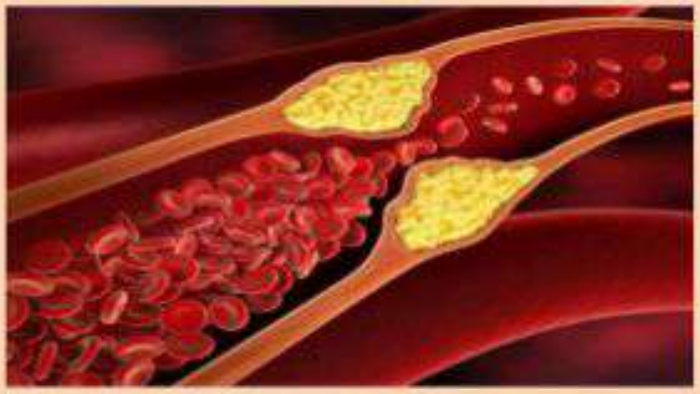
Ischemic heart disease

- Also known as **Coronary Artery Disease (CAD)**
- **Ischemic heart disease (IHD)** is a condition in which there is an **inadequate supply** of blood and oxygen to a portion of the myocardium
- Imbalance between **myocardial oxygen supply** and **demand**.
- Caused mainly by **Atherosclerosis of Coronary Artery**
- It includes
  - **Angina: Stable** & **Unstable**
  - **Myocardial infarction**
  - **Heart failure** & **Arrhythmia**

Atherosclerosis

- Can affect any artery in the body
- **Heart**: angina, MI and sudden death;
- **Brain**: stroke and transient ischaemic attack;
- **Limbs**: claudication and critical limb ischaemia.



