PIPADOR REVISION

MRCOG Part 1 Revision Notes

Cell Death

PATHOLOGY • LECTURE 311

VISUAL EXAMINATION REVISION NOTES

2026 Edition

311

MRCOG PART 1 REVISION NOTES | PATHOLOGY

Cell Death

Necrosis and apoptosis, patterns of necrosis, apoptotic pathways and physiological apoptosis

Necrosis	Apoptosis	Caspases	Coagulative
Pathological; inflammation	Programmed; no inflammation	Executors of apoptosis	Commonest necrosis

■ KEY FACTS: Learning objectives

- Compare necrosis and apoptosis (morphology, mechanisms, consequences).
- Describe the patterns of necrosis with examples.
- Describe the intrinsic and extrinsic apoptotic pathways and physiological apoptosis in reproduction.

311.1 Necrosis vs apoptosis

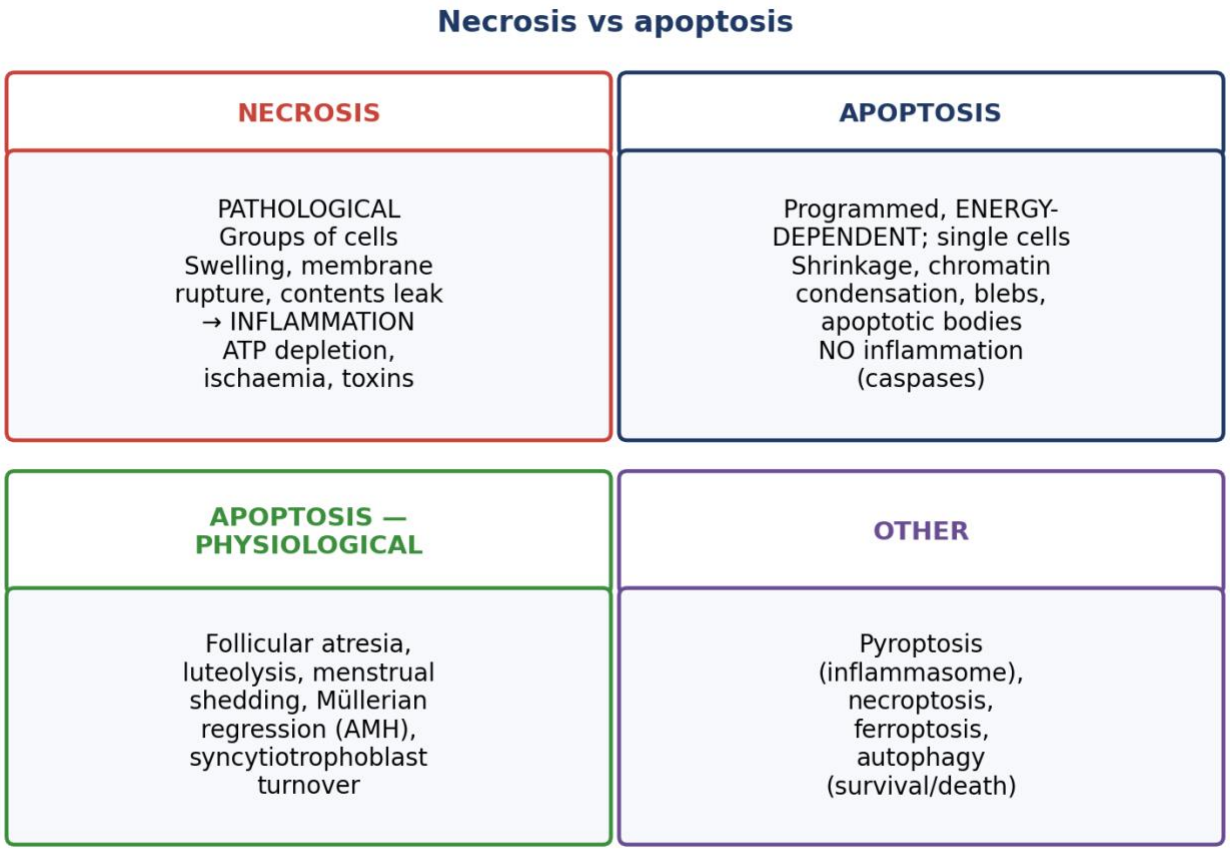


Figure 311.1 — Necrosis and apoptosis

Feature	Necrosis	Apoptosis
Cell size	Swelling	Shrinkage
Nucleus	Pyknosis → karyorrhexis → karyolysis	Chromatin condensation, fragmentation (DNA laddering)

Feature	Necrosis	Apoptosis
Membrane	Disrupted	• Intact • Blebs, apoptotic bodies • Phosphatidylserine flipped out ('eat me')
Inflammation	Yes	No
Energy	Passive (ATP depletion)	ATP-dependent
Examples	Infarction, toxins, ischaemic injury	Development, atresia, CTL killing, DNA damage

