University of Al-Qadisiyah
College of Medicine

4th stage

Ischemic Heart Disease (IHD)

By

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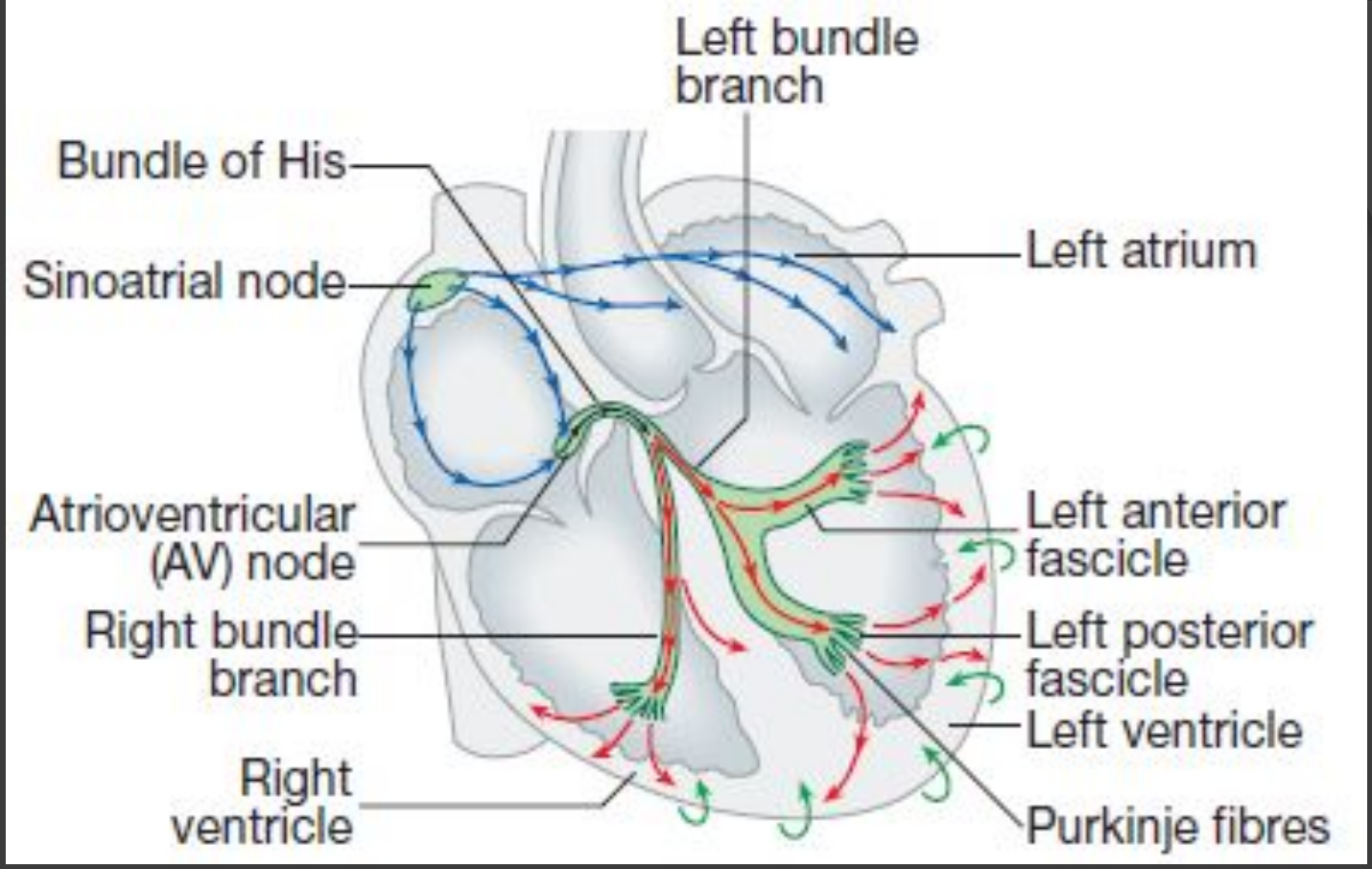


Conduction system

✔The SA node is situated at the junction of the superior vena cava and RA, which influenced by the autonomic nervous system and by catecholamines.

✔During normal (sinus) rhythm, this depolarization wave originate in SAN and propagates through both atria.

✔The only pathway for passing depolarizing wave is AV node with relatively slow conduction producing a necessary time-delay.

✔The His–Purkinje system is composed of the bundle of His extending from the AV node into the interventricular septum with very rapid conduction.

✔RBB and LBB passing along the inter-ventricular septum and into the respective ventricles, with anterior and posterior fascicles of the LBB to LV.

✔Depolarization spreads from epicardium to endocardium.

