PIPADOR REVISION

MRCOG Part 1 Revision Notes

Carbohydrates: Anaerobic Metabolism

BIOCHEMISTRY • LECTURE 214

VISUAL EXAMINATION REVISION NOTES

2026 Edition

MRCOG PART 1 REVISION NOTES | BIOCHEMISTRY

214 Carbohydrates: Anaerobic Metabolism

Anaerobic glycolysis and NAD⁺ regeneration, the Cori cycle, lactic acidosis, and fetal/neonatal lactate

2 ATP	≈ 30–32	LDH	> 4 mmol/L
Per glucose (anaerobic)	ATP per glucose (aerobic)	Pyruvate → lactate; regenerates NAD⁺	Lactate — significant (sepsis marker ≥ 2)

■ KEY FACTS: Learning objectives

- Describe anaerobic glycolysis, the role of lactate dehydrogenase and NAD⁺ regeneration.
- Describe tissues dependent on anaerobic metabolism and the Cori cycle.
- Describe lactic acidosis and its causes, and fetal/neonatal anaerobic metabolism (lactate, base deficit).

214.1 Anaerobic glycolysis

Glucose → 2 pyruvate (glycolysis) ▶ + 2 ATP, + 2 NADH ▶ No O_2 / no mitochondria → NADH cannot enter oxidative phosphorylation ▶ **Lactate dehydrogenase**: pyruvate + NADH → **lactate + NAD⁺** ▶ NAD⁺ regenerated → glycolysis continues (GAPDH step)

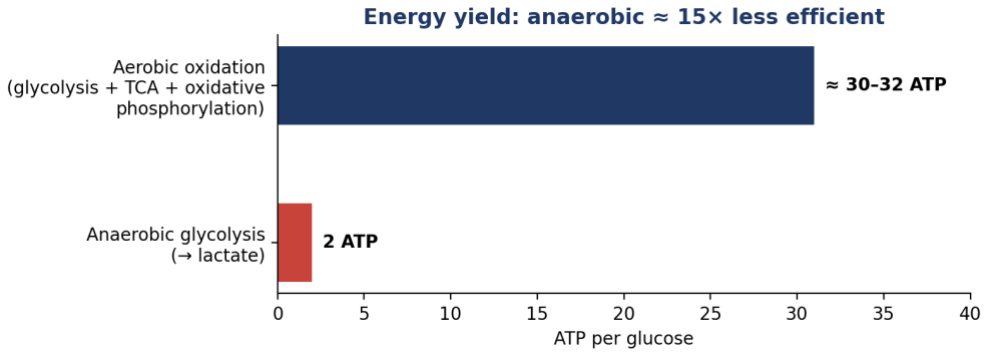


Figure 214.1 — ATP yield: anaerobic vs aerobic

Tissue	Reason for anaerobic metabolism
Red blood cells	No mitochondria — glycolysis only (+ 2,3-BPG shunt, pentose phosphate pathway for NADPH)
Exercising skeletal muscle (fast-twitch)	O_2 supply exceeded; rapid ATP
Lens, cornea, renal medulla, testis	Low O_2/vascularity
Hypoxic tissue	Shock, haemorrhage, ischaemia; fetal hypoxia
Tumours	Warburg effect — aerobic glycolysis (basis of FDG-PET)
Myometrium in prolonged labour	Lactate accumulation linked to dysfunctional labour

214.2 The Cori cycle

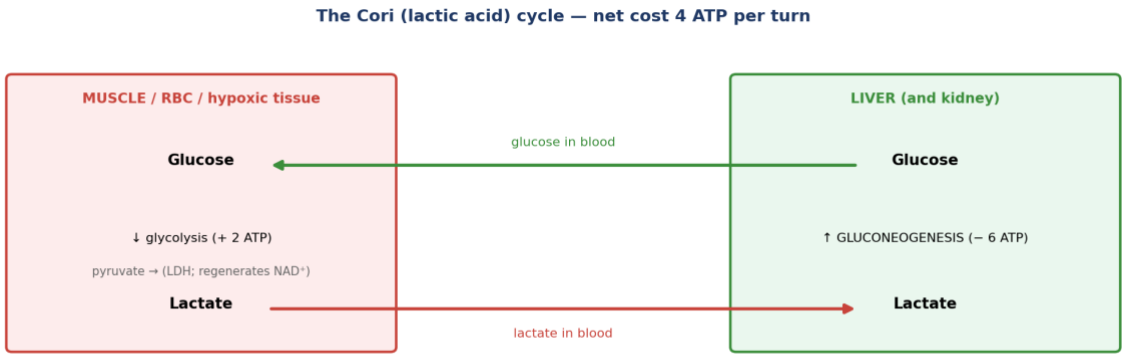


Figure 214.2 — The Cori cycle

Lactate is not a waste product: the **liver** (and kidney) reconverts it to glucose via gluconeogenesis, at a cost of 6 ATP (net cycle cost 4 ATP). Impaired hepatic clearance (liver failure, shock liver) worsens lactic acidosis. The **heart** oxidises lactate directly as fuel.

214.3 Lactic acidosis

Type	Mechanism	Causes (obstetric emphasis)
Type A (hypoxic)	Inadequate O_2 delivery	Sepsis, haemorrhage (major PPH), cardiogenic shock, severe anaemia, CO poisoning, eclampsia/seizures
Type B (non-hypoxic)	Metabolic / drug / toxin	Metformin (rare; renal failure), β_2-agonists (salbutamol, terbutaline), adrenaline, thiamine deficiency (PDH block — hyperemesis), liver failure (AFLP), DKA, malignancy, inborn errors, propofol

■ **RED FLAGS**

- **Lactate ≥ 2 mmol/L** is a sepsis red flag; **≥ 4 mmol/L** indicates severe sepsis/shock — part of 'Sepsis Six' and maternal early warning (MBRRACE: sepsis is a leading cause of maternal death).
- **Thiamine** is needed for pyruvate dehydrogenase — in hyperemesis/alcohol, glucose without thiamine → lactic acidosis and Wernicke's.

214.4 The fetus and neonate

Topic	Key points
Fetal hypoxia	Aerobic → anaerobic metabolism → lactate and H^+ accumulate → metabolic acidosis (↑ base deficit); fetus tolerates acute hypoxia via redistribution and glycogen reserves (heart, liver)
Fetal blood sampling	Scalp lactate ≤ 4.1 normal, 4.2–4.8 borderline, ≥ 4.9 mmol/L abnormal (pH ≤ 7.20 abnormal) — Lecture 159
Cord gases	Respiratory acidosis (↑ PCO_2, normal BD — brief cord compression) vs metabolic acidosis (↑ lactate, BD ≥ 12 mmol/L, pH < 7.00 — risk of HIE)
Reserves	FGR and preterm fetuses have less glycogen → less tolerance to labour hypoxia

214.5 Must-know summary

■ EXAM TIP

- Anaerobic glycolysis yields **2 ATP**; **LDH** converts pyruvate → lactate to **regenerate NAD^+**.
- RBCs (no mitochondria) rely on glycolysis; Warburg effect in tumours.
- Cori cycle: lactate → liver gluconeogenesis → glucose.
- Type A (hypoxic: sepsis, PPH) vs type B (metformin, β-agonists, thiamine deficiency, AFLP) lactic acidosis.
- Fetal scalp lactate ≥ 4.9 abnormal; cord BD ≥ 12 + pH < 7.0 = metabolic acidosis.

■ MNEMONIC / MEMORY AID

'No O_2, No Mitochondria → Lactate'. Lactate is a recycled fuel, not just waste (Cori cycle).