311.2 Patterns of necrosis

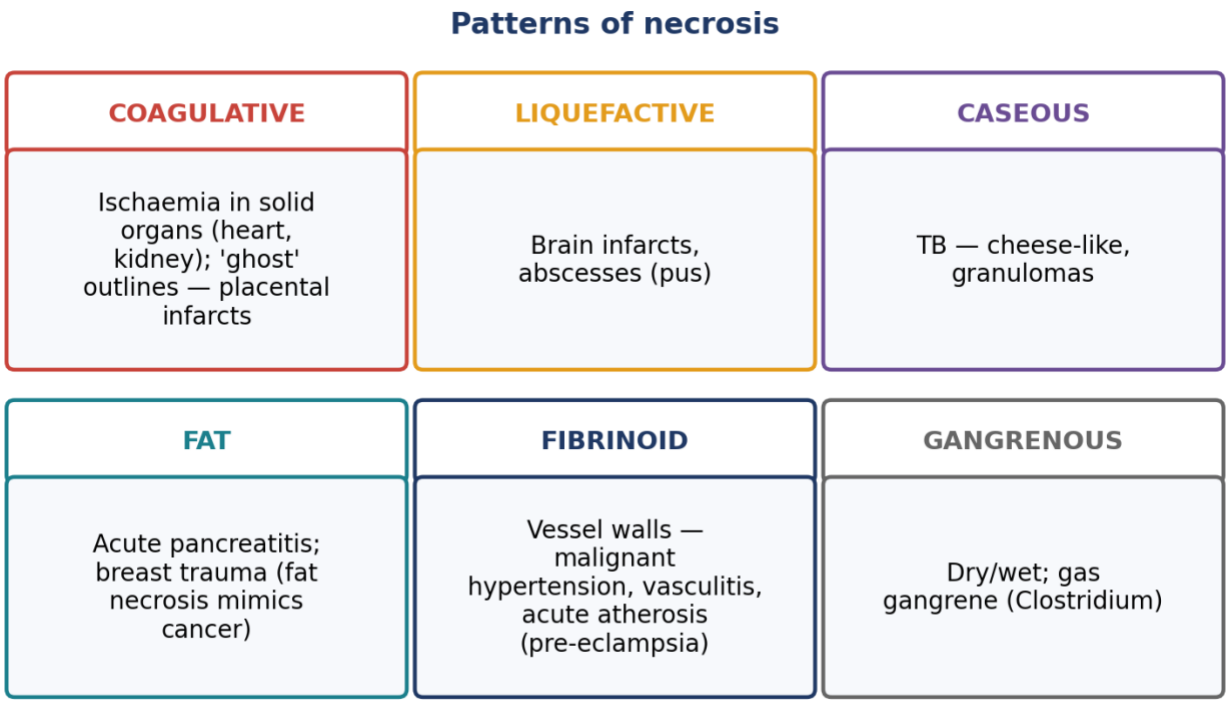


Figure 311.2 — Patterns of necrosis

311.3 Apoptotic pathways

Pathway	Mechanism
Intrinsic (mitochondrial)	DNA damage, growth-factor withdrawal, stress → p53 → BAX/BAK > BCL-2 → cytochrome c release → apoptosome → caspase-9 → caspase-3
Extrinsic (death receptor)	Fas ligand / TNF → death receptors → caspase-8 → caspase-3
Cytotoxic T/NK cells	Perforin + granzymes activate caspases
Dysregulation	• ↓ Apoptosis → cancer (BCL-2 overexpression, p53 loss — HPV E6), autoimmunity • ↑ apoptosis → neurodegeneration, ischaemic injury

- **Reproductive apoptosis**: follicular atresia (≈ 99% of follicles), luteolysis
  - Endometrial shedding
  - Müllerian duct regression (AMH)
  - Interdigital web loss

– Villous trophoblast turnover (increased in pre-eclampsia and FGR placentas).

311.4 Must-know summary

■ **EXAM TIP**

- Necrosis: swelling, membrane rupture, inflammation; apoptosis: shrinkage, apoptotic bodies, no inflammation, caspases.
- Coagulative (infarcts — placental), liquefactive (brain, abscess), caseous (TB), fat (breast/pancreas), fibrinoid (vessels — acute atherosis), gangrenous.
- Intrinsic (cytochrome c, BCL-2 family) and extrinsic (Fas/TNF) pathways converge on caspase-3.

■ **MNEMONIC / MEMORY AID**

Nuclear changes in necrosis — 'Pyknosis, Karyorrhexis, Karyolysis' (shrink, shatter, dissolve).