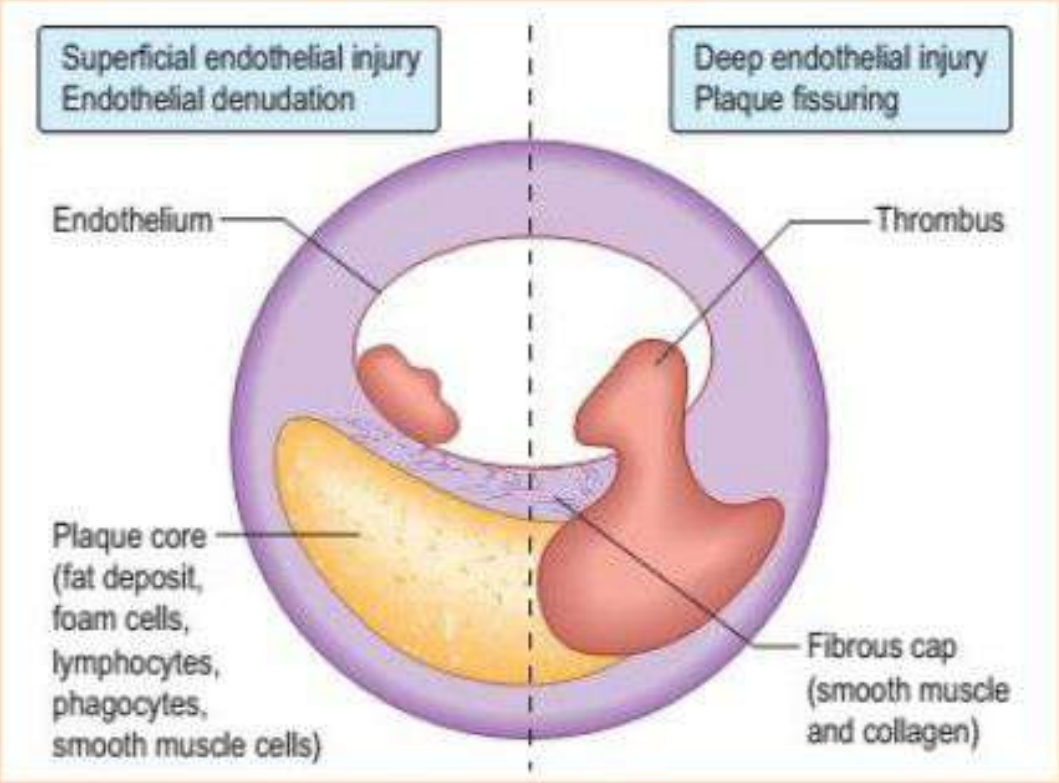
Atherosclerosis

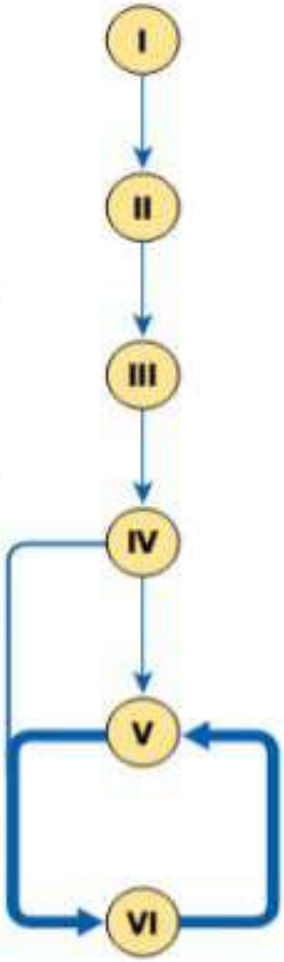
- Progressive **inflammatory disorder** of the arterial wall characterised by focal lipid rich deposits of **atheroma**
- Remain clinically assymptomatic until
  - large enough to impair tissue **perfusion**,
  - Ulceration and disruption of the lesion result in **thrombotic occlusion**
  - **Distal embolisation** of the vessel.
- Clinical manifestations depend upon the **site of the lesion** and the **vulnerability** of the organ supplied

Early Atherosclerosis

- Second and third decades of life
- Tend to occur at sites of **altered arterial shear stress** such as bifurcations
- Starts with any abnormal endothelial function
- Inflammatory cells, predominantly **monocytes**, bind to receptors expressed by endothelial cells,
- Migrate into the **intima**
- Take up oxidised **low-density lipoprotein (LDL)** particles
- Become **lipid-laden macrophages** or **foam cells**.
- As Foam cells dies, it releases its lipid pool in intimal space with **cytokines** and **growth factor**
- In response, smooth muscle cells migrate from the media of the arterial wall into the intima
- Lipid core will be covered by smooth muscle cells and matrix
- Forms **stable atherosclerotic plaque** that will remain asymptomatic until it becomes large enough to obstruct arterial flow