Nerve supply of the heart

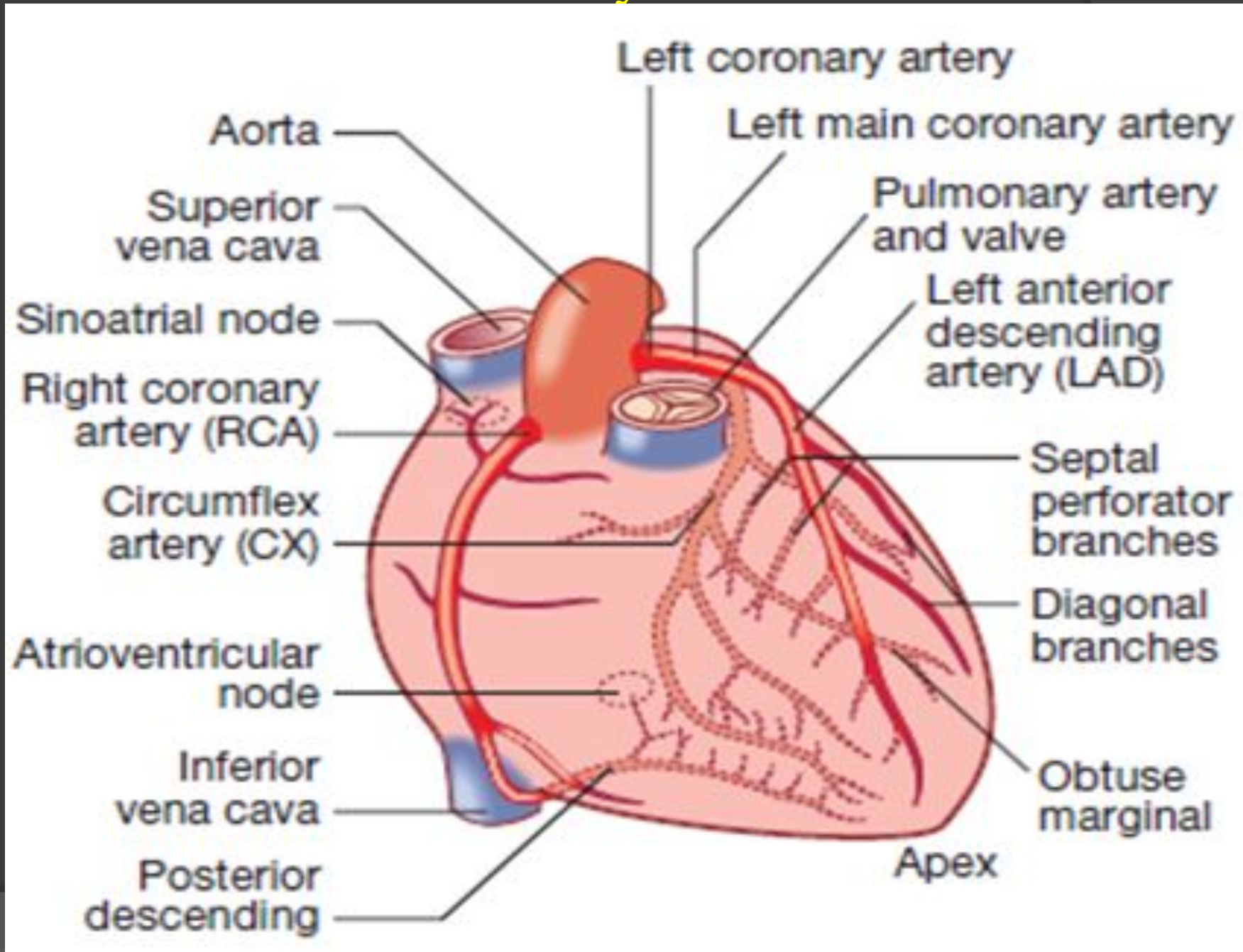
- The heart is innervated by sympathetic and parasympathetic fibers.

- Adrenergic nerves from the cervical sympathetic chain supply muscle fibers in the atria and ventricles, and the conducting system.

- Activation of β1-adrenoceptors results in positive inotropic and chronotropic effects, whereas activation of β2-adrenoceptors in vascular smooth muscle causes vasodilatation.

- Parasympathetic and sensory fibers reach the heart through the vagus nerves.
- Cholinergic nerves supply the AV and SA nodes via muscarinic (M2) receptors.

Coronary Circulation



The coronary circulation

- ✔ The left main stem (LMS) and right coronary arteries (RCA) arise from the left and right sinuses of valsalva from the aortic root, distal to the aortic valve.

- ✔ Within 2.5 cm of its origin, LMS divides into the left anterior descending artery (LAD), which runs in the anterior interventricular groove, and the left circumflex artery (CX), which runs posteriorly in the atrioventricular groove.

- ✔ The LAD gives branches to supply the anterior part of the septum (septal perforators) and the anterior, lateral and apical walls of the LV through diagonal (D) branches.

- ✔ The CX gives obtuse marginal (OM) branches that supply the lateral, posterior and inferior segments of the LV.

- ✔ The coronary anatomy varies greatly from person to person and there are many 'normal variants'. Congenital anomalies about 1-5%

- ✔ RCA runs in the right atrioventricular groove, giving branches that supply the RA, RV and inferoposterior aspects of the LV. The posterior descending artery (PDA) runs in the posterior interventricular groove and supplies the inferior part of the interventricular septum, and (PLV) posterior left ventricular branch supply the apex of LV

- ✔ The RCA supplies the sinoatrial (SA) node in about 60% of individuals and 40% from LAD , while the AV node supply from RCA in about 90% and 10% from LAD.

