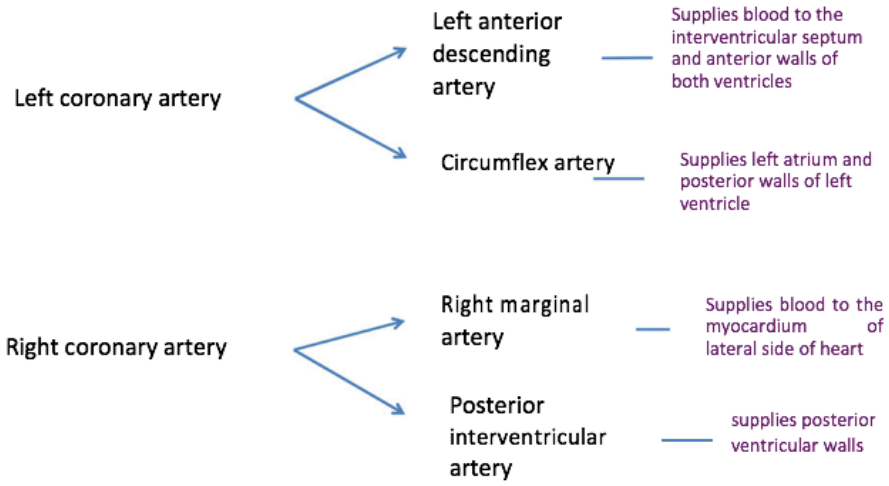
HEALTH VARIATIONS 3 NOTES

Week 1: Acute Myocardial Infarction

CORONARY CIRCULATION

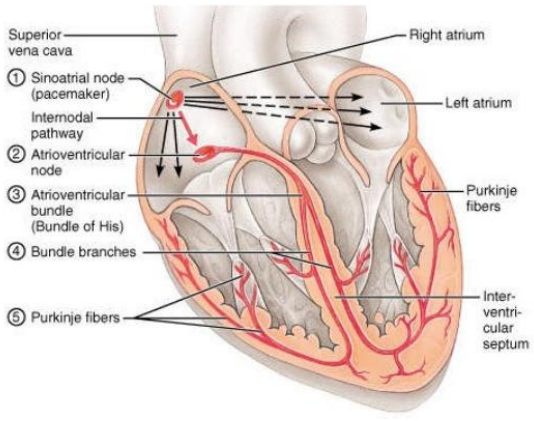
- The coronary circulation delivers nutrients and oxygen to the myocardium and removes wastes.
- The right and left coronary arteries that branch from the ascending aorta deliver oxygenated blood to the myocardium.
- The right coronary artery branches into the marginal artery and posterior interventricular artery.
- The left coronary artery branches into the circumflex and the left anterior descending artery.
- The cardiac veins collect blood from the heart muscles and empty it into the coronary sinus which returns the blood to the right atrium.

AREAS OF THE HEART SUPPLIED BY THE CORONARY ARTERIES



CONDUCTING SYSTEM

- Network of specialized tissue that stimulates contraction
- Modified cardiac myocytes
- The heart can contract without any innervation



HEALTH VARIATIONS 3 NOTES

ELECTROCARDIOGRAM

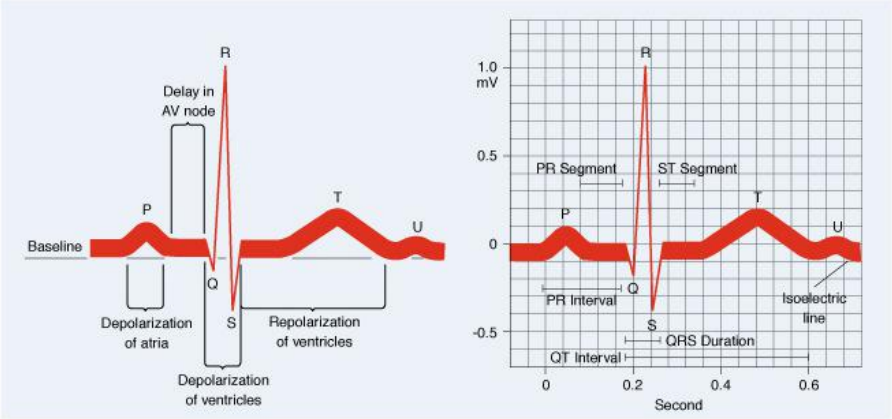


Figure 27-6 Diagram of the electrocardiogram (lead II) and representative depolarization and repolarization of the atria and ventricle. The P wave represents atrial depolarization, the QRS complex ventricular depolarization, and the T wave ventricular repolarization. Atrial repolarization occurs during ventricular depolarization and is hidden under the QRS complex.

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CORONARY ARTERY DISEASE

- Disorder of myocardial blood flow due to stable or unstable coronary atherosclerotic plaque.
- Aetiology- Atheroslecrosis is the most common cause of coronary artery disease.

ATHEROSCLEROSIS

The formation of fibrofatty lesions (atheromas or atheromatous plaques) in the intimal lining of large and medium sized arteries.