Early Atherosclerosis

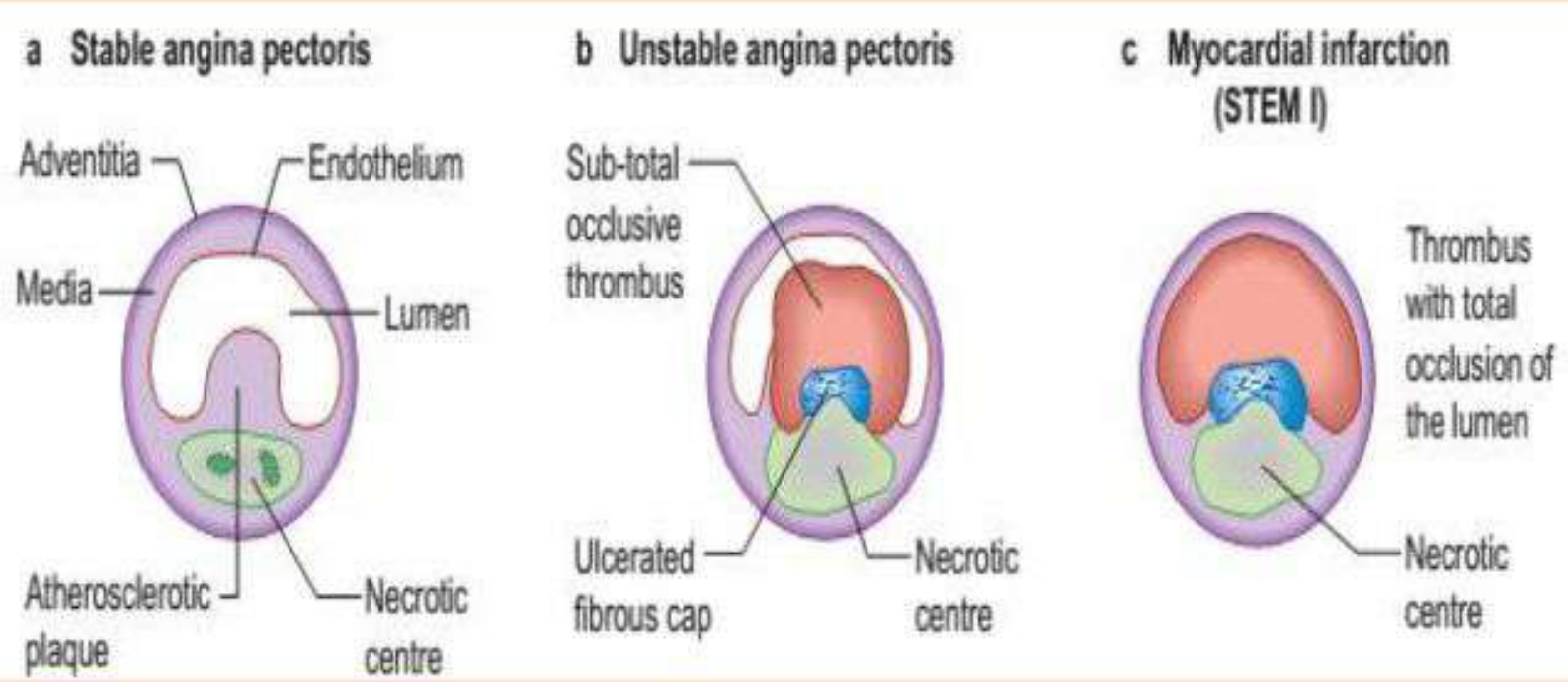


Nomenclature and main histology	Sequences in progression	Main growth mechanism	Earliest onset	Clinical correlation
Type I (initial) lesion Isolated macrophage foam cells 		Growth mainly by lipid accumulation	From first decade	Clinically silent
Type II (fatty streak) lesion Mainly intracellular lipid accumulation 				
Type III (intermediate) lesion Type II changes and small extracellular lipid pools 			From third decade	
Type IV (atheroma) lesion Type II changes and core of extracellular lipid 				Clinically silent or overt
Type V (fibroatheroma) lesion Lipid core and fibrotic layer, or multiple lipid cores and fibrotic layers, or mainly calcific, or mainly fibrotic 		Accelerated smooth muscle and collagen increase	From fourth decade	
Type VI (complicated) lesion Surface defect, haematoma-haemorrhage, thrombus 		Thrombosis, haematoma		

Advanced Atherosclerosis

- In established atherosclerotic plaque, **macrophages** mediate inflammation and **smooth muscle cells** promote repair
- Cytokines released by macrophage starts **degrading** smooth muscle layered over plaque
- Now lesion remains vulnerable to mechanical stress that ultimately causes **erosion, fissuring or rupture** of the plaque surface.
- Any breach in the integrity of the plaque will expose its contents to blood
- Trigger platelet aggregation and thrombosis
- That extend into the atheromatous plaque and the arterial lumen.
  - cause partial or complete obstruction at the site of the lesion
  - distal embolisation resulting in infarction
  - ischaemia of the affected organ

Advanced Atherosclerosis



Risk factor of Atherosclerosis

- Effect of risk factors is **multiplicative** rather than **additive**.
- It is important to distinguish between **relative risk** and **absolute risk**.
- **Absolute Risk**
  - Age
  - Male sex
  - Positive family history

Risk factor of Atherosclerosis

- **Relative Risk**
  - Smoking
  - Hypertension
  - Diabetes mellitus
  - Haemostatic factors.
  - Physical activity
  - Obesity
  - Alcohol
  - Other dietary factors
  - Personality
  - Social deprivation

Risk factor: Absolute Risk

- **Age & Sex**
  - **Premenopausal** women have **lower** rates of disease than **men**
  - Although this sex difference disappears after the **menopause**
- **Positive family history**
  - Runs in families,
  - Due to a combination of shared genetic, environmental and lifestyle factors.

