Ischaemic heart disease
Definition

- An imbalance between the **supply of oxygen** and the **myocardial demand** resulting in myocardial ischaemia.

1-Angina pectoris

2-myocardial infarction

Ischaemic heart disease
Definition

- **Supply**
  - Atheroma, thrombosis, spasm, embolus
- **Demand**
  - Anaemia, hypertension, high cardiac output (thyrotoxicosis, myocardial hypertrophy)

Ischaemic heart disease
Aetiology

1– strong association

Dyslipidaemia, smoking, diabetes mellitus, obesity, hypertension

2- weak association

Lack of exercise, high alcohol consumption, type A personality, soft water

3- other include -Age, Male, +ve family history

Ischaemic heart disease Pathophysiology

ATHEROSIS •

Accumulation of cholesterol within the vessel wall intima. Smooth muscle cell proliferation

- **SCLEROSIS**

  Expansion of fibrous tissue

- **INFLAMMATION**

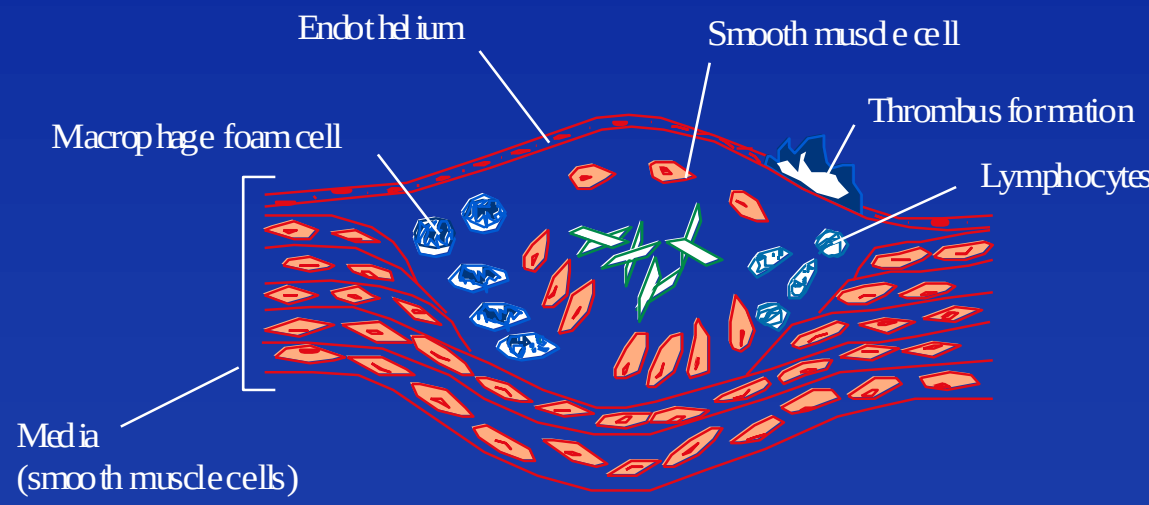
  Chronic inflammatory cells migrate into wall, release cytokines

- **GROWTH FACTORS/INFLAMMATORY MEDIATORS**

Ischaemic heart disease Pathophysiology

An atherosclerotic lesion

Schematic illustration



Ischaemic heart disease
Acute coronary syndromes

Atherosclerosis

Fatal / non-fatal MI

Unstable angina

Coronary Artery spasm

1-Angina Pectoris

Definition: Angina pectoris is severe pain originating from the heart that occurs in response to an inadequate oxygen supply to the myocardial cells. The pain of angina may radiate down the left arm, to the back, to the jaw, or into the abdominal area.

When the workload of any tissue increases, oxygen demand goes up. If the oxygen demand increases in healthy hearts, the coronary arteries dilate and bring more blood flow and oxygen to the muscle. However, if the coronary arteries are stiffened or narrowed with atherosclerosis and cannot dilate in response to an increased demand for oxygen, myocardial ischemia (inadequate blood supply) occurs, and the myocardial cells begin to use anaerobic glycolysis to meet their energy requirements.

