PIPADOR REVISION

MRCOG Part 1 Revision Notes

Response to Cell Injury

PATHOLOGY • LECTURE 312

VISUAL EXAMINATION REVISION NOTES

2026 Edition

312

MRCOG PART 1 REVISION NOTES | PATHOLOGY

Response to Cell Injury

Causes and mechanisms of cell injury, reversible vs irreversible injury, reperfusion, accumulations and calcification

Hypoxia	ATP ↓	Swelling	ROS
Commonest cause of cell injury	First key event	Earliest visible reversible change	Reperfusion injury

■ KEY FACTS: Learning objectives

- Describe causes of cell injury.
- Describe biochemical mechanisms and the transition from reversible to irreversible injury.
- Describe ischaemia–reperfusion injury, intracellular accumulations and examples in obstetrics.

312.1 Causes

- **Hypoxia/ischaemia** — haemorrhage, shock, placental insufficiency, cord compression, anaemia, CO poisoning.
- **Physical** — trauma, heat/cold, radiation (pelvic radiotherapy), electricity.
- **Chemical/drugs** — paracetamol, alcohol, chemotherapy, heavy metals.
- **Infection**, **immunological** reactions (autoimmunity, transplant rejection)
  - **Genetic** defects
  - **Nutritional** imbalance (deficiency or obesity).

