Pathology

Curated preclinical study track (not a medical degree, no licensure claim). Original explanations and practice only, following the reviewed course handout.

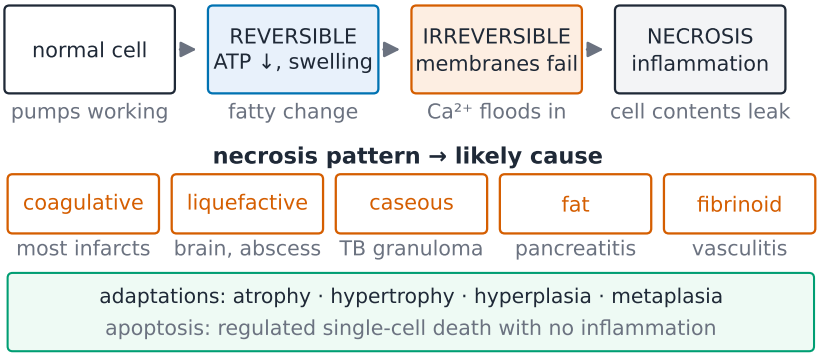
From stress to scar

- Units 10.1-10.4 general pathology (injury, inflammation, haemodynamics, **neoplasia**); 10.5-10.7 systems and the laboratory.
- Every lesion is traced from mechanism to finding.

Cell injury: reversible to irreversible

1. **Reversible** Hypoxia $\rightarrow$ ATP $\downarrow \rightarrow$ Na^+/K^+ pump fails $\rightarrow$ swelling, fatty change; membranes intact.
2. **No return** Membrane damage + Ca^{2+} influx + mitochondrial death — enzymes leak (**troponin**, LDH rise).
3. **Patterns** Coagulative (ischaemia) · liquefactive (brain) · caseous (TB) · fat (pancreas) · fibrinoid (vessels); **apoptosis** is quiet deletion.

Injury progression from swelling to death patterns



ATP depletion swells the cell; membrane damage kills it — the pattern names the cause.

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Inflammation and repair: two clocks

1. **Cells** Acute: **neutrophils**, exudate, cardinal signs. Chronic: mononuclear + scarring; **granulomas** caseous (TB) vs non-caseous (sarcoid).
2. **Programme** Haemostasis (hours) $\rightarrow$ inflammation (days 1-3) $\rightarrow$ **granulation tissue** (days 3-7) $\rightarrow$ remodelling months.
3. **Strength** ~10 % at 1 week · rising fast over the next month · ~70-80 % at 3 months — never 100 %.

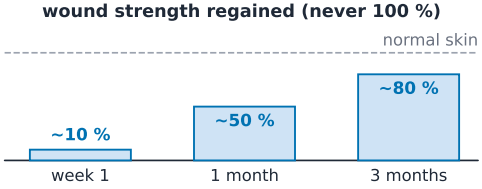
The wound-healing timeline

1 haemostasis
hours
platelet plug, fibrin mesh

2 inflammation
days 1–3
neutrophils → macrophages

3 proliferation
days 3–7
fibroblasts lay type III collagen; new vessels

4 remodelling
weeks–months
type I replaces III; scar matures



inflammation types

acute: neutrophils

chronic: mononuclear cells

granuloma, caseous → TB

non-caseous → sarcoid

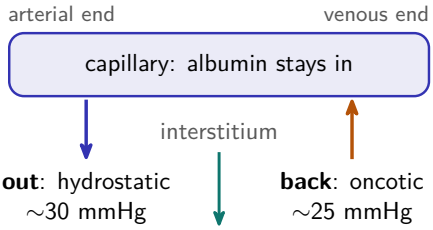
Haemostasis, inflammation, proliferation, remodelling: each phase has its clock.

Haemodynamic disorders: fluids and clots

1. **Starling** Hydrostatic out ~30/15 mmHg · oncotic (albumin) ~25 back · lymph drains the surplus.
2. **Oedema** ↑ hydrostatic (failure) · ↓ oncotic (liver/kidney) · ↑ permeability (inflammation) · lymph block.
3. **Clots** Injury + stasis + hypercoagulability (**Virchow's triad**) → thrombus → embolus → infarct; shock: cardiogenic, hypovolaemic class III ~30 % / IV >40 %, distributive - septic is warm and vasodilated first.

