PIPADOR REVISION

MRCOG Part 1 Revision Notes

Fetal Growth

PHYSIOLOGY • LECTURE 146

VISUAL EXAMINATION REVISION NOTES

2026 Edition

MRCOG PART 1 REVISION NOTES | PHYSIOLOGY

146 Fetal Growth

Growth patterns, determinants, hormones, assessment, SGA/FGR, macrosomia and fetal programming

≈ 3.5 kg	< 10th	< 3rd	≈ 200–250 g
Mean term birthweight (UK)	SGA (EFW/AC centile)	Severe SGA — FGR regardless of Doppler	Weight gain per week at 33–36 wk

■ KEY FACTS: Learning objectives

- Describe the phases and velocity of fetal growth and body composition.
- Describe genetic, maternal, placental and hormonal determinants of fetal growth.
- Describe methods of assessing fetal growth and definitions of SGA, FGR and LGA.
- Distinguish early and late FGR and outline causes, consequences and the developmental origins of adult disease.

146.1 Patterns of fetal growth

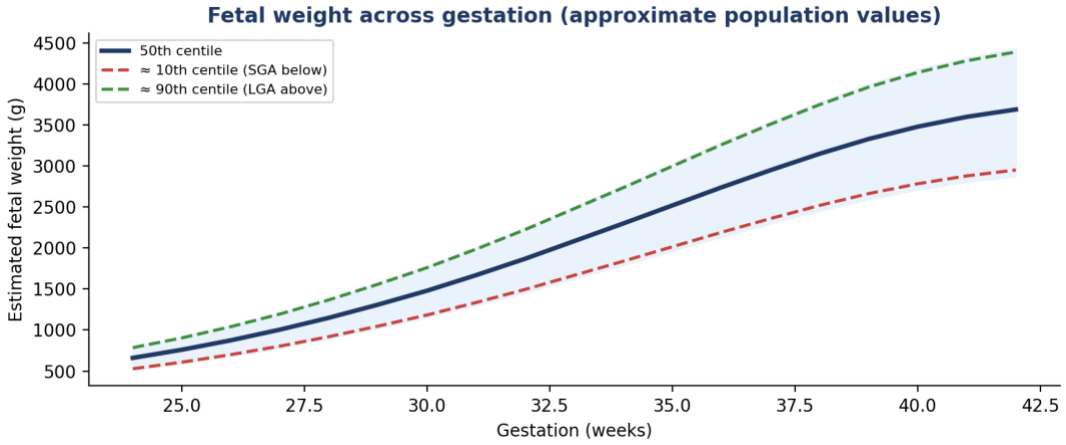


Figure 146.1 — Approximate fetal weight centiles

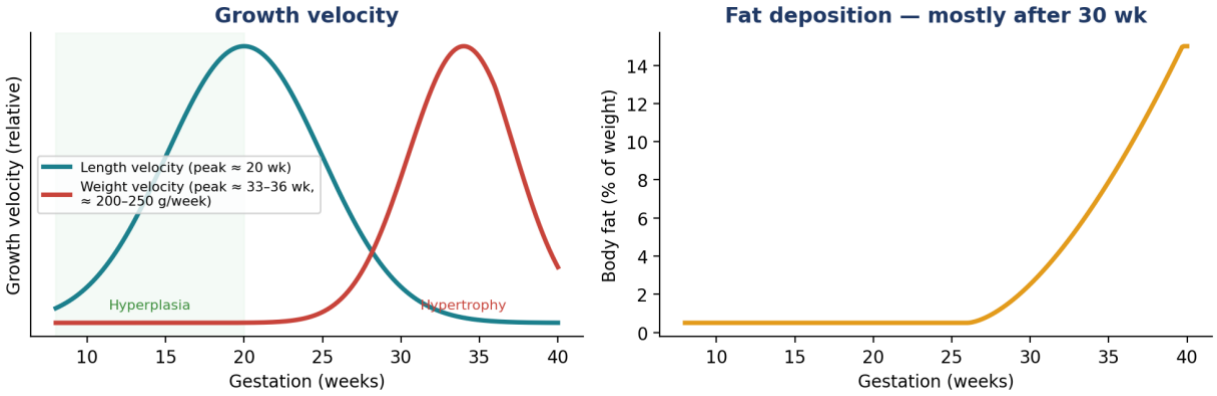


Figure 146.2 — Length and weight velocity; fat deposition

Phase	Gestation	Cellular growth	Effect of an insult
1. Hyperplasia	to ≈ 16–20 wk	↑ Cell number	Symmetrical small fetus (fewer cells): aneuploidy, infection, drugs — poorer catch-up