312.2 Mechanisms

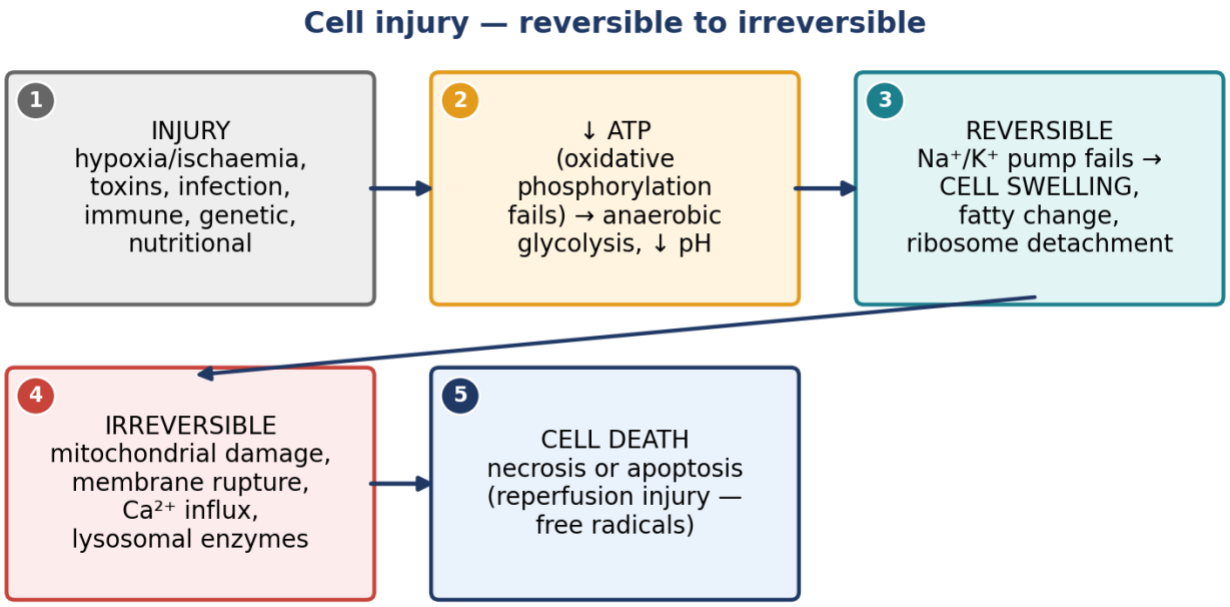


Figure 312.1 — Reversible and irreversible injury

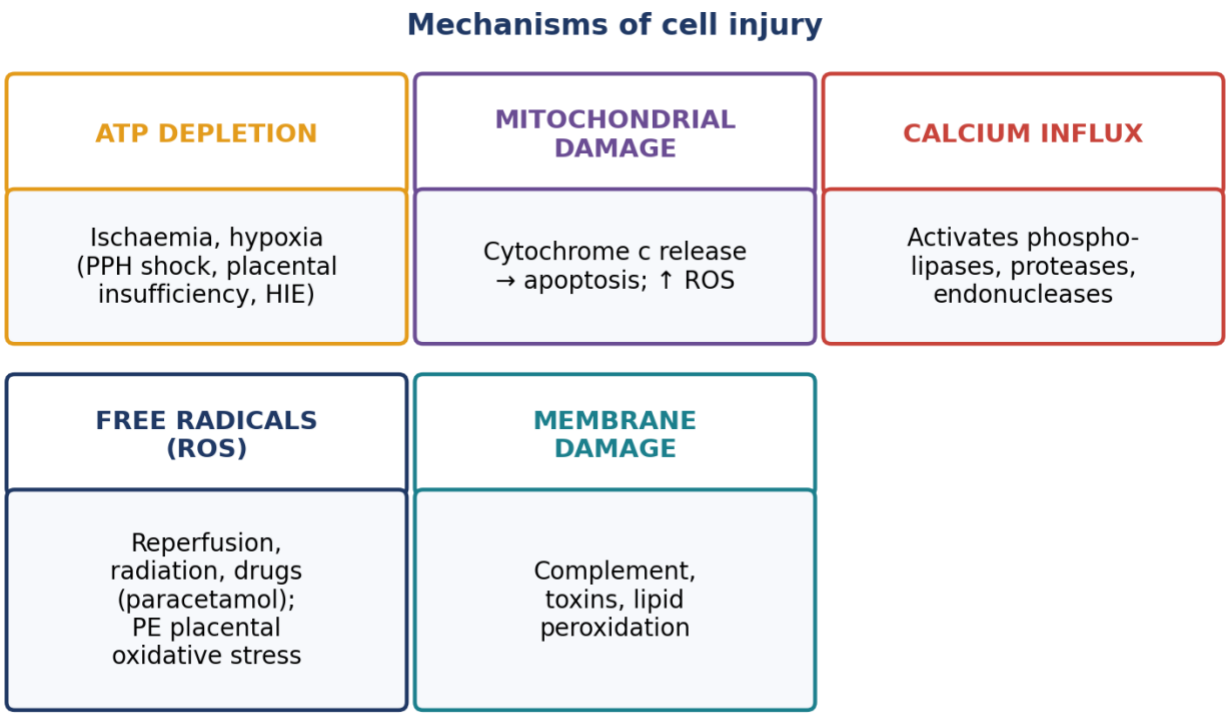


Figure 312.2 — Mechanisms of injury

Stage	Morphology
Reversible	Cellular swelling (hydropic change), fatty change (liver — AFLP is microvesicular), membrane blebs, ER dilatation, clumped chromatin
Irreversible ('point of no return')	• Severe mitochondrial dysfunction (no ATP recovery) and profound membrane damage • Nuclear changes • Enzyme leak (troponin, ALT, LDH, CK)

312.3 Reperfusion and accumulations

- **Ischaemia–reperfusion injury**: restoration of blood flow generates reactive oxygen species, calcium overload, neutrophil and complement activation → further damage (myocardial, neonatal HIE secondary energy failure — therapeutic hypothermia reduces it).
- **Placenta**: intermittent perfusion from poorly remodelled spiral arteries → oxidative stress → release of anti-angiogenic factors in pre-eclampsia.
- **Intracellular accumulations**: lipids (fatty liver)
  - Proteins (Russell bodies)
  - Glycogen (diabetes)
  - Pigments — lipofuscin (ageing)
  - Haemosiderin (iron overload, old haemorrhage — endometriosis), bilirubin.
- **Pathological calcification**: dystrophic (necrotic tissue — placental calcification, psammoma bodies in serous ovarian tumours) vs metastatic (hypercalcaemia).

312.4 Must-know summary

■ EXAM TIP

- Hypoxia → ↓ ATP → Na^+/K^+ pump failure → swelling (reversible).
- Irreversible: mitochondrial failure, membrane damage, Ca^{2+} influx, enzyme leak.
- Reperfusion injury: ROS, Ca^{2+}, inflammation — HIE, placental oxidative stress.
- Dystrophic calcification (psammoma bodies, placenta) vs metastatic.

■ MNEMONIC / MEMORY AID

Irreversible injury hallmarks — 'Mitochondria fail, Membranes break'.