- ✔ Proximal occlusion of the RCA therefore often results in sinus bradycardia and may also cause AV nodal block.

- ✔ Abrupt proximal occlusions of RCA, due to coronary thrombosis, result in infarction of the inferior part of the LV and often the RV.
- ✔ Abrupt occlusion of the LAD or CX causes infarction in the corresponding territory of the LV, and occlusion of the LMS is usually fatal.
- ✔ The venous system follows the coronary arteries but drains into the coronary sinus in the atrioventricular groove, and then to the RA.
- ✔ An extensive lymphatic system drains into vessels that travel with the coronary vessels and then into the thoracic duct.

Coronary dominance

- The dominant coronary artery is the one from which the PDA arises.
- The RCA is the dominant artery in about 85% of patients.
- If the PDA arises from the terminal portion of the LCX, the term left dominance is applied to the circulation (8%).
- For many, the posterior septum is supplied by branches arising from both the RCA and LCX (balanced or co-dominant), (7%) with dual-posterior descending arteries (one from the RCA and the other from the LCX) or no clear PDA with multiple smaller branches arising from both arteries.

Physiology: ***Blood flow***

- ✔ COP= SV x HR, SV is the volume of blood ejected in each cardiac cycle, and depend on end-diastolic volume and pressure (preload), myocardial contractility and systolic aortic pressure (afterload).

- ✔ The tone of resistance vessels is tightly regulated by humoral, neuronal and mechanical factors.

- ✔ Systemic and locally released vasoactive substances influence tone; vasoconstrictors include noradrenaline (norepinephrine), angiotensin II and endothelin-1, whereas adenosine, bradykinin, prostaglandins and nitric oxide are vasodilators.

- ✔ Resistance to blood flow rises with viscosity and is mainly influenced by red cell concentration (haematocrit).

- ✔ Coronary blood vessels receive sympathetic and parasympathetic innervation. Stimulation of α-adrenoceptors causes vasoconstriction; stimulation of β2-adrenoceptors causes vasodilatation; the predominant effect of sympathetic stimulation in coronary arteries is vasodilatation. Parasympathetic stimulation also causes modest dilatation of normal coronary arteries.

- ✔ As a result of vascular regulation, an atheromatous narrowing (stenosis) in a coronary artery does not limit flow, even during exercise, until the cross-sectional area of the vessel is reduced by at least 70%.

Coronary endothelial function

The endothelium plays a vital role in the control of vascular homeostasis. The function includes:

1. Synthesis and releases many vasoactive mediators that cause vasodilatation, including: nitric oxide, prostacyclin and endothelium-derived hyperpolarising factor.

2. Synthesis and releases vasoconstrictors, including: endothelin-1 and angiotensin II. Damage to the endothelium may disrupt the balance between vasodilators and vasoconstrictors and lead to vascular dysfunction, tissue ischaemia and hypertension.

3. Major influence on the recruitment of inflammatory cells and on the formation and dissolution of thrombus.

4. Store and releases the multimeric glycoprotein, von Willebrand factor, which promotes thrombus formation by linking platelet adhesion to denuded surfaces, especially in the arterial vasculature.

5. Tissue plasminogen activator is rapidly released from a dynamic storage pool within the endothelium once intravascular thrombus forms to induce fibrinolysis and thrombus dissolution.

Presenting problems in cardiovascular disease

A. Chest pain

Characteristics of cardiac pain

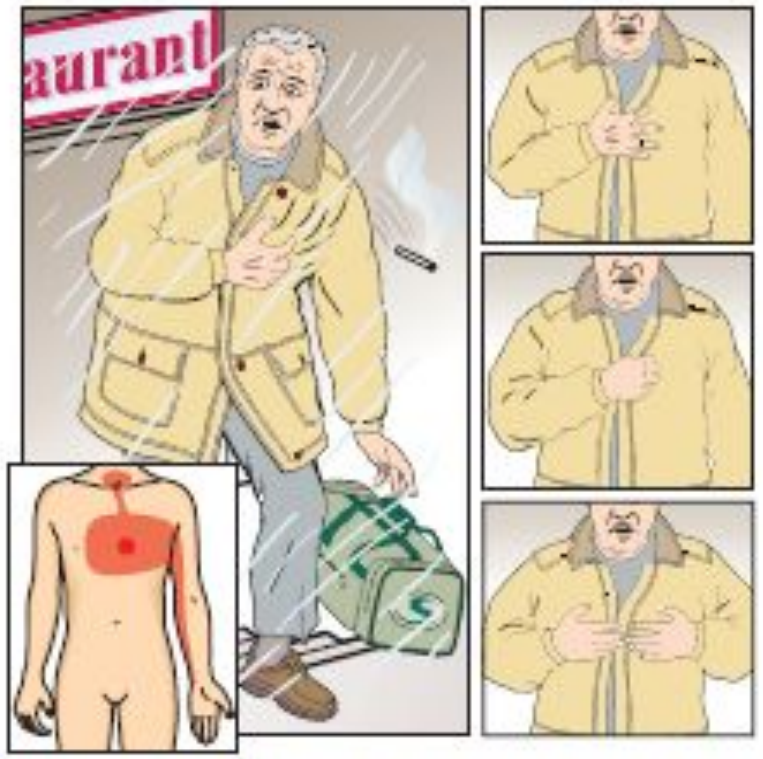


Fig. 18.16 Typical ischaemic cardiac pain. Characteristic hand gestures used to describe cardiac pain. Typical radiation of pain is shown in the schematic.

