PIPADOR REVISION

MRCOG Part 1 Revision Notes

Coagulation and Thrombosis

PATHOLOGY • LECTURE 314

VISUAL EXAMINATION REVISION NOTES

2026 Edition

314

MRCOG PART 1 REVISION NOTES | PATHOLOGY

Coagulation and Thrombosis

Haemostasis, thrombosis and embolism, pregnancy hypercoagulability, thrombophilia, anticoagulation and DIC

Virchow	× 4–6	≈ × 20	Leading
Stasis, hyper-coagulability, injury	VTE risk in pregnancy	First 6 weeks postpartum	Direct cause of maternal death (VTE)

■ KEY FACTS: Learning objectives

- Describe normal haemostasis (primary, secondary, fibrinolysis) and natural anticoagulants.
- Describe thrombosis (Virchow's triad), embolism and the fate of thrombi.
- Describe pregnancy hypercoagulability, thrombophilias, DIC and anticoagulation in pregnancy.

314.1 Normal haemostasis

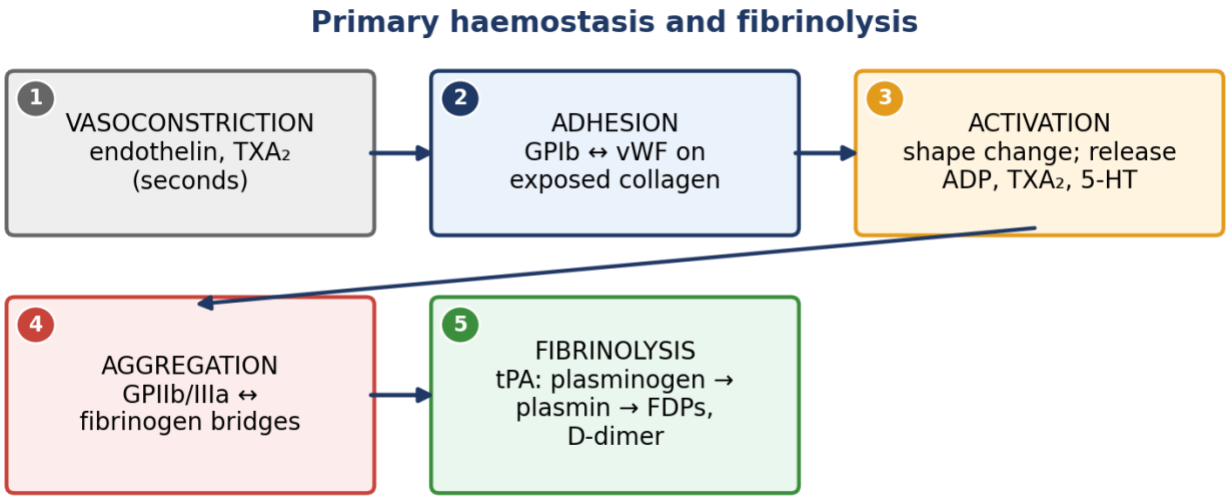


Figure 314.1 — Primary haemostasis and fibrinolysis

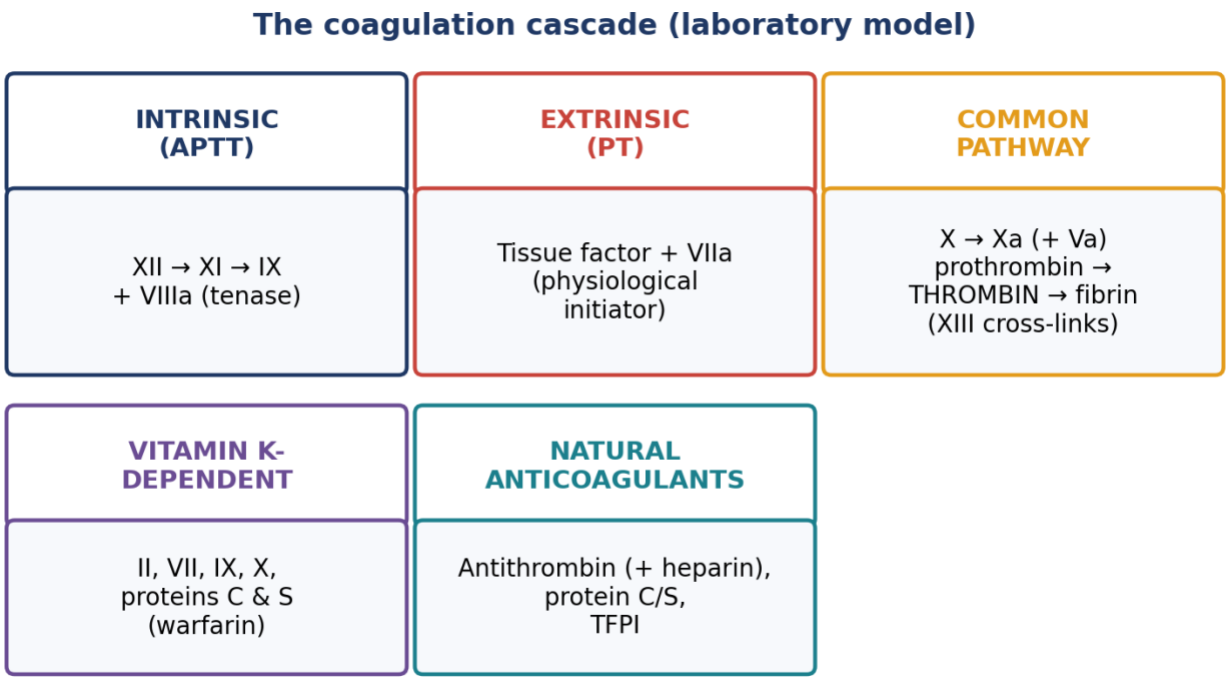


Figure 314.2 — The coagulation cascade

- In vivo, coagulation follows the **cell-based model**: tissue factor–VIIa initiates on cell surfaces, small amounts of thrombin amplify by activating platelets and cofactors V and VIII, and propagation on platelet surfaces generates a thrombin burst.
- Natural anticoagulants (antithrombin, protein C/S, TFPI) and fibrinolysis (plasmin) confine clotting to the site of injury.

