

## Community:

- CVD is the most leading cause of death (mortality) in developed countries; about one third of all deaths (50% of which are coronary deaths).
- CVD accounts for nearly 30% of all disability cases (morbidity).
- CVD in:
  - different age groups? 30-44 years
  - men and women? Middle aged men
  - different ethnic groups? African-American more than White
  - different geographical places? Using the age distribution of a Hypothetical European Standard Population
- Modifiable (Conventional) CVD risk factors:
  - Obesity
  - High BP
  - DM
  - Tobacco use (passive smoking has additional risk)
  - Physical inactivity
  - Unhealthy diet
  - Abnormal blood lipids
  - Depression and CVD - bidirectional link
- US Guidelines:
  - Total cholesterol <240 mg/dl (6.2 mmol/l) { $\geq 240 \rightarrow$  HIGH}
  - LDL <160 mg/dl (3.8 mmol/l) {160-189  $\rightarrow$  HIGH}
  - HDL  $\geq 40$  mg/dl (1 mmol/l) {<40  $\rightarrow$  LOW}
  - TAG <200 mg/dl (2.3 mmol/l) {200-499  $\rightarrow$  HIGH}
- Most smokers (around 80%) live in low and middle income countries.
- Tobacco use is the **single most** preventable cause of CVDs.
- Burning tobacco produces two forms of smoke:
  - Mainstream smoke
    - Particulate phase (Nicotine + TAR  $\rightarrow$  BV damage)
    - Gas phase (CO  $\rightarrow$  ischemia)
  - Sidestream smoke
    - Firsthand
    - Secondhand
    - Thirdhand

- WHO 4 steps strategy for tobacco control:
  - 1) Ban advertising and expand public health information.
    - a) Health warnings on all tobacco products & media campaigns
  - 2) Use taxes and regulations to reduce consumption.
  - 3) Encourage cessation of tobacco use
  - 4) Build anti-tobacco partnership
    - a) Prevent tobacco industry lobbying
- Jordan is considered as a high smoking prevalence country (10-20 cigarettes per day per smoker).
- **42.2% of people aged 15 and above in Jordan smoke tobacco.** (more prevalent in men -55.9%- than women -23.7%-)
- Increased prevalence of smoking are associated with: **male sex, higher income, lower academic attainment, and higher number of friends or family who smoke.**
- Dietary Sources of Cholesterol:

Type of Fat	Main Source	Effect on Cholesterol levels
Monounsaturated	Olives, olive oil, canola oil, peanut oil, cashews, almonds, peanuts and most other nuts; avocados	Lowers LDL, Raises HDL
Polyunsaturated	Corn, soybean, safflower and cottonseed oil; fish <i>sunflower</i>	Lowers LDL, Raises HDL
Saturated <i>- oil</i>	Whole milk, butter, cheese, and ice cream; red meat; chocolate; coconuts, coconut milk, coconut oil, egg yolks, chicken skin	Raises both LDL and HDL
Trans	Most margarines; vegetable shortening; partially hydrogenated vegetable oil; deep-fried chips; many fast foods; most commercial baked goods	Raises LDL

- JNC 7 Risk Factors:
  - Cigarette smoking
  - Hypertension (BP>140/90)
  - Low HDL (<40 mg/dL)
  - Age (men>45 ; women >55)
  - Familial history of premature CHD
  - CHD
  - CHD Risk equivalents

- Poor diet (risk factor) contributes to **72%** of CVD deaths.
- High fiber diet + diet in whole grain cereals lower CHD risk.
- Only important MUFA is **oleic acid** (olive oil, canola oil and nuts).
- Most important PUFA is **linoleic acid** (soybean & sunflower oils).
- Intake of trans fatty acids increases CHD risk. ( ↑ LDL & ↓ HDL)
- High sodium intake leads to high BP which is a major CHD risk. No more than 70 mmol or 1.7 g of sodium per day.
- **Potassium intake should be at a level which will keep the sodium to potassium ratio close to 1.**
- Fruits, vegetables, fish, and nuts are protective and reduce CHD risk. {**Regular fish consumption as 1-2 servings per week is protective.**}
- Boiled, unfiltered coffee raises LDL and total cholesterol due to a lipid called cafestol (high in unfiltered coffee).
- **Restrict the intake of saturated FA to less than 10% of daily energy intake and less than 7% for high-risk groups.**
- **Daily intake of fresh fruit and vegetables in an adequate quantity (400-500 g per day) to reduce CHD risk.**

