PIPADOR REVISION

MRCOG Part 1 Revision Notes

Metabolism During Starvation

BIOCHEMISTRY • LECTURE 238

VISUAL EXAMINATION REVISION NOTES

2026 Edition

238

MRCOG PART 1 REVISION NOTES | BIOCHEMISTRY

Metabolism During Starvation

Hormonal control and phases of fasting, organ roles and protein sparing, accelerated starvation of pregnancy, hyperemesis, the neonate and refeeding syndrome

≈ 24 h	Day 2–3	≈ 2/3	≈ 12–18 h
Liver glycogen exhausted	Ketogenesis rises	Brain fuel from ketones (weeks)	Ketosis onset in late pregnancy

■ KEY FACTS: Learning objectives

- Describe the hormonal control and phases of metabolism from the fed state to prolonged starvation.
- Describe the roles of liver, muscle, adipose tissue and brain, and protein sparing.
- Describe 'accelerated starvation' in pregnancy, starvation in labour and hyperemesis, the fetus/neonate, and refeeding syndrome.

238.1 Hormonal control

Hormone	Fed state	Fasting / starvation
Insulin	↑ — glycogenesis, lipogenesis, protein synthesis, GLUT4 uptake	↓ — permits lipolysis, ketogenesis
Glucagon	↓	↑ — glycogenolysis, gluconeogenesis, ketogenesis (liver)
Cortisol, adrenaline, GH	Low	↑ — lipolysis, gluconeogenesis, protein breakdown
Thyroid (T_3)	Normal	↓ (rT_3 ↑) — ↓ BMR (energy conservation)
Leptin	Normal	↓ — ↑ appetite, ↓ GnRH (amenorrhoea), ↓ thyroid

238.2 Phases of fasting

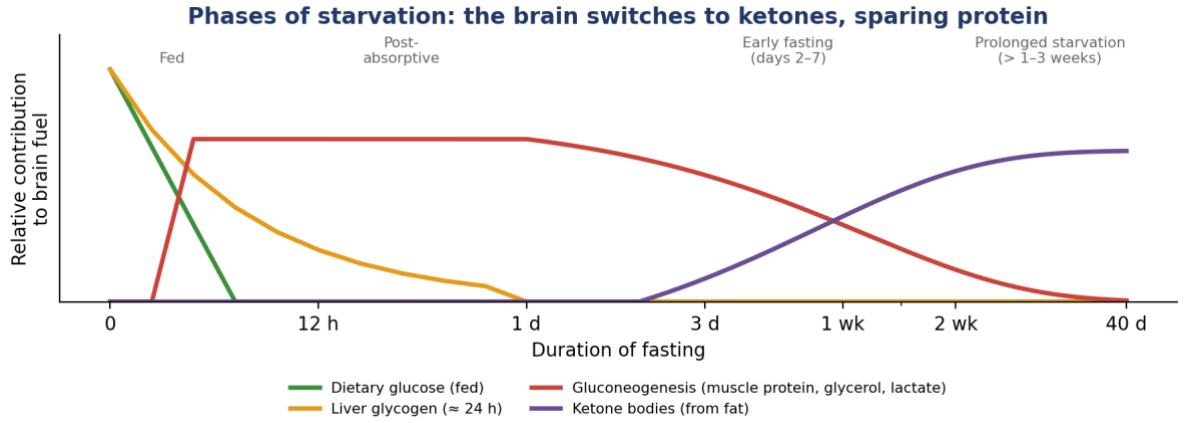


Figure 238.1 — Fuel sources during fasting and starvation

Phase	Timing	Metabolism
Fed (absorptive)	0–4 h	Glucose from diet; insulin → storage (glycogen, TG, protein)

Phase	Timing	Metabolism
Post-absorptive	4–24 h	Hepatic glycogenolysis maintains glucose; gluconeogenesis begins; FFAs for muscle
Early fasting	Days 2–7	Glycogen gone → gluconeogenesis from muscle amino acids (alanine, glutamine), glycerol, lactate; ↑ lipolysis → ketogenesis rising; negative N balance (≈ 75 g protein/day)
Prolonged starvation	> 1–3 weeks	Brain adapts to ketones (≈ ⅔ of fuel) → glucose need falls → protein sparing (≈ 20 g/day); kidney contributes gluconeogenesis; ↓ BMR (↓ T_3); fat stores determine survival (≈ 2 months)
Terminal	Fat exhausted	Accelerated protein breakdown (respiratory/cardiac muscle) → death

■ KEY FACTS

- Organ roles: **liver** (glucose and ketone producer — cannot use ketones), **muscle** (amino acid donor; uses FFAs/ketones), **adipose** (FFAs + glycerol), **brain** (glucose → ketones), **RBCs** (glucose only — anaerobic; lactate recycled by Cori cycle).
- Even-chain fatty acids cannot make glucose; only **glycerol** and amino acids (and odd-chain FA) are gluconeogenic.