Risk factor: Relative Risk

- **Smoking**
  - strong consistent
  - Dose linked relationship between cigarette smoking and IHD, especially in younger (< 70 years) individuals
- **Hypertension:** directly proportional
- **Hypercholesterolaemia:** directly proportional to **serum cholesterol concentrations (LDL)**
- **Diabetes mellitus:** potent risk factor for all forms of atherosclerosis
  - **Men with type 2 diabetes**: two- to four-fold greater annual risk of CAD,
  - **Women with type 2 diabetes:** (3–5-fold) risk

Risk factor: Relative Risk

- **Haemostatic factors**
  - **Platelet activation** and high levels of fibrinogen
  - **Antiphospholipid antibodies** - recurrent arterial thromboses
- **Physical activity**
  - Physical **inactivity** roughly **doubles** the risk of coronary heart disease
  - Regular exercise
    - Increased serum HDL cholesterol concentrations,
    - Lower BP,
    - Collateral vessel development
- **Obesity:** often associated with HTN and cardiovascular disease

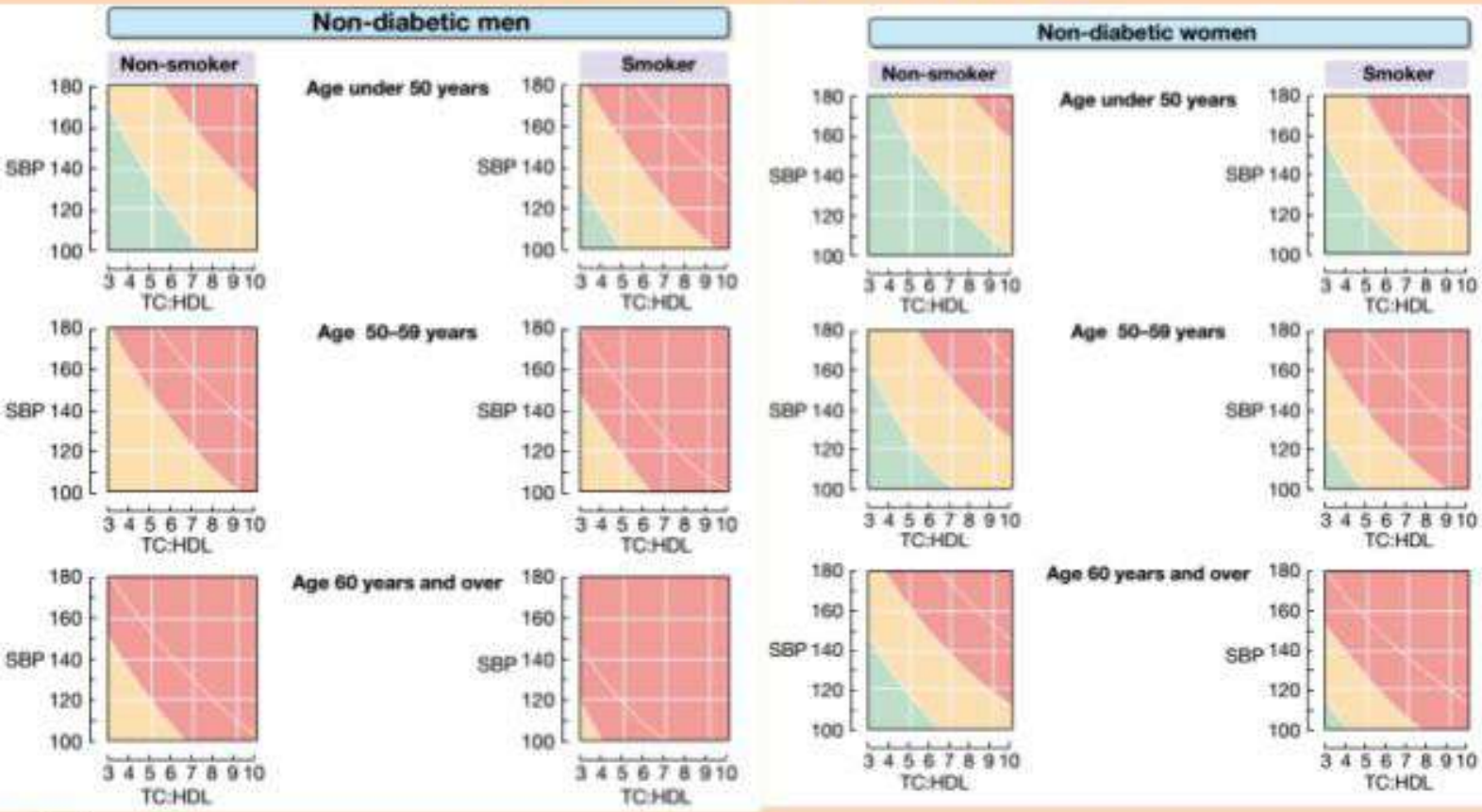
Risk factor: Relative Risk

- **Alcohol:** excess consumption
- **Other dietary factors**
  - Diets deficient in fresh **fruit**, **vegetables** and **polyunsaturated fatty acids (PUFA)**
- **Personality:** little or no evidence to support the popular belief that stress is a major cause of CAD
- **Social deprivation**

Primary Prevention

- Strategies taken before onset of disease in high risk individual.
- Two complementary strategies
- **Population strategies**
  - modify the risk factors of the whole **population**
  - through diet and lifestyle advice
  - For ex: Public restricting of smoking
- **Targeted strategies**
  - identify and treat high risk **individuals** who usually have a **combination of risk factors**
  - can be identified by using **composite scoring system**

