PIPADOR REVISION

MRCOG Part 1 Revision Notes

Placental Physiology

PHYSIOLOGY • LECTURE 158

VISUAL EXAMINATION REVISION NOTES

2026 Edition

158

MRCOG PART 1 REVISION NOTES | PHYSIOLOGY

Placental Physiology

Structure, circulations, transfer and drugs, endocrine and immunological functions, and placental disease

≈ 500 g	≈ 11–13 m²	500–750 mL/min	2 + 1
Term placenta (≈ 22 cm diameter)	Villous exchange surface at term	Uteroplacental blood flow at term	Umbilical arteries + vein

■ KEY FACTS: Learning objectives

- Describe placental structure and the maternal and fetal circulations.
- Describe mechanisms of placental transfer and factors affecting it, including drugs.
- Describe placental endocrine and immunological functions.
- Relate placental physiology to pre-eclampsia, FGR and other placental disorders.

158.1 Structure

Feature	Detail
Type	Haemochorial — maternal blood directly bathes the syncytiotrophoblast; villous; discoid
Villous tree	Stem → intermediate → terminal villi (gas exchange; vasculosyncytial membranes); anchoring villi attach to the basal plate (cytotrophoblast shell); floating villi in the intervillous space
Syncytiotrophoblast	Multinucleate, no cell borders; transport, endocrine (hCG, hPL, steroids), formed by fusion of cytotrophoblast
Cytotrophoblast	Stem cells; gives rise to extravillous trophoblast (invasion, spiral artery remodelling); sparse at term
Barrier thickness	≈ 20–30 µm early → ≈ 4–5 µm at term (↑ diffusion)
Gross	15–20 cotyledons (maternal surface); chorionic plate (fetal); umbilical cord ≈ 50–60 cm: 2 arteries, 1 vein, Wharton's jelly; single umbilical artery ≈ 1% (renal/cardiac anomalies, FGR)

158.2 Circulations

≈ 80–100 **spiral arteries** (remodelled — low resistance, non-vasoactive) ▶ Jets into **intervillous space** (≈ 150 mL; exchanged 2–3×/min) ▶ Flows over villi (multivillous 'pool' arrangement) ▶ Drains via basal-plate veins ▶ **Not autoregulated** — flow falls with maternal hypotension and contractions

Fetal side: 2 umbilical arteries (deoxygenated, from internal iliacs) → chorionic arteries → villous capillaries → **umbilical vein** (oxygenated) → ductus venosus (Lecture 128). Fetoplacental flow ≈ 40% of combined ventricular output; the fetal placental vascular bed is regulated by local NO and lacks autonomic innervation.