### Surgery:

- Indications for Revascularization
  - To improve quality of life
  - Patients with activity-limiting symptoms
  - Patients with anatomy for which revascularization has a proven survival benefit
- PCI VS CABG?
  - Single-vessel / two-vessel (including right circumflex artery) diseases → PCI
  - Three-vessel disease (or two) which complete revascularization can't be accomplished / disease in LADA and/or circumflex coronary artery → CABG
- Arterial grafts have significantly better long-term graft patency.
- Arterial conduits:
  - 1- LIMA → Left Internal Mammary (Thoracic) Artery
  - 2- RA → Radial Artery
- Vein grafts → Great Saphenous Vein

- Patency rate:
  - PCI with stent: 90% for 1 year
  - Saphenous vein graft: 80% for 5 years
  - **LIMA to LADA: best conduit for CABG with about 90% patency rate for 15-20 years**
- Cardioplegia: to intentionally and temporarily arrest the heart in the diastole period “diastolic arrest” by a cold potassium solution.
- Recommendations to decrease mortality/morbidity after CABG:
  - Aspirin
  - Beta blockers
  - Statin
  - Glycemic control by Insulin infusion
  - Prophylactic antimicrobials
- Atrial Fibrillation AF occurs in 15-40% of patients undergoing CABG and up to 60% of those who undergo combined CABG with valve replacement.

### Infective Endocarditis IE:

- Most frequently IE affects the mitral and aortic valves.
- Staphylococcus aureus is the most common cause of IE.
- Intravenous drug abuse IE → tricuspid then mitral then aortic — causes right side heart failure — s. Aureus
- PVE → S. Epidermidis (coagulase negative) (nosocomial)
- Streptococci — most frequent cause of native valve infections.
- Embolic phenomena (most frequently associated with S aureus) in left sided endocarditis.
- Pulmonary embolism in right sided endocarditis.
- Duke’s clinical criteria: 2 major criteria - 1 major and 3 minor - 5 minor criteria
  - Major criteria: positive blood cultures for IE - evidence of endocardium involvement
  - Minor criteria: predisposition - fever - vascular phenomena-immunologic phenomena

## Physiology:

- Fibrinogen and globulin are responsible for blood viscosity, while albumin is responsible for blood osmotic pressure.
- The blood velocity at any point in the circulatory system is inversely proportionate to the total cross-sectional area.
- In the Atrial Contraction Phase (Systole): The 4<sup>th</sup> heart sound which is weak and inaudible due to vibration of atrial muscle during the contraction and rushing of blood into the ventricles.
- In rapid (maximum) ejection phase: the second component of the 1<sup>st</sup> heart sound due to rushing of blood into the aorta and vibration of the aortic wall.
- Cardiac output / body surface area = cardiac index (CI).
- $CI = CO/BSA = 5L / 1.73m = 3.2 L/min/m^2$ .
- $EF = (SV/EDV) \% - (normally\ 55\%)$
- In heavy stroking: red line is due to dilatation of the capillaries - red flare is due to dilatation in arterioles.
- Capillary wall is devoid of smooth muscles. (No muscle layer)