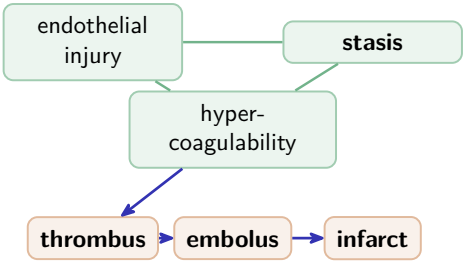
Starling forces and Virchow's triad

Starling forces



oedema: hydrostatic ↑, oncotic ↓, permeability ↑ or lymph blocked

Virchow's triad



pale infarct: solid organs
red infarct: lung, gut (dual supply)

Push out, pull back, drain the surplus; three forces make a clot.

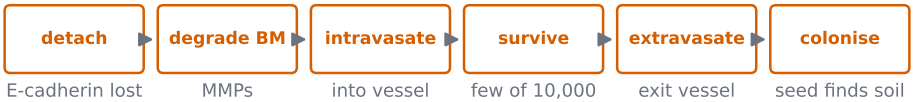
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Neoplasia: gates to metastasis

1. **Cascade** Detach (E-cadherin lost) $\rightarrow$ degrade basement membrane (MMPs) $\rightarrow$ intravasate $\rightarrow$ survive (under 1 in 10,000 cells) $\rightarrow$ extravasate $\rightarrow$ colonise a hospitable soil (seed and soil).
2. **Axes** Grade = appearance (Gleason 6-10, Grade Groups 1-5); stage = TNM extent — prognosis follows stage first.
3. **Routes** Lymphatic (carcinoma) · blood (sarcoma, kidney, liver) · transcoelomic (ovary).

The invasion-metastasis cascade

six gates, each low-probability — metastasis is selection, not leakage



benign vs malignant

	benign	malignant
differentiation	well-kept	lost (pleomorphic)
growth	slow, capsule	rapid, invasive
spread	stays local	METASTASISES
example	lipoma	adenocarcinoma

grade vs stage — two different axes

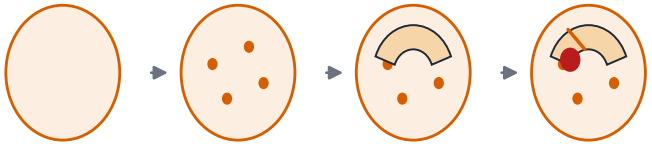
GRADE = how the cells LOOK (differentiation)
prostate: Gleason 2–10 (two patterns summed)
STAGE = how FAR the disease has reached
TNM: T1–4 size · N0–3 nodes · M0/1 spread
routes: lymph (carcinoma) · blood (sarcoma)
transcoelomic (ovary seeds the peritoneum)

Detach, degrade, enter, survive, exit, colonise — six gates to **metastasis**.

Cardiovascular and respiratory pathology: plaque and ratio

1. **Plaque** Injury $\rightarrow$ LDL $\rightarrow$ **fatty streak** $\rightarrow$ fibrolipid plaque $\rightarrow$ rupture $\rightarrow$ thrombosis $\rightarrow$ infarct.
2. **Clock Troponin** 3-4 h, stays $\sim$7-10 days; CK-MB 1-3 days flags a second event.
3. **Ratio** FEV1/FVC $<$0.70 obstructive ($1.6/4.0 = 0.40$); both low, ratio $\geq$0.8 restrictive ($1.8/2.0 = 0.90$).