This form of energy production is very inefficient and results in the production of lactic acid. Lactic acid decreases myocardial pH and causes the pain associated with angina pectoris.

Classification of angina

Exertional angina, Stable,Atherosclerotic,Classic,

Due to obstruction of coronaries by atheroma.

1- Stable angina

Stable angina, also called classic angina, occurs when atherosclerotic coronary arteries cannot dilate to increase flow when oxygen demand is increased. Increased work of the heart can accompany physical exercise, such as sports participation or climbing stairs. Exposure to the cold.

Mental stress, such as that caused by anger or by mental tasks such as mathematics, may trigger classic angina. The pain of stable angina typically goes away when the individual stops the activity.

2. **Variant, Vasospastic angina**

Variant angina (Prinzmetal's angina) almost always occurs during periods of rest, usually at night. The cause is a spasm of a coronary artery. Many people who have this type also have severe atherosclerosis

3- **Unstable angina**.

Due to spasm and partial obstruction of coronaries. Unstable angina, one of the acute coronary syndromes that includes heart attack, is characterized by a change in the pattern of angina episodes, occurring more frequently, at rest, and/or not responding to treatment

Clinical Manifestations

Constricting or squeezing pain in the pericardial or substernal area of the chest, possibly radiating to the arms, jaw, or thorax.

In stable and unstable angina, pain is typically relieved by rest.

Prinzmetal's angina is unrelieved by rest but usually disappears in about 5 minutes.

Diagnostic Tools

Alteration in the ST segment of the ECG may occur.

Areas of reduced blood flow may be observed using radioactive imaging during an induced angina episode as part of an exercise stress test.

Cardiac enzymes and proteins may be measured to rule out MI.

Chest Pain
Differential diagnosis

- **Cardiac pathology**
  - Pericarditis
- **Pulmonary pathology**
  - Pulmonary embolus,, pneumonia
- **Gastrointestinal pathology**
  - Peptic ulcer disease, reflux’
- **Musculoskeletal pathology**
  - Trauma

Myocardial Infarction

Myocardial Infarction

Myocardial infarction (MI) is the death of myocardial cells that occurs following prolonged oxygen deprivation.. Myocardial cells begin to die

Effect of an MI on Cardiac Depolarization, Cardiac Contractility, and Blood Pressure

With the release of potassium ion and the various intracellular enzymes, and with the accumulation of lactic acid, the electrical conduction pathways of the heart are altered. This can result in interruption of atrial or ventricular depolarization, or in initiation of a dysrhythmia.

With the death of muscle cells and changes in the heart's electrical patterns, the heart begins to pump in a less coordinated manner, causing contractility to decrease. Stroke volume falls, causing a fall in systemic blood pressure

Symptoms and signs of myocardial ischaemia

-Changes in blood pressure

-Changes in heart rate /rhythm

Causes of Myocardial Infarct

Myocardial infarct is usually the outcome of long-standing coronary artery disease (CAD). For example, a common cause of an MI is the rupture of an atherosclerotic plaque from one of the coronary arteries, and the subsequent obstruction of blood flow that occurs as it is trapped downstream. An MI might also occur if a thrombotic lesion adhering to a damaged artery becomes large enough to totally obstruct flow downstream,

If a heart chamber becomes so hypertrophied that it is unable to meet its oxygen demands, .for example, in a patient with long-standing hypertension.

Risk factors of MI

Family History
Smoking
Hypertension
Diabetes Mellitus
Hypercholesterolaemia
Lack of exercise

Acute Myocardial Infarction
Confirming the diagnosis

- Typical chest pain

- Electrocardiographic changes
  - ST elevation
- Myocardial enzyme elevation
  - Creatine kinase
  - Troponin

Clinical Manifestations

Although some individuals do not show any obvious signs of an MI (a silent heart attack), :significant clinical manifestations usually occur

1- pain, often described as severe and crushing in nature. The pain may radiate anywhere on the upper body, but most often radiates to the left arm, neck, or jaw. Nitrates and rest might relieve ischemia outside the necrotic zone by decreasing the workload of the heart but will not relieve the pain of infarct completely

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.2-Nausea and vomiting, probably related to intense pain, are common

3-Feelings of weakness related to decreased blood flow to the skeletal muscles occur.