## Anatomy:

- Arch of azygos is in middle mediastinum, while the azygos and hemiazygos are in the posterior mediastinum.
- The pericardium lies within the middle mediastinum, posterior to the body of the sternum and the **2nd to the 6th** costal cartilages and anterior to the **5th to the 8th** thoracic vertebrae.
- **Angle of Louis** (sternal angle at the manubriosternal junction) (at 2nd rib opposite to T4/T5 vertebral disc) marks the following:
  - Division of mediastinum into superior and inferior
  - Ascending aorta becomes arch of aorta and at T4 it becomes descending thoracic aorta // {ascending aorta ends, arch of aorta begins, arch of aorta ends, descending aorta begins}
  - SVC receives arch of azygos vein
  - End of trachea (carina)
  - At T5 level, thoracic duct crosses behind the esophagus to the left
- The heart is related anteriorly to the sternum, costal cartilages, and the medial ends of the **3rd to 5th ribs** on the left side.

## Pathology:

- Myocardial rupture occurs most commonly within 3 to 7 days after infarction.
- “Widow maker” → coronary occlusion in proximal LADA (40% to 50% of all MIs).
- Acute Rheumatic Fever:
  - Aschoff bodies – myocardial inflammatory lesions
  - Antischkow cells “caterpillar cells”
  - Diagnosed by Jones criteria
- Chronic Rheumatic Fever:
  - Fibrous scar (instead of aschoff bodies)
  - “Fishmouth” or “buttonhole” stenosis
- Most common congenital valvular lesion is bicuspid aortic valve.
- Primary mitral valve prolapse is one of the most common forms of valvular heart disease.
- Endomyocardial fibrosis is one of the most common form of restrictive cardiomyopathy.
- The essential feature of hypertension is left ventricular hypertrophy “boxcar nuclei”.
- Atherosclerosis:
  - Fatty streaks - lipid-filled foamy macrophages
  - Atherosclerotic plaque - in descending severity
    - Infrarenal abdominal aorta
    - Coronary arteries
    - Popliteal arteries
    - Internal carotid arteries
    - Circle of Willis
- The two most important predisposing conditions for aortic aneurysms are atherosclerosis and hypertension.

## Biochemistry:

- Cardiac Biomarkers:

Biomarker	Specificity	Sensitivity	Start	Peak	End	
Myoglobin	Not specific	More sensitive than CK	1-4 hr	6-9 hr	18-24 hr	If normal after pain by 8h, MI can be ruled out
Total-CK	Not specific					Used on chest pain patients who are admitted later than 10-12 hr
CK-MB	High specificity (more than total-CK, less than troponin I)	Sensitive (was the gold marker before troponin I)	4-6 hr	12-24 hr	2-3 days	Useful for early diagnosis & <b>re-infarction</b> diagnosis
Troponin T	Not specific	More sensitive than CK-MB	6 hr	72 hr (3 days)	7-10 days (can't tell infarct from re-infarct)	May be elevated in patients with chronic renal failure
Troponin I	Specific	More sensitive than CK-MB	4-6 hr	14-24 hr	3-5 days (disappears from blood after a week)	Useful for diagnosis of delayed admission cases
LDH	Not specific		Increase later than CK-MB	48 hr	5-6 days	Useful when a patient with chest pain presents late

H-FABP			30 min (extremely early)	6-8 hr	24 hr	Used to quickly rule out MI
GPBB	Specific	Very sensitive (superior)	First hour of MI	1-3 hr after ischemia		Catch the MI before it happens (in ischemic phase)
Copeptin			Within minutes (15 min)			Adding copeptin to CTNI can rule out AMI
IMA	Not specific	Sensitive for ischemia	Within few minutes	2-4 hr	6 hr	Negative test (along with negative troponin and ECG) has 99% negative predictive value of MI

- High CK-MB levels after 5 days indicate re-infarction.
- Micro-RNA 208 is cardiac specific and has superior sensitivity at early time.
- Two negative troponin I tests 6 hours apart are good evidence of no recent AMI.
- Myoglobin is used for early diagnosis.
- The most specific cardiac biomarker is troponin I.
- CK-MB (not troponin) is used to diagnose re-infarction.
- AST is a nonspecific cardiac marker. It may increase because of hepatic congestion due to right-sided heart dysfunction.