near to pericardium, Clinical features of acute pericarditis: *pleuritic chest pain*, *dry cough*, *friction rub*, *diffuse ST elevations on ECG*.
- Never ever give NSAIDS or cortisol in this type of pericarditis because they inhibit the scar formation.
- After scar tissue formation the fibrinous pericarditis disappear.

3) 3-14 days post MI:

a) **Free wall rupture**: leads to cardiac tamponade and it's fatal.

b) **Rupture of interventricular septum**: leads to Rt. HF

c) **Papillary muscle rupture**: leads to mitral regurgitation. more common with inferior MI.

d) **Ventricular pseudo-aneurysm**: it's a myocardial rupture contained with peri cardium and scar tissue it tends to become a free wall rupture.

4) 2 weeks – months post MI:

a) **Ventricular aneurysm**:

- persistent ST elevation and thrombus formation(embolism) and it's associated with ventricular tachyarrhythmias, Rupture → cardiac tamponade
- Systolic murmur, S3 and/or S4 are heard in aneurysm.

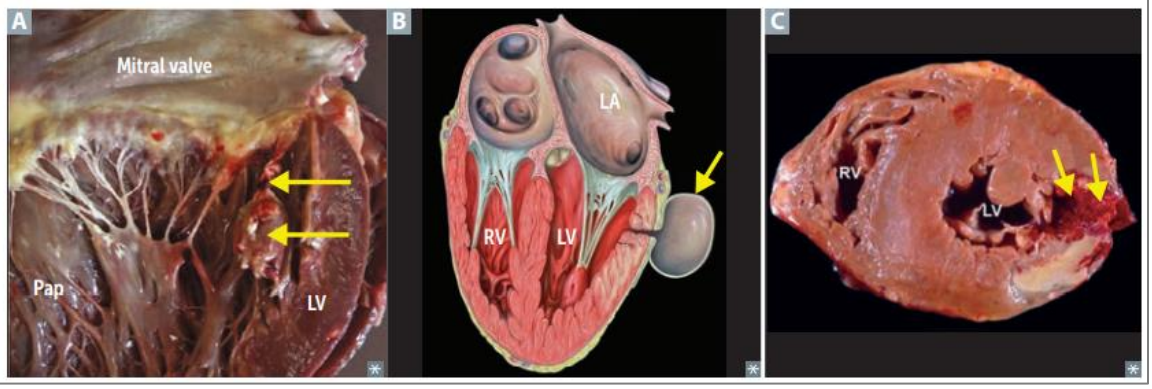
b) **Dressler's syndrome (post myocardial infarction):**

- Immunologically based syndrome consisting of fever, malaise, pericarditis, leukocytosis, and pleuritis, occurring weeks to months after an MI.
- *We can use NSAIDS and corticosteroids here because the problem is immunologic*. *(Vs Fibrinous pericarditis)*

c) **CHF**: after any ischemic event, the probability of ischemic dilated cardiomyopathy increases. Ischemic dilated cardiomyopathy is considered as a common cause of systolic heart failure.

- Recurrent Re-infarction is probable scenario that come from either extension of infarction or re-infarction in new area, can be detected by re elevation of CK-MB

Myocardial infarction complications	
Cardiac arrhythmia	Occurs within the first few days after MI. Important cause of death before reaching the hospital and within the first 24 hours post-MI.
Postinfarction fibrinous pericarditis	1–3 days: friction rub.
Papillary muscle rupture	2–7 days: posteromedial papillary muscle rupture A ↑ risk due to single blood supply from posterior descending artery. Can result in severe mitral regurgitation.
Interventricular septal rupture	3–5 days: macrophage-mediated degradation → VSD → ↑ O_2 saturation and pressure in RV.
Ventricular pseudoaneurysm formation	3–14 days: free wall rupture contained by adherent pericardium or scar tissue B; ↓ CO, risk of arrhythmia, embolus from mural thrombus.
Ventricular free wall rupture	5–14 days: free wall rupture C → cardiac tamponade. LV hypertrophy and previous MI protect against free wall rupture. Acute form usually leads to sudden death.
True ventricular aneurysm	2 weeks to several months: outward bulge with contraction ("dyskinesia"), associated with fibrosis.
Dressler syndrome	Several weeks: autoimmune phenomenon resulting in fibrinous pericarditis.
LV failure and pulmonary edema	Can occur 2° to LV infarction, VSD, free wall rupture, papillary muscle rupture with mitral regurgitation.