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4-The skin becomes cool, and pale due to sympathetic vasoconstriction

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4-Urine output decreases related to decreased renal blood flow and increased aldosterone and ADH

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5-Tachycardia develops, due to increased cardiac sympathetic stimulation and anxiety

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6-A mental state of great anxiety, perhaps related to release of stress hormones and ADH .(vasopressin

Diagnostic Tools

1-A good history and physical, including family history of heart disease.

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2-Blood pressure may be decreased or normal depending on extent of myocardial damage and success of the baroreceptor reflexes.
Heart rate is usually increased.

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3-The ECG may show acute changes with elevation in the ST segment and T wave inversion. Within 1 or 2 days of the infarct, deepening of the Q wave occurs.

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4-Systemic signs of inflammation occur, including fever, elevated number of leukocytes. These signs begin about 24 hours after the infarct and continue for up to 2 weeks

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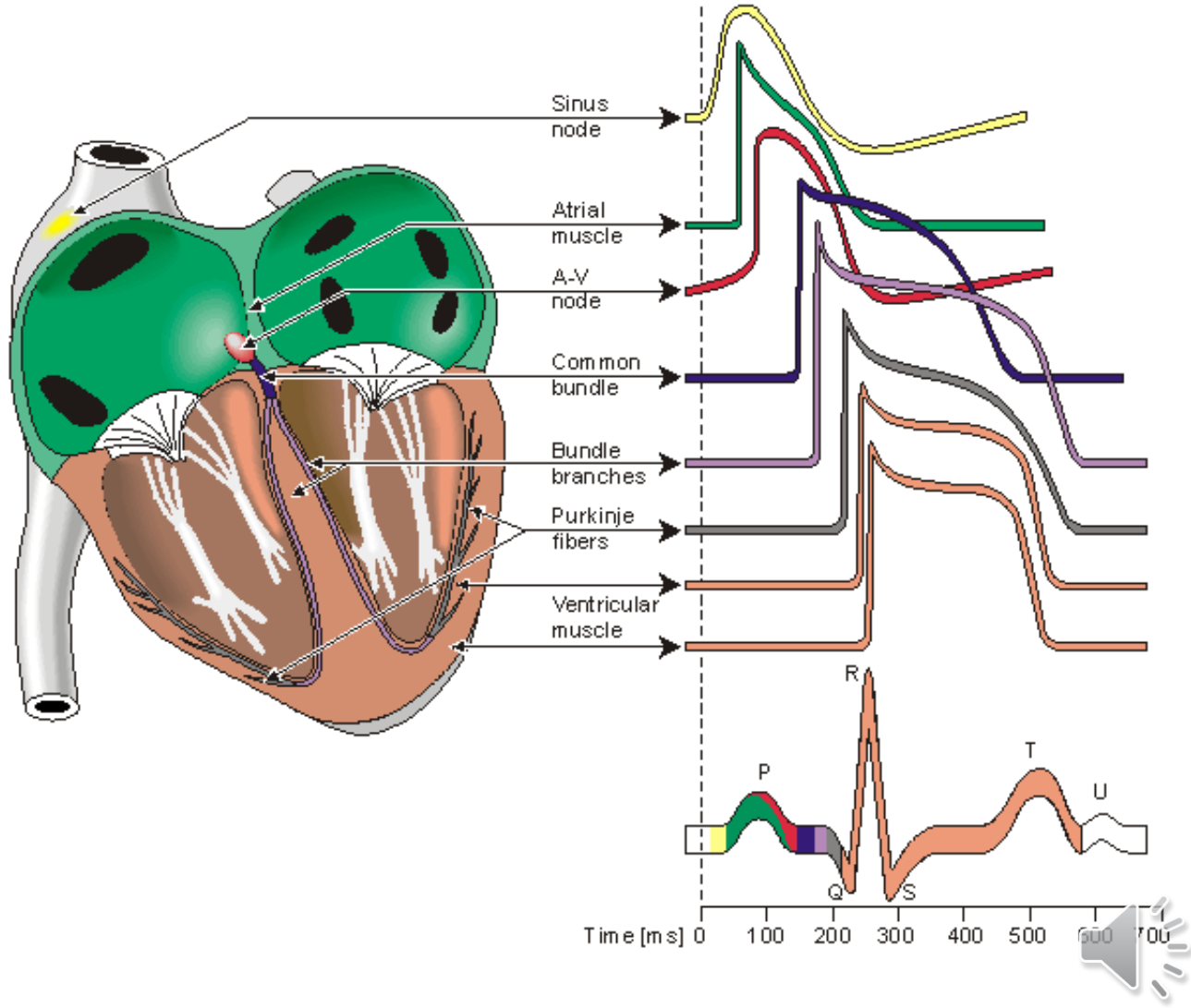
5-Cardiac enzyme levels
A-(creatine kinase, serum glutamic oxaloacetic transaminase, and lactic dehydrogenase) in the serum increase as a result of myocardial cell death. The increases occur in a characteristic pattern, beginning immediately after an infarct and continuing for about a week
B-Troponin levels become detectable in the blood within 15 to 20 minutes.
.C- Myoglobin is detected within 1 hour, peaking within 4 to 6 hours of the infarct