Risk factors

- Hypercholesterolemia
- Family History
- Age
- Smoking
- Obesity
- Hypertension
- Diabetes mellitus
- Serum Homocysteine
- Infectious agents

HEALTH VARIATIONS 3 NOTES

Pathogenesis

Endothelial injury

Chronic endothelial injury from

- Hyperlipidaemia (An abnormal increase in the levels of fats (lipids), including cholesterol, in the blood)
- Hypertension
- Smoking
- Homocysteine (naturally occurring amino acid found in blood plasma)
- Immune reactions

Migration of inflammatory cells

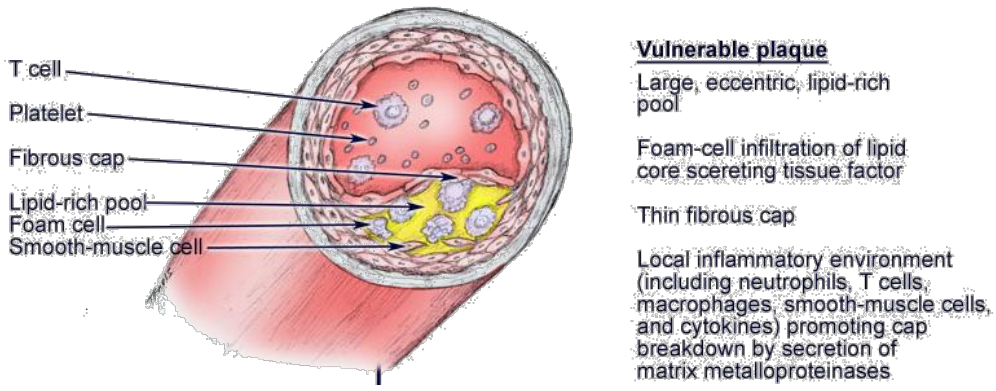
- Increased permeability → cholesterol seeps across damaged endothelium
- Monocyte and platelet adhesion
- Monocytes transform into macrophages and engulf lipoproteins

LIPID ACCUMULATION

- Macrophages ingest oxidised LDLs to become **Foam cells** causing formation of **fatty streaks**
- Macrophages produce growth factors that contribute to the migration and proliferation of smooth muscle cells

PLAQUE

- Superficial fibrous cap composed of smooth muscle cells and dense extracellular matrix
- Beneath and to side of cap – macrophages, smooth muscle cells and lymphocytes
- Below cap – central core of lipid-filled foam cells and fatty debris



Unstable plaque can rupture and cause platelet adhesion and thrombus formation → unstable angina and myocardial infarction

HEALTH VARIATIONS 3 NOTES

CLASSIFICATION OF CORONARY HEART DISEASE

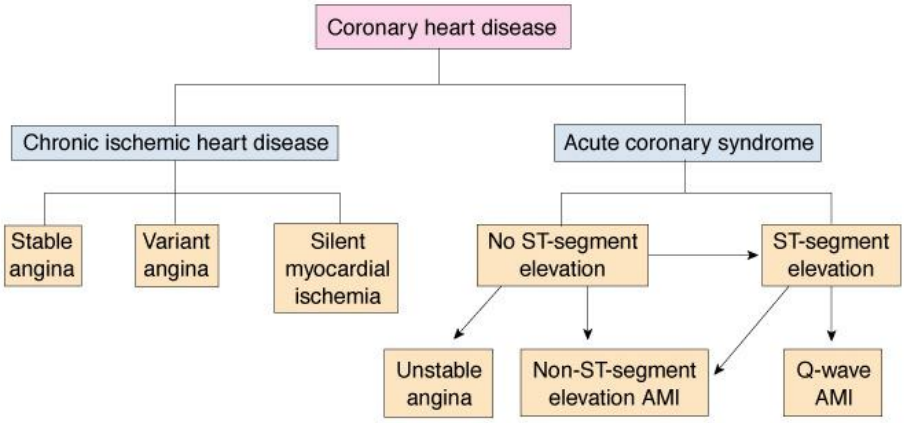


Figure 26-8 Types of coronary heart disease. AMI, acute myocardial infarction.

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CHRONIC ISCHAEMIC HEART DISEASE

1. STABLE ANGINA

- Caused by vessel narrowing and hardening of the arterial walls so that vessels cannot dilate in response to increased myocardial demand associated with physical exertion or stress.
- Pain relieved by rest and nitrates

2. VARIANT ANGINA

- Pain caused by vasospasm in one of the coronary arteries with or without atherosclerosis
- Usually occurs at rest, often between midnight and early morning
- Pathophysiological mechanisms not completely understood. May be linked to a deficiency of nitric oxide which results in enhanced activity of potent vasoconstrictors such as angiotensin ii and endothelin.

3. SILENT ISCHAEMIA

- "The presence of objective evidence of myocardial ischaemia in the absence of chest discomfort or other angina equivalents". E.g. exercise testing or ambulatory monitoring shows transient ST segment changes.