Common causes of chest pain

Anxiety/emotion

Cardiac
- Myocardial ischaemia (angina)
- MI
- Pericarditis
- Mitral valve prolapse

Aortic
- Aortic dissection
- Aortic aneurysm

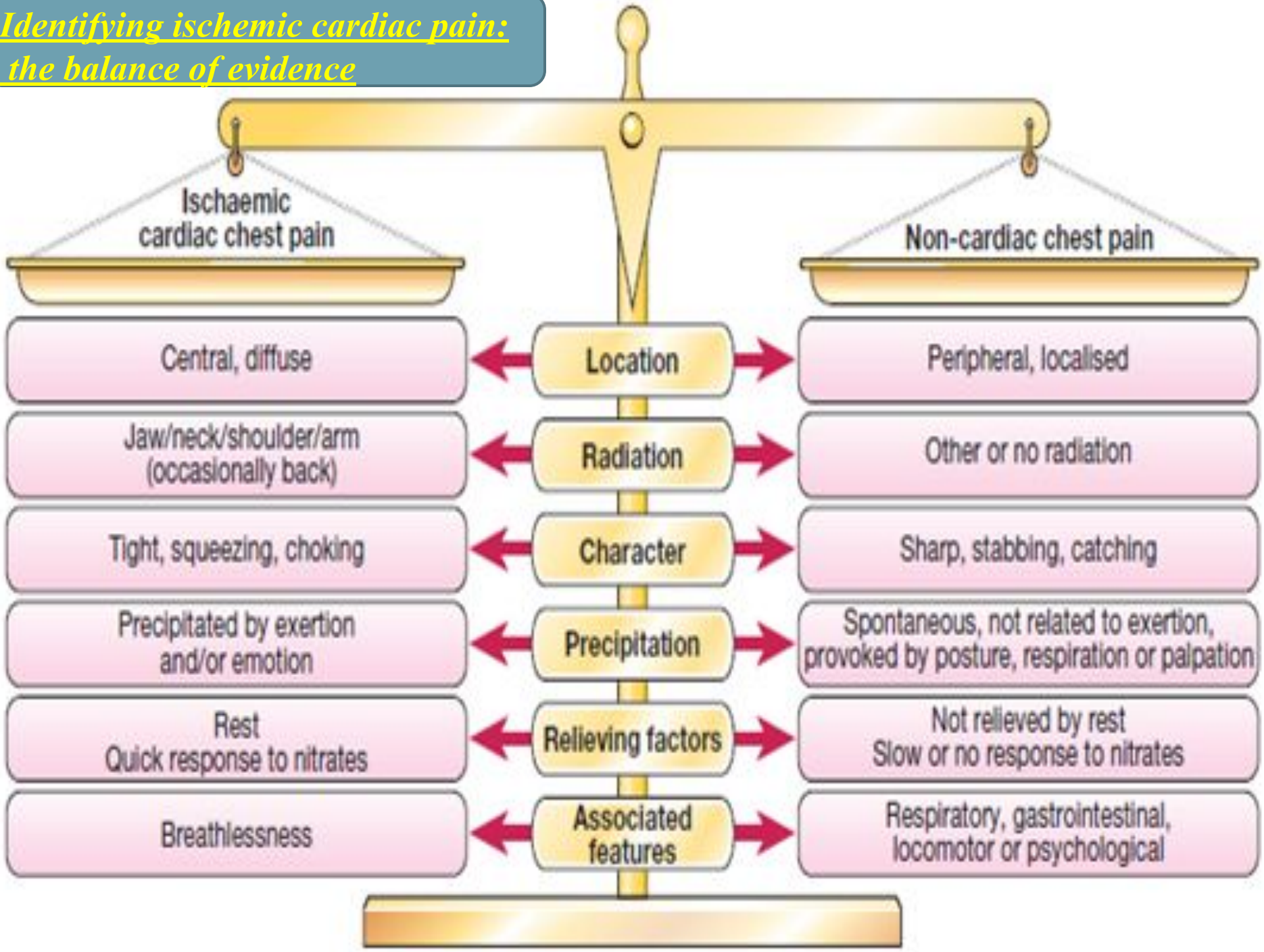
Oesophageal
- Oesophagitis
- Oesophageal spasm
- Mallory–Weiss syndrome

Lungs/pleura
- Bronchospasm
- Pulmonary infarct
- Pneumonia
- Tracheitis
- Pneumothorax
- Pulmonary embolism
- Malignancy
- Tuberculosis
- Connective tissue disorders (rare)

Musculoskeletal
- Osteoarthritis
- Rib fracture/injury
- Costochondritis (Tietze's syndrome)
- Intercostal muscle injury
- Epidemic myalgia (Bornholm disease)

Neurological
- Prolapsed intervertebral disc
- Herpes zoster
- Thoracic outlet syndrome

Identifying ischemic cardiac pain: the balance of evidence



B. Breathlessness (dyspnea)

Dyspnea of cardiac origin may vary in severity from an uncomfortable awareness of breathing to a frightening sensation of 'fighting for breath.