Electrophysiology of the heart

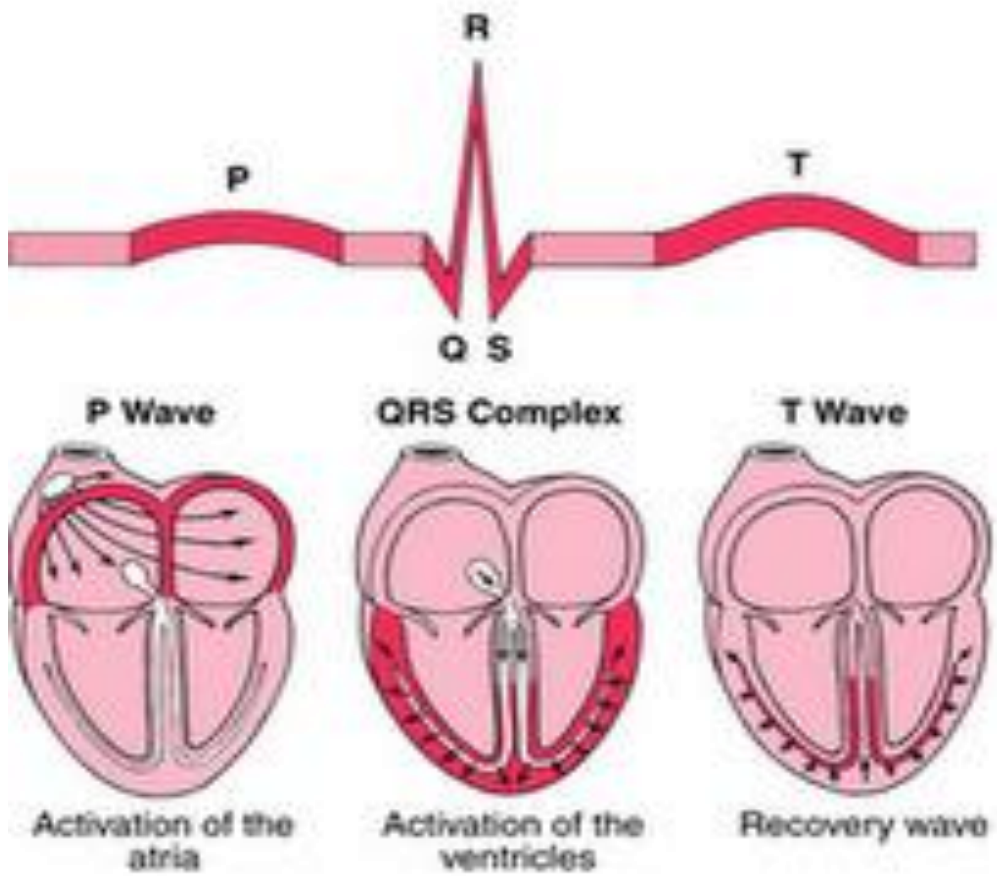
different waveforms for each of the specialized cells

