

# 1.5 Atherosclerosis, blood clotting and CVD risk

Name: \_\_\_\_\_ Class: \_\_\_\_\_ Date: \_\_\_\_\_ **Total: 20 marks**

**KEY WORDS** endothelium • thromboplastin • prothrombin  
• fibrinogen • fibrin • plaque

## Objective

Practise statements 1.10–1.13 with Core Practical 2: the course of atherosclerosis, the clotting cascade, blood pressure, and how risk factors combine.

## 1 Key knowledge and worked examples

■ **Atherosclerosis is a sequence, not an event**

Damage to the **endothelium** (smoking, high blood pressure, cholesterol) → inflammatory response → **low-density lipoproteins** invade the wall → smooth muscle and connective tissue build a **plaque** → the artery narrows and loses elasticity → thrombosis or aneurysm risk.

■ **The clotting cascade in order**

Damage exposes collagen → platelets and damaged cells release **thromboplastin** → with  $\text{Ca}^{2+}$ , thromboplastin converts **prothrombin** (soluble) to **thrombin** (enzyme) → thrombin converts **fibrinogen** (soluble) to **fibrin** (insoluble mesh) → mesh traps platelets and cells: a clot.

■ **Blood pressure and risk**

**Blood pressure** is systolic over diastolic (for example 16/10.5 kPa). Hypertension forces blood through narrowed vessels and speeds endothelial damage. Risk factors do not add – they multiply.

■ **Worked example: mean blood pressure**

A monitor reads SBP 11.0 kPa and DBP 6.7 kPa. Mean arterial pressure  $\approx \text{DBP} + (\text{SBP} - \text{DBP})/3 = 6.7 + 4.3/3 = 8.1$  kPa. This is the pressure the arterioles actually feel.

## 2 Practice

**2.1** Put these in order: fibrin forms, thromboplastin released, prothrombin converted, endothelium damaged. [2]

**2.2** Explain why a clot is more likely to form where an atherosclerotic plaque has developed. [2]

**2.3** State two ways in which the structure of an artery changes as a plaque develops. [2]

## 3 Exam-style questions

**3.1** A man has a blood pressure of 17.2/11.4 kPa and a family history of heart disease. Explain how his hypertension contributes to the development of atherosclerosis, and state why his family history counts as a risk factor even though he cannot change it. [5]

**3.2** Haemophilia reduces the conversion of prothrombin to thrombin. Explain how this leads to prolonged bleeding, referring to the molecules that are affected and the step that fails. [4]

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**3.3** A study of 10 000 people records for each participant: age, BMI, smoking, blood pressure and LDL-cholesterol. The risk of a heart attack in the next ten years is 1 % for non-smokers with normal BMI and pressure, 3 % for smokers with normal BMI and pressure, and 11 % for smokers with high BMI and high pressure. Explain what these figures show about how risk factors combine, and calculate how many times greater the combined risk is than the baseline risk. [5]

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