238.3 Starvation in pregnancy

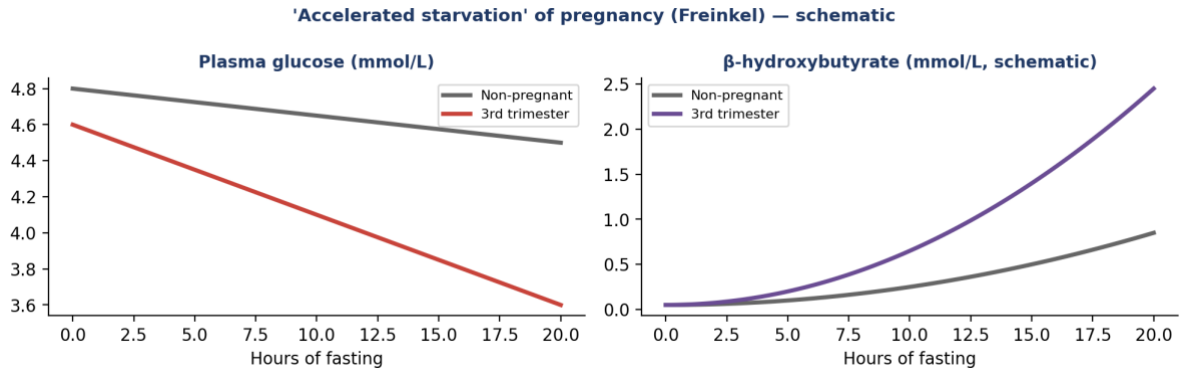


Figure 238.2 — Accelerated starvation of pregnancy

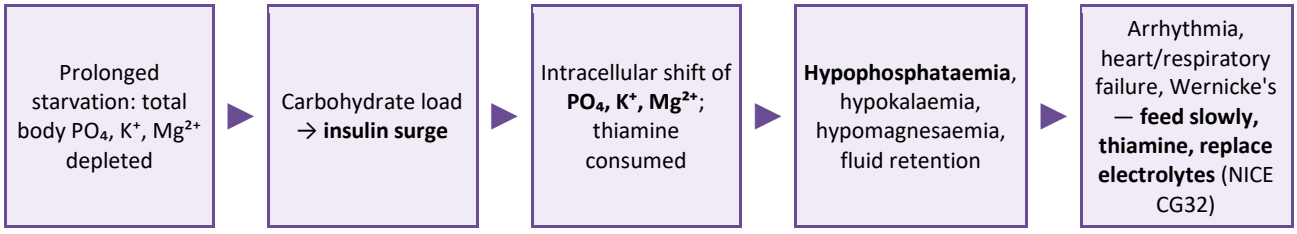
Situation	Physiology / management
Accelerated starvation (Freinkel)	Continuous fetal glucose uptake, ↑ plasma volume and insulin resistance → after an overnight fast pregnant women have lower glucose, higher FFAs and ketones — ketosis within 12–18 h; avoid prolonged fasting, frequent small meals, bedtime snack in diabetes
Hyperemesis gravidarum	Starvation ketosis, dehydration, hypokalaemia, hyponatraemia, metabolic alkalosis, weight loss > 5%; thiamine before dextrose (Wernicke's); refeeding risk in severe cases (RCOG GTG 69)
Fasting in labour / before CS	Ketosis, ↑ lactate; clear fluids/isotonic drinks permitted in low-risk labour; dextrose infusions can cause maternal hyperglycaemia → fetal hyperinsulinism → neonatal hypoglycaemia and hyponatraemia (avoid large hypotonic volumes)
Ramadan fasting	Generally tolerated by healthy women; risks of dehydration and ketosis — individualised advice; exemption in pregnancy permitted
Eating disorders / severe undernutrition	FGR, preterm birth, micronutrient deficiencies; refeeding syndrome
Diabetes	Insulin-treated women: DKA at near-normal glucose with vomiting/fasting; sick-day rules

238.4 Fetus and neonate

- Maternal starvation: the fetus uses **maternal ketones** and lactate; severe/prolonged deprivation → **FGR**, programming (Dutch Hunger Winter — DOHaD).

- At birth the neonate experiences abrupt 'starvation': **glucagon and catecholamine surge** → glycogenolysis, then gluconeogenesis and ketogenesis (breastfed infants use ketones in the first days); **hypoglycaemia** risk in preterm, FGR (low glycogen) and infants of diabetic mothers (hyperinsulinism).

238.5 Refeeding syndrome



238.6 Must-know summary

■ EXAM TIP

- Fasting: ↓ insulin, ↑ glucagon/cortisol/catecholamines; glycogen lasts ≈ 24 h; then gluconeogenesis (muscle protein) and ketogenesis.
- Prolonged starvation: **brain switches to ketones → protein sparing**; ↓ T_3.
- Pregnancy: **accelerated starvation** — ketosis within 12–18 h; hyperemesis → thiamine first; DKA at lower glucose.
- Neonate: glucagon/catecholamine surge; hypoglycaemia in preterm, FGR, IDM.
- Refeeding: **hypophosphataemia**; slow feeding + thiamine.

■ MNEMONIC / MEMORY AID

Starvation sequence — 'Glycogen, Gluconeogenesis, then Ketones' (the 3 Gs → K).