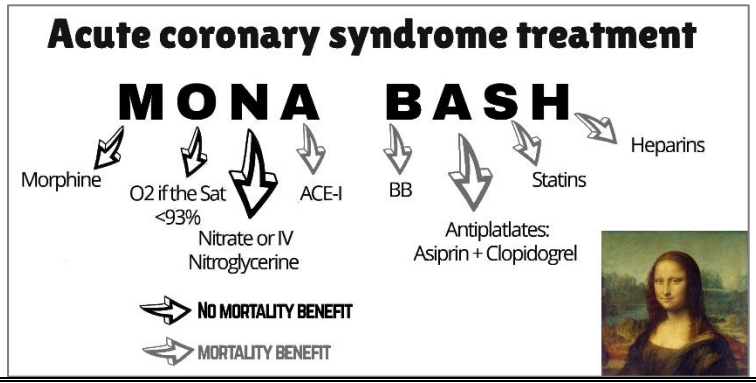


- **Management of MI:**
  - MI is emergent life-threatening condition that required rapid intervention.
  - The main goal is to open the occluded artery

Management of MI:

1) Hospital Admission to CCU
2) Preparation for invasive therapy (surgery)
   - If patient administered in first 12 hours
     - **Option 1**: PCI within 90 mins counted from time of arrival must be done.
     - If PCI is not available move to **option 2**: thrombolytic therapy (tPA, streptokinase, alteplase) within 30 mins counted from time of arrival. "if not C/I"
   - After 12 h we do CABG.
3) While preparing for PCI:
   a. Oxygen for hypoxia
   b. Morphine / Nitrates for pain (C/I in inferior MI)
   c. **Aspirin** (part of long – term therapy)
   d. P2Y12 inhibitors "antiplatelet therapy as aspirin" as clopidogrel and ticagrelor.
   e. **β-blockers** (not used if sinus bradycardia existed, if there's no C/Is, can be considered as long-term therapy)
   f. **ACE inhibitors** (part of long-term therapy)
   g. **Statins** (long term therapy)
   h. Heparin (LMWH)
4) Risk factors modification + long term medical therapy as a long-maintenance therapy + cardiac rehabilitation.

   (Cardiac rehabilitation: A program of exercise and education that is designed to improve health and recover from a heart attack)



Differential diagnosis of chest pain

- The chief complain of CAD patients is **Chest pain,** but chest pain is also indicator of other diseases. So, we must keep in mind the differential Dx of chest pain for making an oriented Dx.
- Overall, the chest pain **history, ECG, CXR, cardiac biomarkers is more useful than the physical examination**.
- Important aspects of the history include duration, quality, location, radiation, frequency, alleviating or precipitating factors (especially exercise), and associated symptoms.

DDx:

1. **Stable angina**
2. **ACS (UA, NSTEMI, MI)**: it's difficult to differentiate them based on history and clinical features alone, so we need ECG and cardiac biomarkers.
   Acute coronary syndromes often present without "classic" symptoms: instead, they may have **dyspnea, shortness of breath**.
3. **Pericarditis**: stabbing and sharp/relieved by leaning forward and NSAIDS / increasing by lying down / Pericardial rub on cardiac exam.
4. **Myocarditis**: vague and mild pain
5. **Aortic dissection**: The pain is sudden sharp, tearing, and extremely severe. It typically radiates to back, prolonged time with no relieving factors.
6. **Pulmonary embolism**: Dyspnea, tachycardia, pain is usually pleuritic (increasing with inhaling and exhaling)
7. **Pleuritis**: Pain is sharp and increases on inspiration, and usually there's evidence of infection.

8. **Pneumonia**: especially if fever is present.
9. **Gastroesophageal reflux disease (GERD)**: made worse with recumbency or after meals, may be associated with regurgitation (heart burn) and relieved by antacids.
10. Pneumothorax: sudden Onset abrupt with sharp pleuritic chest pain and dyspnea; breath sounds absent; chest x-ray confirms.
11. Esophageal spasm: episodes of spasm may be brought on by cold liquids, relieved by nitroglycerin, and may closely resemble angina or infarction; diagnosis may be confirmed by upper endoscopy or esophageal manometry.
    Esophageal causes usually associated with dysphagia.
12. **Musculoskeletal disorders (Costochondritis, muscle strain, rib fracture)**: suspected when chest wall **tenderness** present when palpated.
13. **Psychologic (Anxiety)**: this is Dx of exclusion of other causes of chest pain.

Notes on physical exam in chest pain:

1) If **tenderness** is present, it suggests Musculoskeletal problem.
2) **Vital signs**:
   a) *Hypotension* may suggest cardiogenic shock after MI
   b) *Tachycardia* and *tachypnea* suggest pulmonary embolism
   c) Check BP in both arms: *a difference >20 mm Hg systolic* suggests aortic dissection
   3) **Fever:** Fever may suggest pneumonia or mediastinitis (esophageal rupture) as the cause of chest pain.
4) Check heart sounds

5) Check if new murmurs are present or not,
   a) **Aortic regurgitation** occurs in over half of patients with aortic dissection
b) **Mitral regurgitation** can occur in patients with angina or infarction and is due to papillary muscle dysfunction.
6) Examine RS, GI to exclude their causes.
7) Examine Lower limb:
   - The extremities should be examined for pulses, edema, and signs of atherosclerotic vessel disease.
   - *Absence of pedal pulses* may occur in aortic dissection.
   - *Calf swelling* or *edema* raises the odds of pulmonary embolism as the cause of chest pain.

Tests to order in chest pain patients

1- ECG
2- CXR
3- Cardiac biomarkers
4- Echocardiography
5- Chest CT