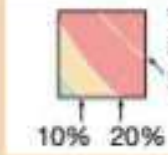
Composite Scoring System



CVD risk < 10% over next 10 years

CVD risk 10–20% over next 10 years

CVD risk > 20% over next 10 years

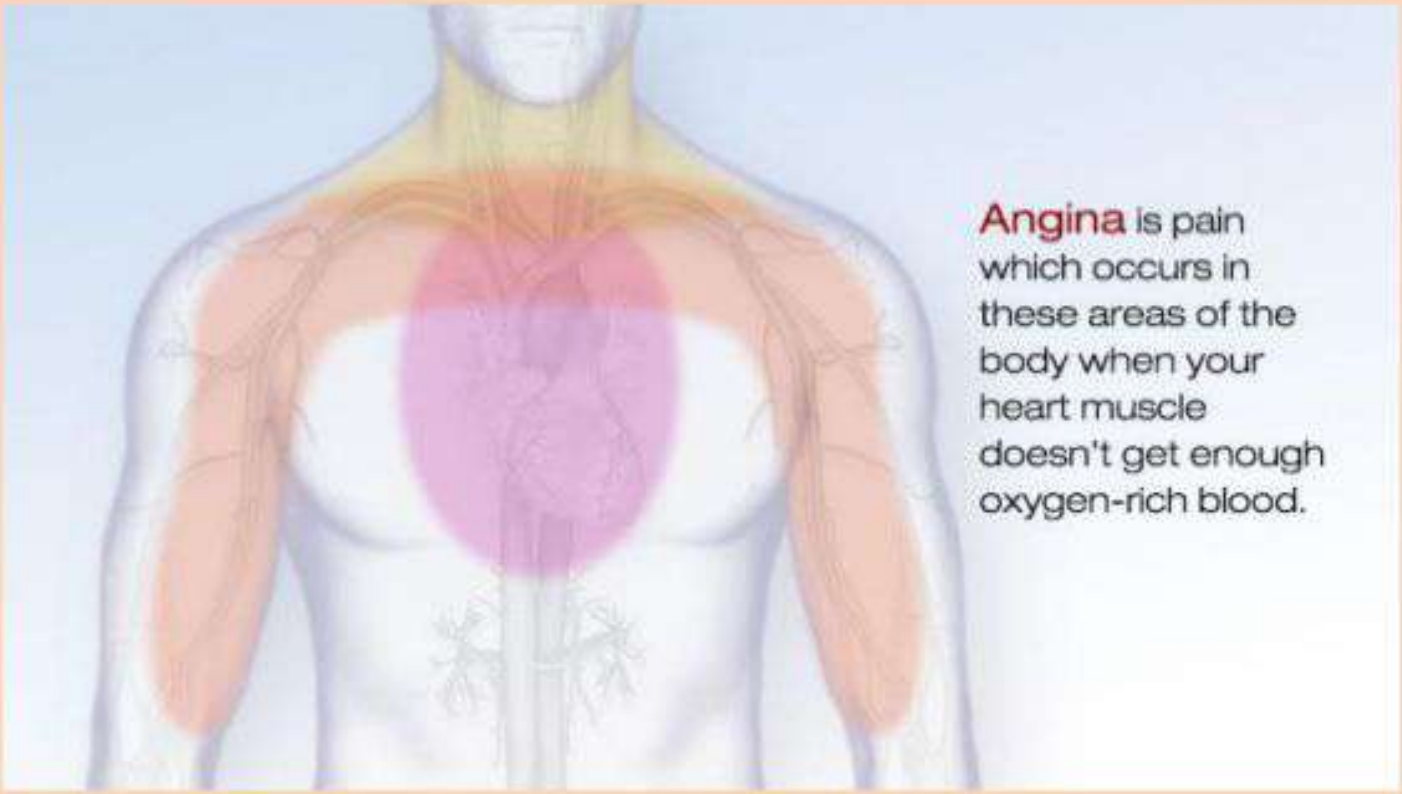


SBP = systolic blood pressure mmHg
TC:HDL = serum total cholesterol to HDL cholesterol ratio

Secondary Prevention

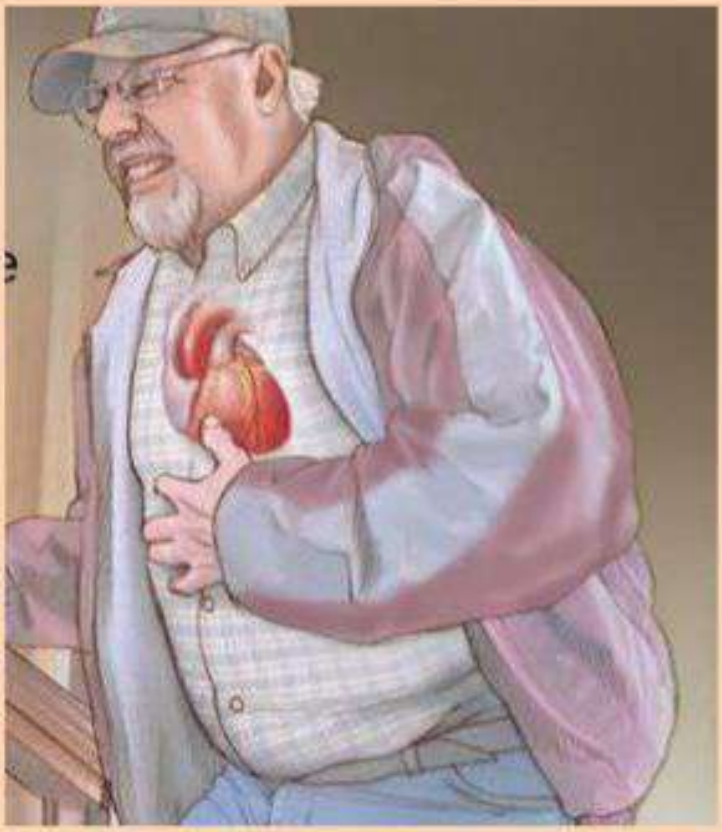
- Already have evidence of **atheromatous vascular disease** are at **high risk** of future cardiovascular events.
- Various secondary measures in this case
  - energetic correction of modifiable risk factors,
    - Smoking
    - Hypertension
    - Hypercholesterolaemia,
  - **Statin therapy** irrespective of their serum cholesterol concentration
  - Target BP of ≤ 140/85 mmHg
  - **Aspirin** and **ACE inhibitors**
  - **Beta-blockers**: h/o MI or heart failure.

Angina


Angina is pain which occurs in these areas of the body when your heart muscle doesn't get enough oxygen-rich blood.

Introduction

- A type of chest pain
- Not a disease, its a symptom of an underlying heart problem specially IHD
- Described as 'heavy', 'tight' or 'gripping'.
- Typically, central/retrosternal
- Mild ache to most severe that provokes sweating and fear
- Associated breathlessness.



Canadian cardiovascular society functional classification of angina

CLASS	Characteristic
Class I	No angina with ordinary activity. Angina with **strenuous activity**
Class II	Angina during ordinary activity, e.g. walking up hills, walking rapidly upstairs, with **mild limitation** of activities
Class III	Angina with low levels of activity, e.g. walking 50–100 yards on the flat, walking up one flight of stairs, with **marked restriction** of activities
Class IV	Angina at **rest** or with any level of exercise