314.2 Thrombosis

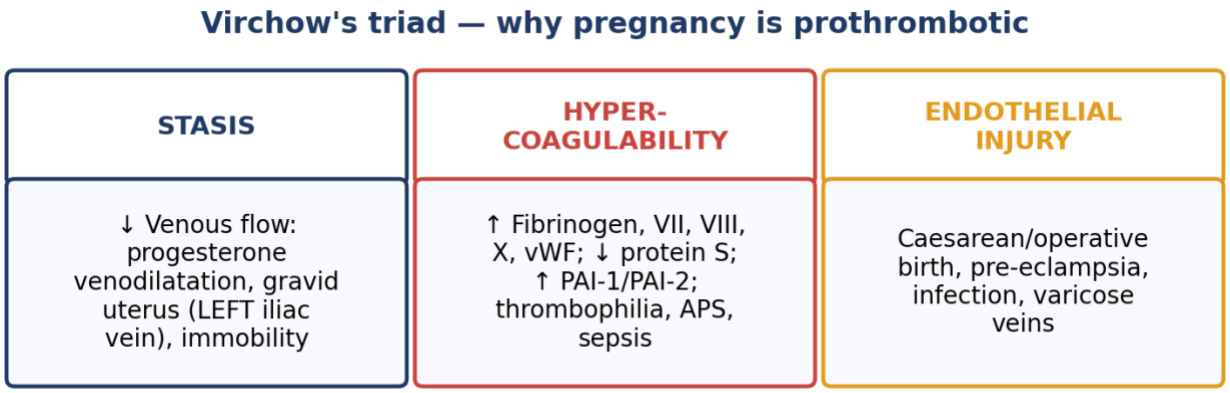


Figure 314.3 — Virchow's triad in pregnancy

Feature	Arterial thrombus	Venous thrombus
Main driver	Endothelial injury (atheroma rupture)	Stasis + hypercoagulability
Composition	Platelet-rich ('white'), lines of Zahn	Red cell/fibrin-rich ('red')
Consequence	Infarction (stroke, MI)	DVT → pulmonary embolism
Prevention	Antiplatelets (aspirin)	Anticoagulants (LMWH)

- Fates of a thrombus: propagation, embolisation, dissolution (fibrinolysis), organisation and recanalisation (post-thrombotic syndrome).
- Other emboli in obstetrics: **amniotic fluid embolism** (anaphylactoid, DIC), air embolism, fat embolism.

314.3 Pregnancy and thrombophilia

Topic	Details
Pregnancy changes	• ↑ Fibrinogen, VII, VIII, X, vWF • ↓ protein S • Acquired APC resistance • ↓ fibrinolysis (PAI-1, placental PAI-2) — protects against PPH, ↑ VTE
Left-sided DVT	• ≈ 80–90% left-sided (right iliac artery compresses left iliac vein) • Often iliofemoral
Inherited thrombophilia	Factor V Leiden, prothrombin G20210A, deficiencies of antithrombin (highest risk), protein C, protein S
Acquired	Antiphospholipid syndrome, nephrotic syndrome, malignancy, obesity, immobility, IVF/OHSS, dehydration (hyperemesis), sepsis, caesarean
Prevention (RCOG GTG 37a)	• Risk assessment at booking, admission and postpartum • LMWH thromboprophylaxis by score • Mobilisation, hydration, stockings
Treatment (GTG 37b)	• Therapeutic **LMWH** (weight-based) until ≥ 6 weeks postpartum and ≥ 3 months total • Warfarin avoided (embryopathy 6–12 wk) but OK postnatally/breastfeeding • DOACs avoided in pregnancy and breastfeeding

314.4 Disseminated intravascular coagulation

- Widespread activation of coagulation → microvascular thrombi + **consumption** of platelets and factors → bleeding.
- Obstetric triggers: **placental abruption**, amniotic fluid embolism, massive PPH, **pre-eclampsia/HELLP**, sepsis, retained dead fetus, AFLP.
- Labs: ↓ platelets, ↓ fibrinogen (< 2 g/L predicts severe PPH), ↑ PT/APTT, ↑ D-dimer
  - Treat the cause, replace (FFP, cryoprecipitate/fibrinogen concentrate, platelets), tranexamic acid in PPH.

314.5 Must-know summary

■ **EXAM TIP**

- Primary (platelets: adhesion via vWF/GPIb, aggregation via GPIIb/IIIa) → secondary (thrombin, fibrin) → fibrinolysis (plasmin).
- Virchow's triad; pregnancy is hypercoagulable (↑ fibrinogen, VIII, vWF; ↓ protein S).
- VTE risk highest postpartum; left-sided DVT; LMWH for prophylaxis and treatment; no warfarin in 1st trimester, no DOACs.
- DIC: abruption, AFE, PPH, HELLP, sepsis — low fibrinogen.

■ **MNEMONIC / MEMORY AID**

Virchow — 'SHE': Stasis, Hypercoagulability, Endothelial injury.