New York Heart Association (NYHA) functional classification	
Class I	No limitation during ordinary activity
Class II	Slight limitation during ordinary activity
Class III	Marked limitation of normal activity without symptoms at rest
Class IV	Unable to undertaken physical activity without symptoms, symptoms may be present at rest

C. Arrhythmia

Any arrhythmia may cause breathlessness but usually only does so if the heart is structurally abnormal, such as with the onset of atrial fibrillation in a patient with mitral stenosis.

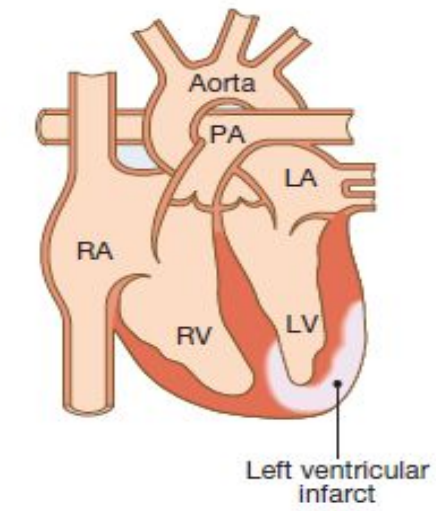
D. Angina equivalent

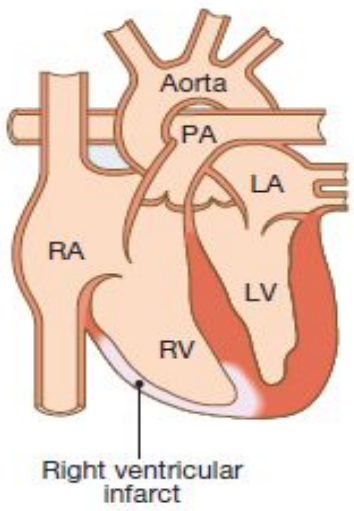
Breathlessness is a common feature of angina. Patients will sometimes describe chest tightness as ‘breathlessness’. Myocardial ischaemia may also induce true breathlessness by provoking transient left ventricular dysfunction or heart failure. When breathlessness is the dominant or sole feature of myocardial ischaemia, it is known as ‘angina equivalent’.

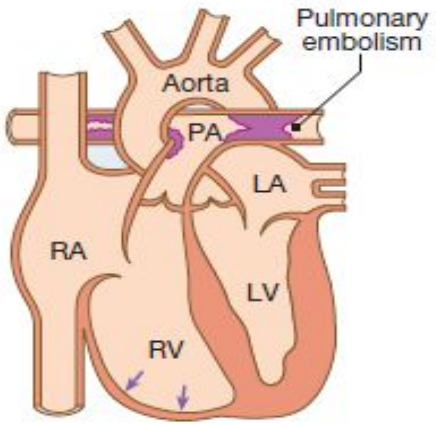
E. Acute circulatory failure (cardiogenic shock):

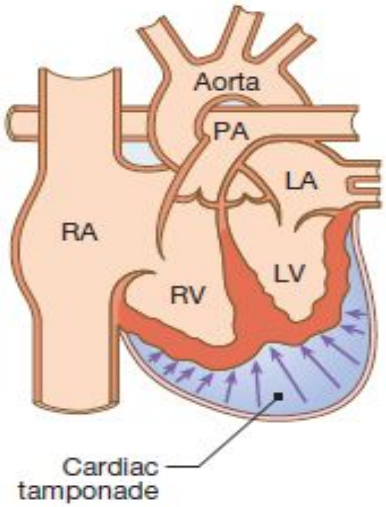
‘Shock’ is used to describe the clinical syndrome that develops when there is critical impairment of tissue perfusion due to some form of acute circulatory failure.