158.3 Placental transfer

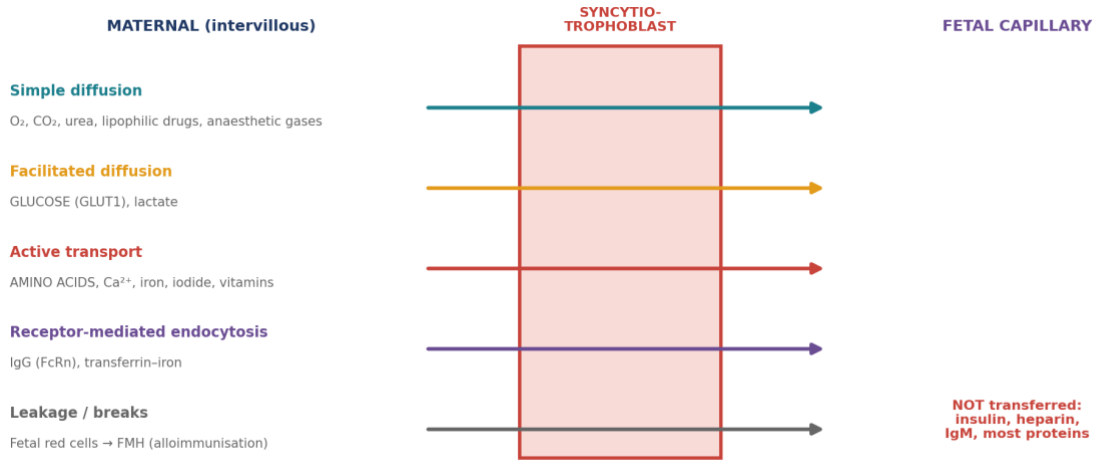


Figure 158.1 — Mechanisms of placental transfer

■ KEY EQUATIONS

Fick's law: transfer ∝ (surface area × concentration gradient) ÷ membrane thickness — plus **blood flow** on both sides (O_2 and CO_2 transfer is **flow-limited**).

Drug transfer favoured by: **low molecular weight (< 500 Da)**, **lipid solubility**, **un-ionised** form, low protein binding. Fetal pH is lower → weak bases (LAs, pethidine) become ionised and **trapped** ('ion trapping') in acidosis.

Crosses readily	Crosses poorly / not at all
Warfarin (embryopathy), phenytoin, valproate, ACEi/ARBs, tetracyclines, lithium, antithyroid drugs (PTU, carbimazole), most anaesthetic agents, opioids, β-blockers, corticosteroids (betamethasone/dexamethasone — 11β-HSD2 resistant)	Heparins (UFH, LMWH), insulin, suxamethonium and non-depolarising muscle relaxants (ionised), IgM, prednisolone/hydrocortisone (largely inactivated by 11β-HSD2), thyroxine (minimal)

158.4 Endocrine functions

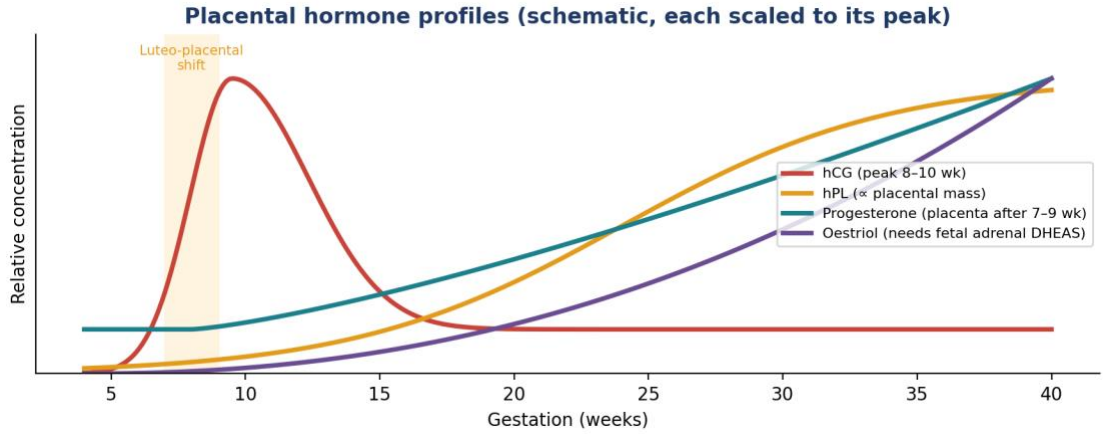


Figure 158.2 — Placental hormone profiles across gestation

Hormone	Key actions
hCG	Rescues corpus luteum; TSH-like; fetal testosterone (Leydig) — Lecture 145
hPL (hCS)	Maternal insulin resistance and lipolysis → glucose and FFAs for fetus; mammary development; ∝ placental mass
Progesterone	Synthesised from maternal LDL-cholesterol (no fetal precursors needed); uterine quiescence, immune modulation; placenta takes over at 7–9 weeks

Hormone	Key actions
Oestrogens (oestriol ≫)	Fetoplacental unit: fetal adrenal DHEAS → fetal liver 16-hydroxylation → placental sulphatase + aromatase → oestriol. ↓ in fetal adrenal hypoplasia, anencephaly, placental sulphatase deficiency (X-linked ichthyosis — delayed labour), trisomy 21
CRH	Placental clock of parturition (Lecture 147)
Placental GH, leptin, kisspeptin, relaxin	Maternal metabolic adaptation; IGF-1 regulation
PlGF / sFlt-1	Angiogenic balance — sFlt-1 ↑, PlGF ↓ in pre-eclampsia