Phase	Gestation	Cellular growth	Effect of an insult
2. Hyperplasia + hypertrophy	≈ 20–32 wk	Both	Mixed
3. Hypertrophy	> 32 wk	↑ Cell size; fat and glycogen deposition	Asymmetrical (head-sparing): placental insufficiency — ↓ liver/fat, small AC

Length velocity peaks in the **second trimester**; weight velocity peaks at **≈ 33–36 weeks**. Body fat rises from ≈ 1% at mid-gestation to **≈ 12–15% at term** (insulin-dependent) — the human neonate is unusually fat (brain energy reserve).

146.2 Determinants of fetal growth

Category	Factors
Genetic	Fetal genome, sex (boys ≈ 150 g heavier), ethnicity; imprinted genes — paternally expressed IGF2 promotes growth, maternally expressed IGF2R/H19 restrains it (Beckwith–Wiedemann ↑; Silver–Russell ↓)
Maternal	Maternal constraint (uterine size — Shire × Shetland horse crosses), height, BMI, parity, age, nutrition, altitude, smoking, alcohol, drugs, hypertension, diabetes, renal disease, APS
Placental	Uteroplacental blood flow (spiral artery remodelling), villous surface area, transporter capacity (GLUT1, amino acid system A), placental size
Fetal	Aneuploidy (T13, T18, triploidy), syndromes, infection (CMV, toxoplasma, rubella, syphilis, malaria, Zika), multiple pregnancy

Hormone	Role in fetal growth
Insulin	Key 3rd-trimester growth hormone (fat, glycogen, muscle). Fetal hyperinsulinaemia → macrosomia (Pedersen hypothesis); insulin deficiency (pancreatic agenesis) → severe FGR
IGF-2	Main growth factor in early gestation and for placental growth
IGF-1	Later gestation; regulated by fetal nutrient (glucose/insulin) supply — ↓ in FGR
Growth hormone	Little role in fetal growth (anencephalic and GH-deficient fetuses grow nearly normally); important postnatally
Thyroid hormone	Maturation (brain, bone) rather than size
Cortisol	Maturation (lungs, liver, gut); excess restrains growth (11β-HSD2 placental barrier protects fetus)
Leptin, hPL, placental GH	Maternal nutrient partitioning (insulin resistance) → more glucose to fetus

146.3 Assessing fetal growth

Method	Use
CRL (11+2 to 14+1 wk; 45–84 mm)	Most accurate dating; HC if CRL > 84 mm
Symphysis–fundal height	From 24 wk, measured and plotted serially (customised or population chart); cm ≈ weeks ± 2 — refer for scan if < 10th centile or static/slow growth
Ultrasound biometry	BPD, HC, AC (best single predictor of SGA/macrosomia), FL → EFW (Hadlock; error ≈ ± 10–15%); interval ≥ 2 weeks between scans
Doppler	Umbilical artery (primary surveillance in SGA), MCA and cerebroplacental ratio (late FGR), ductus venosus (early FGR timing), uterine artery (risk in 2nd trimester)
Liquor volume	Deepest pool < 2 cm — oligohydramnios (↓ renal perfusion)
CTG / cCTG	Short-term variation (Dawes–Redman) in early FGR

■ EXAM TIP

- SGA (< 10th centile) ≠ FGR: many SGA fetuses are **constitutionally small**. FGR = failure to reach growth potential (Doppler abnormality, slowing growth, < 3rd centile).
- Saving Babies' Lives (NHS England) and **RCOG GTG 31** guide risk assessment, aspirin for high-risk women, serial scans and delivery timing.