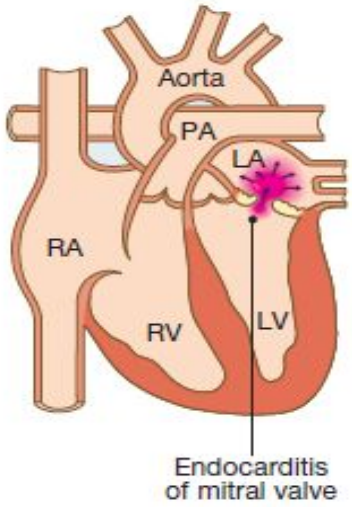
Causes of cardiogenic shock:











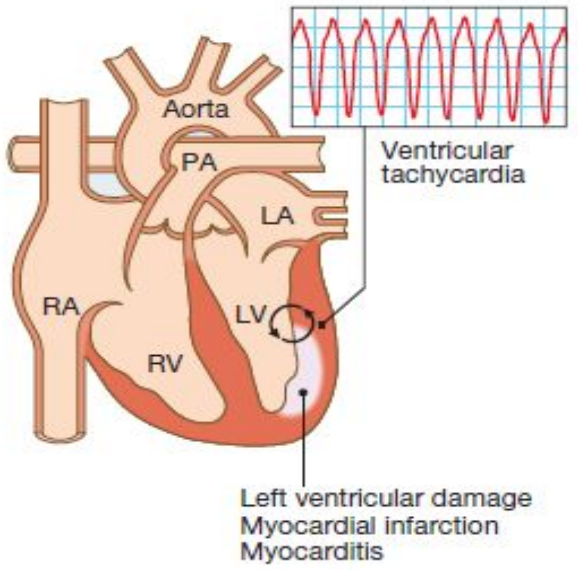


Fig. 18.20 Some common causes of cardiogenic shock.

F. Palpitation

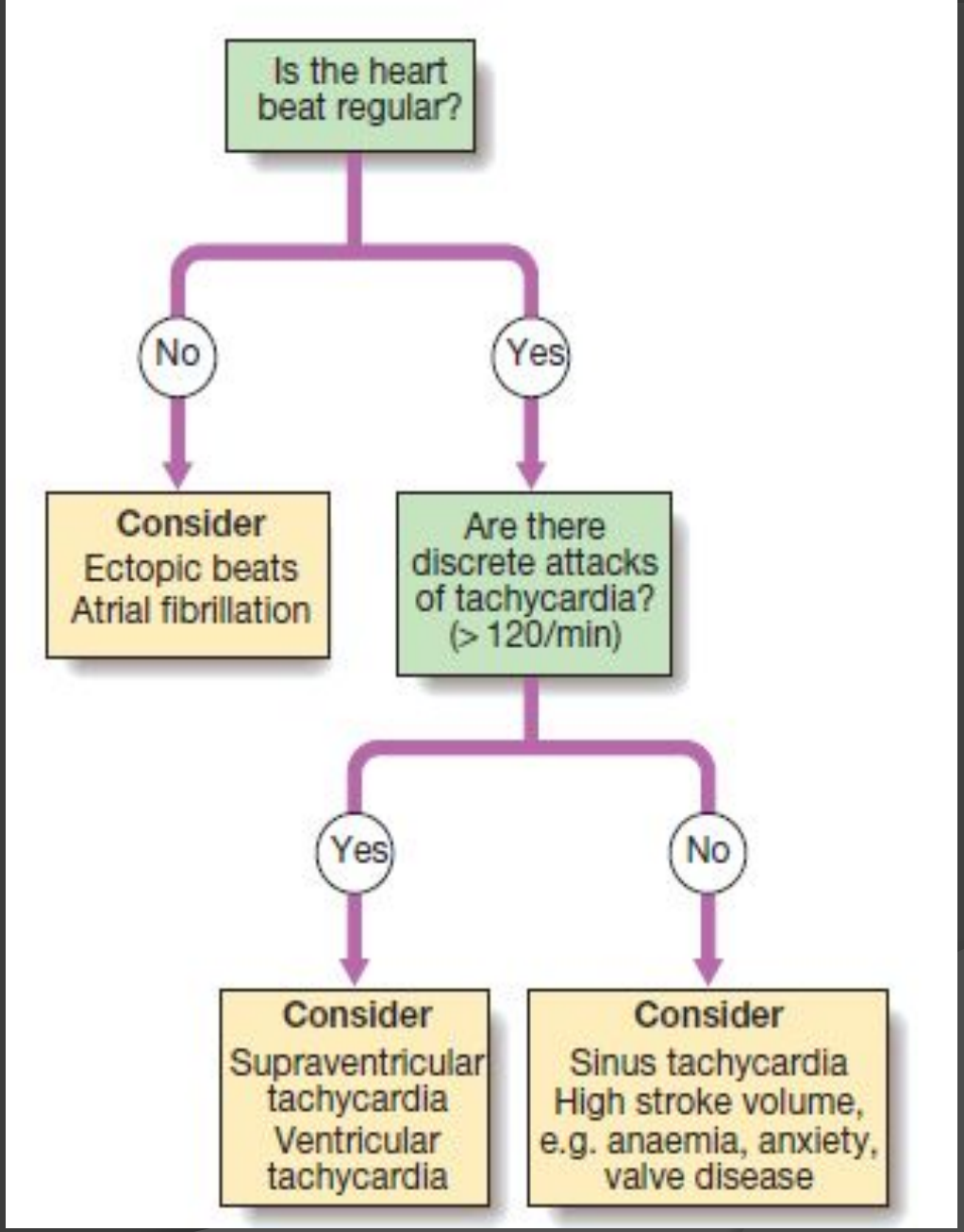
** Very common , may present as chest pain, forceful heart beat, even breathlessness, and the diagnosis should be confirmed by an ECG recording during an episode using an ambulatory ECG monitor. Recurrent but short-lived bouts of an irregular heart beat are usually due to atrial or ventricular extrasystoles (ectopic beats).

