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 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 $\approx$ DBP + (SBP − 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]

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]

Solutions

Original-task marking: M1 method, A1 accuracy, B1 reasoning.

2.1 Endothelium damaged → thromboplastin released → prothrombin converted to thrombin → fibrin forms.

2.2 The plaque narrows the lumen, so blood flows faster and more turbulently past it, damaging the already-weakened endothelium and exposing collagen, which triggers platelet adhesion and the clotting cascade.

2.3 Any two: lumen narrows; wall loses elasticity / hardens as smooth muscle and calcium-rich plaque accumulate; endothelium damaged.

3.1 High pressure forces blood against the endothelium, damaging its cells; the damage begins the inflammatory response and lets LDL-cholesterol enter the artery wall; plaque grows, narrowing the artery further and raising pressure again – a positive feedback of deterioration. Family history indicates inherited alleles that raise cholesterol handling, blood-pressure regulation or vessel-wall susceptibility, so his baseline risk is higher whatever his behaviour.

3.2 With little thrombin formed, fibrinogen is not converted to fibrin, so no insoluble fibrin mesh forms; platelets arrive but are not bound into a stable mesh by fibrin, so the platelet plug washes away and bleeding continues.

3.3 Smoking alone multiplies the baseline risk by 3; adding high BMI and high blood pressure raises it to 11 times baseline (11%/1%, A1) – far more than the sum of the separate effects would suggest. Risk factors interact: each one multiplies the effect of the others on the same arterial system.