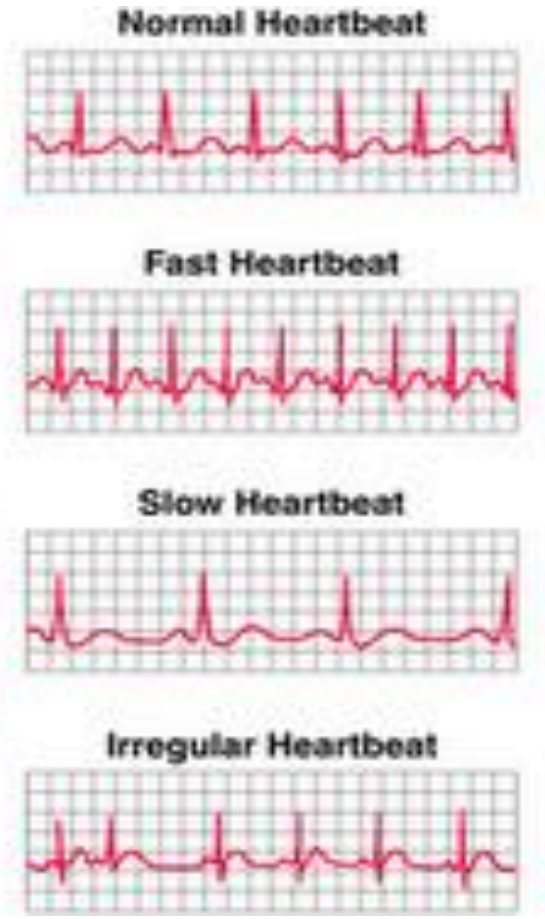
Schematic representation of the heart and normal cardiac electrical activity (intracellular recordings from areas indicated and ECG). Sinoatrial node, atrioventricular node, and Purkinje cells display pacemaker activity (phase 4 depolarization). The ECG is the body surface manifestation of the depolarization and repolarization waves of the heart.

- **The P wave is generated by atrial depolarization,**
- **the QRS by ventricular muscle depolarization, and the**
- **T wave by ventricular repolarization.**
- **Thus, the PR interval is a measure of conduction time from atrium to ventricle,**
- **the QRS duration indicates the time required for all of the ventricular cells to be activated (ie, the intraventricular conduction time).**
- **The QT interval reflects the duration of the ventricular action potential.**

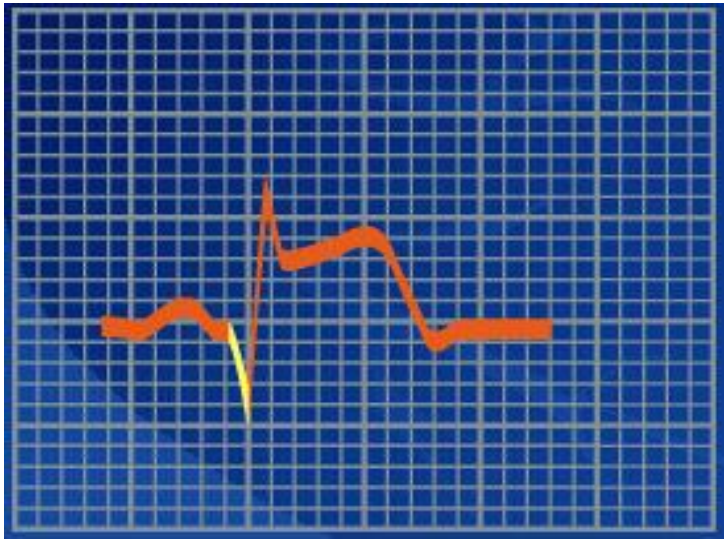


Electrophysiology of the heart





Deep Q wave



- Only diagnostic change of myocardial infarction