** These may also reflect a hyperdynamic circulation, such as anaemia, pregnancy and thyrotoxicosis, and can occur in some forms of valve disease.

** Most cases are due to an awareness of the normal heart beat, a sinus tachycardia or benign extrasystoles, in which case an explanation and reassurance may be all that is required.

** Palpitation associated with pre-syncope or syncope may reflect more serious structural or electrical disease and should be investigated without delay.

Approach for patient with palpitation

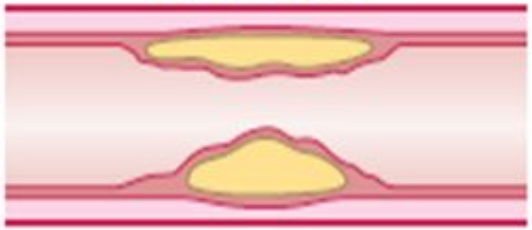


CAD: clinical manifestation and pathology

Clinical problem	pathology
Stable angina	Ischemia due to fixed stenosis of one or more coronary arteries
Unstable angina	Ischemia caused by dynamic obstruction of coronary artery due to plaque rupture erosion with superimposed thrombosis
MI	Myocardial necrosis caused by acute occlusion of coronary artery due to plaque rupture or erosion with superimposed thrombosis
Heart failure	Myocardial dysfunction due to infarction or ischemia
Arrhythmia	Altered conduction due to ischemia or infarction

CAD is the most common form of heart disease & almost always due to atheroma and its complications, particularly thrombosis.

Occasionally, the coronary arteries are involved in other disorders such as aortitis, polyarteritis and other connective tissue disorders.

	Stable angina	Acute coronary syndrome
		
Pathophysiology	• Fixed stenosis • Stable fibrous plaque	• Dynamic stenosis • Ruptured or inflamed plaque
Clinical features	• Demand-led ischaemia • Related to effort • Predictable • Symptoms over long term	• Supply-led ischaemia • Symptoms at rest • Unpredictable • Symptoms over short term • Frequent or nocturnal symptoms
Risk assessment	• Symptoms on minimal exertion • Exercise testing Duration of exercise Degree of ECG changes Abnormal BP response • CT coronary angiogram	• ECG changes at rest • ECG changes with symptoms • Elevation of troponin

Pathophysiology, clinical features and risk assessment of patients with stable or unstable coronary heart disease.

Chronic stable angina (CSA) or chronic coronary syndrome

- Angina pectoris is the symptom complex caused by *transient myocardial ischaemia* and occur due to imbalance between myocardial oxygen supply and demand. *Effort-related chest pain is the hallmark of angina pectoris or 'choking in the chest'.*

- Coronary atheroma is the most common cause of angina, although the symptom may be a manifestation of other disease (aortic valve disease and hypertrophic cardiomyopathy)

- Investigations should be done in form of echo if patient has murmur. Other include full blood count, FBS, lipids, TFT and ECG, TMT may confirm Dx & identify high-risk patients

- CT coronary angiography is very useful to exclude the presence of CAD.

Clinical classification of CSA:

Comment	Definition	CCS class
Angina only with extraordinary exertion at work	Ordinary physical activity does not cause angina	I
Angina with walking more than two blocks or climbing more than one flight of stairs	Slight limitation of ordinary activity	II
Walking one to two blocks or climbing one flight of stairs	Marked limitation of ordinary physical activity	III
Angina at rest or with minimal activity or stress	Inability to carry on any activity without discomfort	IV

Clinical features

** The history is the most important factor in making the diagnosis.

** Stable angina is characterized by central chest pain, discomfort or breathlessness that is precipitated by exertion or other forms of stress, and is promptly relieved by rest.

** Some patients find the discomfort comes when they start walking (‘warm-up angina’).

** Physical examination is frequently unremarkable in CSA but should include a careful search for evidence of valve disease (particularly aortic), important risk factors (e.g. HT, DM), left ventricular dysfunction (cardiomegaly, gallop rhythm), other manifestations of arterial disease (carotid bruits, PAD) and unrelated conditions that may exacerbate angina (anaemia, thyrotoxicosis).