146.4 Early versus late FGR

	Early FGR (< 32 wk)	Late FGR (≥ 32 wk)
Prevalence	≈ 20–30% of FGR	≈ 70–80% of FGR
Placenta	Severe — failed spiral artery remodelling	Milder dysfunction
Association	Pre-eclampsia (≈ 50%)	Less often
Doppler	UA PI ↑ → AEDF/REDF, then DV abnormality	UA often normal; ↓ MCA PI, ↓ CPR
Challenge	Management (timing delivery vs prematurity)	Detection — causes many term stillbirths
Delphi definition	AC/EFW < 3rd, or UA AEDF, or AC/EFW < 10th + abnormal UtA/UA Doppler	AC/EFW < 3rd, or ≥ 2 of: < 10th centile, crossing > 2 quartiles, CPR < 5th / UA PI > 95th

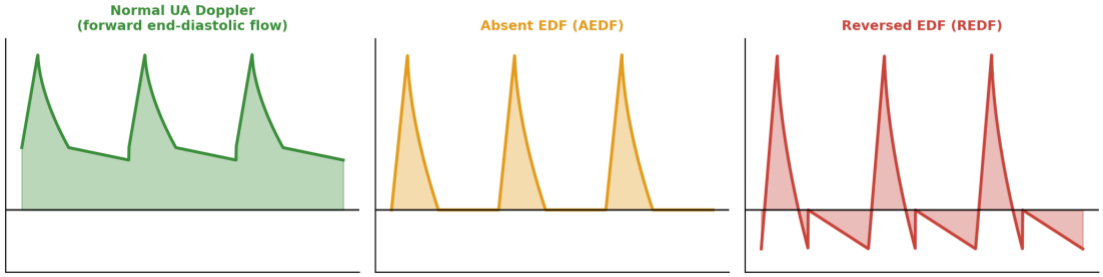


Figure 146.3 — Umbilical artery Doppler deterioration in placental FGR

Placental insufficiency ▶ ↓ O_2 & nutrients → **↓ insulin/IGF-1** ▶ Glycogen and fat depletion (↓ AC); **brain-sparing** ▶ ↓ Renal perfusion → **oligohydramnios** ▶ Hypoxaemia → acidosis → stillbirth

146.5 Large for gestational age and macrosomia

LGA > 90th centile; **macrosomia** > 4000 g (or > 4500 g). Causes: **maternal diabetes** (fetal hyperinsulinaemia — disproportionate growth of insulin-sensitive tissues: shoulders, abdomen, liver), obesity, excessive weight gain, multiparity, post-term, Beckwith–Wiedemann. Risks: **shoulder dystocia**, perineal trauma, PPH, neonatal hypoglycaemia, childhood obesity.

146.6 Developmental origins of health and disease

The **Barker hypothesis**: low birthweight (and rapid postnatal catch-up) is associated with adult **hypertension, coronary heart disease, type 2 diabetes** and metabolic syndrome — **fetal programming** via altered organ structure (↓ nephron number), endocrine set-points (HPA axis) and **epigenetics**. The Dutch Hunger Winter cohort showed effects depending on the trimester of famine exposure.

146.7 Must-know summary

■ KEY FACTS

- Hyperplasia (early) → hypertrophy (late); **weight velocity peaks ≈ 33–36 wk**; fat ↑ to ≈ 15% at term.
- **Insulin** is the main 3rd-trimester growth hormone; **IGF-2** early; **GH has little role** in utero.
- **AC** is the best single biometric measure; SFH plotted serially from 24 wk.
- Early FGR: UA Doppler, pre-eclampsia, management problem. Late FGR: CPR, detection problem.
- Symmetrical SGA → intrinsic (aneuploidy, infection); asymmetrical → placental insufficiency.

■ MNEMONIC / MEMORY AID

Causes of FGR — 'Mother, Placenta, Fetus'; infections — '**TORCH + malaria**'. **IGF2: 'Paternal genes Push growth; maternal genes Moderate'**.