Plaque progression with the troponin clock



normal wall
endothelium intact

fatty streak
foam cells — earliest

fibrolipid plaque
cap over lipid core

RUPTURE
thrombus occludes → MI

risk is the THIN cap,
not the plaque size

the troponin clock

troponin: 3–4 h, stays ~7–10 days
CK-MB: 1–3 days (second-event clock)
necrosis 4–12 h → neutrophils 1–3 d

one ratio, two diseases

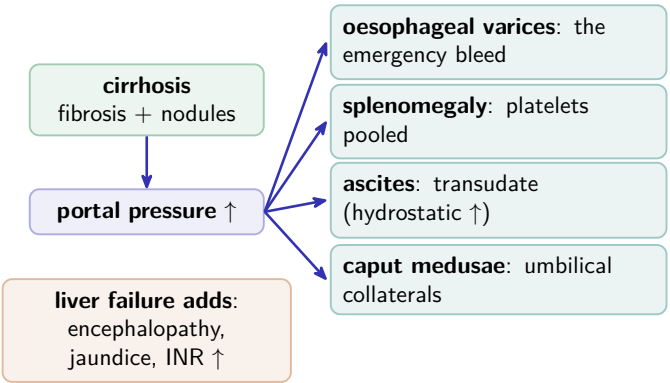
obstructive: FEV1/FVC < 0.70 (trapped)
restrictive: both low, ratio ≥ 0.8
1.6/4.0 = 0.40 · 1.8/2.0 = 0.90

From **fatty streak** to rupture, with the clocks and the spirometry fork.

Liver and kidney: patterns

1. **Portal Cirrhosis** (fibrosis + regenerative nodules) $\rightarrow$ **portal hypertension**: varices, splenomegaly (platelets pooled $\rightarrow$ thrombocytopenia), ascites, caput medusae; failure adds encephalopathy (ammonia), jaundice, coagulopathy (vitamin K, INR).
2. **Panel** ALT/AST injury · ALP obstruction · albumin + INR synthesis.
3. **Kidney** Nephritic: pores torn - haematuria with red-cell casts, hypertension, oliguria, proteinuria $<$3.5 g/day. Nephrotic: podocytes injured - frothy urine, proteinuria $\geq$3.5 g/day, hypoalbuminaemia, periorbital **oedema**, hyperlipidaemia.

Portal hypertension consequences



two kidney lesions

nephritic: pores torn
blood + casts, hypertension
protein $< 3.5\,\mathrm{g/day}$

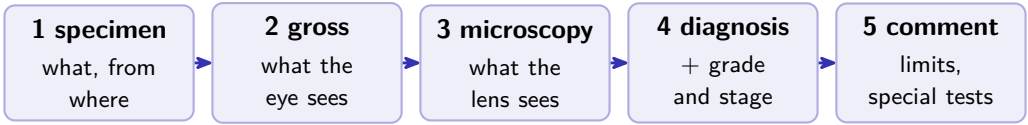
nephrotic: podocytes injured
protein $\geq 3.5\,\mathrm{g/day}$
low albumin, oedema, lipids ↑

One blocked vein, four collateral results; two kidney lesions.

Laboratory medicine: the tube first

1. **Interval Reference interval**: the central 95 % of healthy people (2.5th-97.5th percentiles) - 5 % of healthy outside (2.5 % each side) by definition; trends beat thresholds.
2. **Pre-analytical Haemolysis** $\rightarrow$ K^+ falsely high · EDTA tube $\rightarrow$ $K^+\uparrow$ $Ca^{2+}\downarrow$ · glucose falls ~5-7 % per hour unspun.
3. **Report** Specimen $\rightarrow$ gross $\rightarrow$ microscopy $\rightarrow$ diagnosis (+ grade/stage) $\rightarrow$ comment; glucose mmol/L $\times 18 =$ mg/dL.

The structured pathology report



Reference interval the central 95 % of healthy people: 1 healthy person in 20 falls outside it. Trends beat thresholds.

$$\text{glucose (mg/dL)} = 18 \times \text{glucose (mmol/L)}$$

5.5 mmol/L $\times$ 18 $\approx$ 99 mg/dL: the same concentration in two units

Check yourself

- The point of no return is…? (Membrane damage with Ca^{2+} influx.)
- Wound strength at 3 months…? (~80 %, never 100 %.)
- The nephrotic line…? (Proteinuria $\geq$ 3.5 g/day.)
- Glucose 5.5 mmol/L in mg/dL…? ($\approx$ 99.)