Factors influencing myocardial oxygen supply and demand

Oxygen demand: cardiac work		
• Heart rate • LVH stenosis	* BP	* Myocardial contractility * Valve disease e.g. aortic
Oxygen supply: coronary blood flow		
• Duration of diastole • Coronary vasomotor tone	* Coronary perfusion pressure * Oxygenation (Hb, oxygen saturation)	

Activities precipitating angina

Common
Physical exertion
Heavy meal
Cold exposure
Intense emotion
Uncommon
Lying flat (decubitus angina)
Vivid dream (nocturnal angina)

Risk stratification in stable angina	
High risk	*Low risk*
Post infarct angina	Predictable exertional angina
Poor effort angina	Good effort angina
Ischemia at low work load	Ischemia only at high workload
Left main or 3VD	Single or 2 vessels diseases
Poor LV function	Good LV function

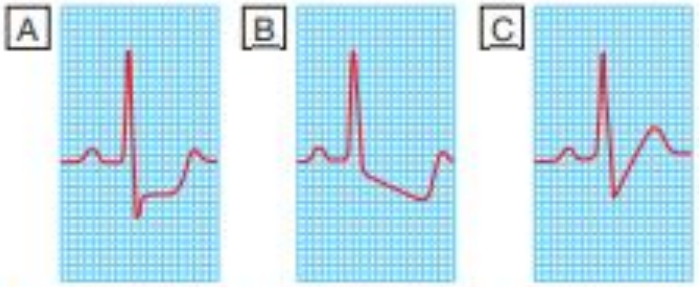
Investigations

1. *Resting ECG*

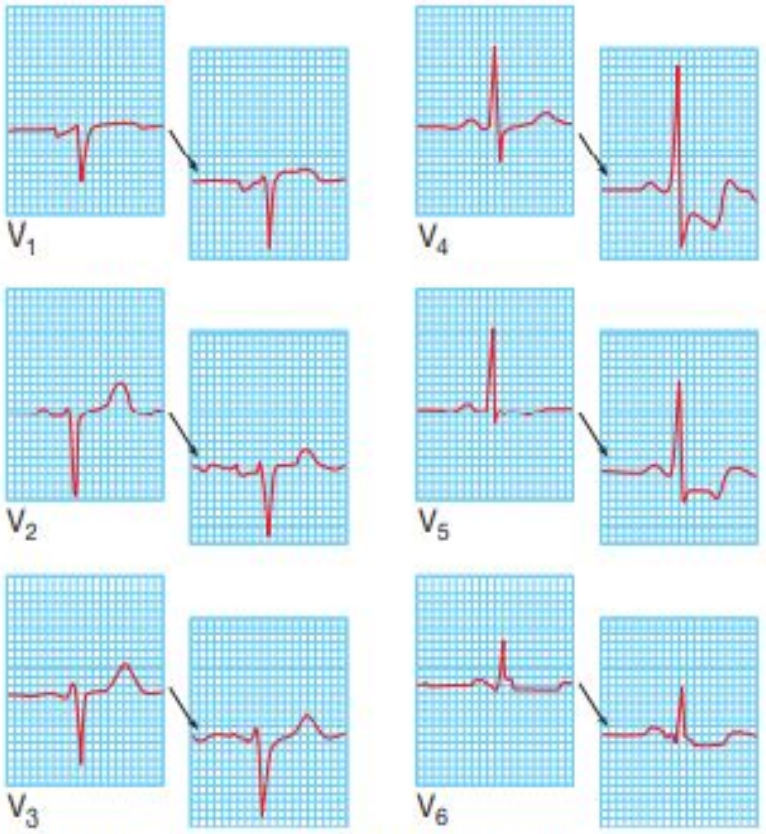
- The ECG may show evidence of previous MI but is often normal.
- T-wave flattening or inversion in some leads, providing non-specific evidence of myocardial ischaemia.
- The most convincing ECG evidence of myocardial ischaemia is the demonstration of *reversible ST segment depression or elevation, with or without T-wave inversion.*

2. *Exercise ECG*

- Planar or down-sloping ST segment depression of 1 mm or more is indicative of ischaemia. Up-sloping ST depression is less specific and often occurs in normal individuals.
- Exercise testing may produce false-positive results in the presence of digoxin therapy, LVH, BBB or WPW syndrome, young female and mitral valve prolapse



Forms of exercise-induced ST depression. A Planar ST depression is usually indicative of myocardial ischaemia. B Down-sloping depression also usually indicates myocardial ischaemia. C Up-sloping depression may be a normal finding.



A positive exercise test (chest leads only). The resting 12-lead ECG shows some minor T-wave changes in the inferolateral leads but is otherwise normal. After 3 minutes' exercise on a treadmill, there is marked planar ST depression in leads V_4 and V_5 (right-hand offset). Subsequent coronary angiography revealed critical three-vessel coronary artery disease.

3. Other forms of stress testing

A. *Myocardial perfusion scanning.*

Helpful in evaluation of patients with an equivocal exercise test and those who are unable to exercise, by obtaining scans at rest and during stress after the administration of an intravenous radioactive isotope, such as 99 technetium tetrofosmin.

B. Thallium and tetrofosmin are taken up by viable perfused myocardium. A perfusion defect present during stress but not at rest provides evidence of reversible myocardial ischaemia, whereas a persistent perfusion defect seen during both phases of the study is usually indicative of previous MI.

C. *Stress echo.* It uses transthoracic echo to identify ischaemic segments of myocardium (exhibit reversible defects in contractility during exercise or pharmacological stress) and areas of infarction (do not contract at rest or during stress).

4. *Coronary arteriography*


THANK YOU