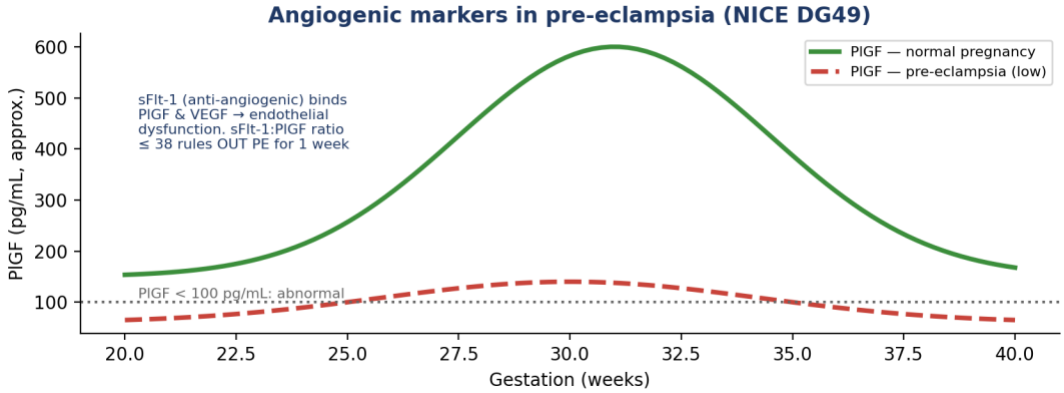


Figure 158.3 — PlGF in normal pregnancy and pre-eclampsia

158.5 Immunological and barrier functions

Trophoblast HLA-C/E/G expression, uterine NK cells and regulatory T cells mediate **tolerance** (Lecture 155). The placenta transfers **IgG** (FcRn), metabolises maternal cortisol (**11β-HSD2** — protects the fetus from glucocorticoid excess; deficiency linked to FGR) and limits some pathogens — but **TORCH organisms, Listeria, malaria, Zika, parvovirus** can cross.

158.6 Placental disorders

Disorder	Physiology
Pre-eclampsia	Failed spiral artery remodelling → ischaemic, oxidatively stressed placenta → sFlt-1, sEng release → maternal endothelial dysfunction; aspirin from 12 wk for high risk; PlGF-based testing (NICE DG49)
FGR	Placental insufficiency → reduced villous surface, abnormal Doppler (Lecture 146)
Abruption	Decidual haemorrhage → separation; hypertension, cocaine, trauma, smoking; DIC
Placenta praevia / accreta spectrum	Low implantation; absent decidua (previous CS scar) → invasion into myometrium
Chorioamnionitis	Ascending infection; PPROM, preterm labour, neonatal sepsis, cerebral palsy
Gestational trophoblastic disease	Molar pregnancy — very high hCG (Lecture 145)
Monochorionic placentation	Vascular anastomoses → TTTS, TAPS, sIUGR

158.7 Must-know summary

■ EXAM TIP

- Haemochorial placenta; barrier thins to **≈ 4–5 µm**; surface ≈ 11–13 m^2; uteroplacental flow **not autoregulated**.
- Diffusion: O_2, CO_2; facilitated: **glucose (GLUT1)**; active: **amino acids, Ca^{2+}, iron**; endocytosis: **IgG**.
- **Heparin and insulin do not cross**; warfarin, antithyroid drugs, ACEi do. 11β-HSD2 inactivates cortisol/prednisolone but not betamethasone.
- Oestriol requires **fetal adrenal DHEAS** + placental sulphatase/aromatase.
- Pre-eclampsia: **↑ sFlt-1, ↓ PlGF**.

■ MNEMONIC / MEMORY AID

Placental functions — 'GRIEF': Gas exchange, Removal of waste, Immunity (IgG), Endocrine, Food (nutrients).