

Pathology

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

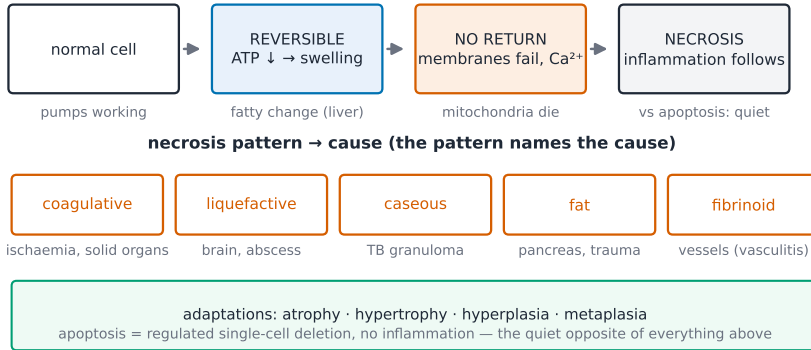
From stress to scar

- 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 + Ca²⁺ 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

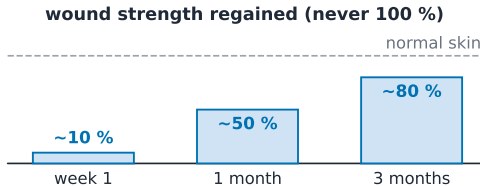
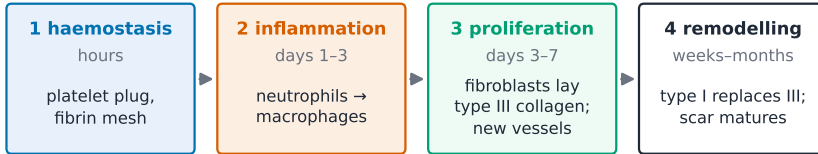


ATP depletion swells the cell; membrane damage kills it — the pattern names the cause.

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) → inflammation (days 1-3) → **granulation tissue** (days 3-7) → 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



inflammation types

acute: neutrophils

chronic: mononuclear cells

granuloma, caseous → TB

non-caseous → sarcoid

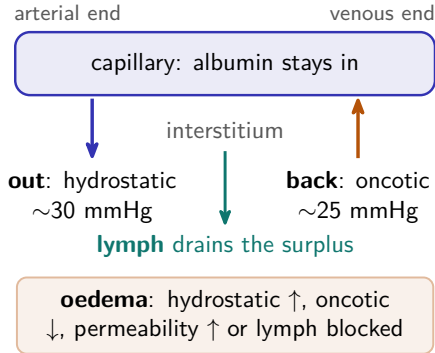
Haemostasis, inflammation, proliferation, remodelling: each phase has its clock.

Haemodynamic disorders: fluids and clots

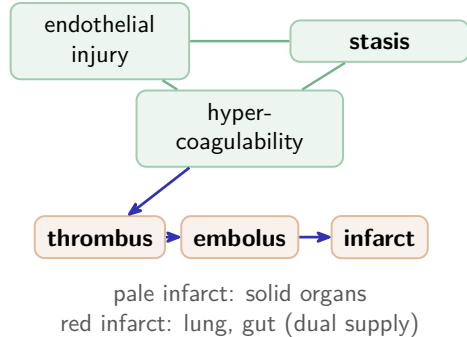
- 1 Starling** Hydrostatic out $\sim 30/15$ mmHg · oncotic (albumin) ~ 25 back · lymph drains the surplus.
- 2 Oedema** $\uparrow$ hydrostatic (failure) · $\downarrow$ oncotic (liver/kidney) · $\uparrow$ permeability (inflammation) · lymph block.
- 3 Clots** Injury + stasis + hypercoagulability (**Virchow's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

Starling forces and Virchow's triad

Starling forces



Virchow's triad



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

Neoplasia: gates to metastasis

- 1 Cascade** Detach (E-cadherin lost) → degrade basement membrane (MMPs) → intravasate → survive (under 1 in 10,000 cells) → extravasate → 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

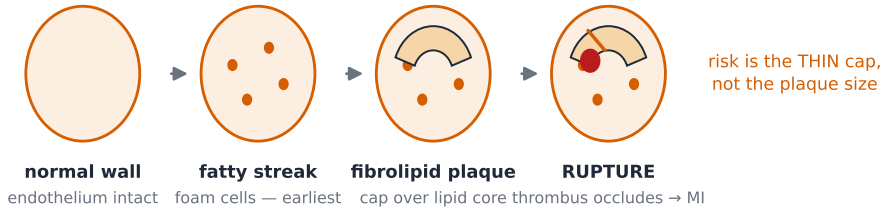
GRADE = how the cells LOOK (differentiation)
 prostate: Gleason 2-10 (two patterns summed)
 STAGE = how FAR the disease has reached
 TNM: T1-4 size · N0-3 nodes · M0/1 spread
 routes: lymph (carcinoma) · blood (sarcoma)
 transcoelomic (ovary seeds the peritoneum)

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 ≥ 0.8 restrictive ($1.8/2.0 = 0.90$).

Plaque progression with the troponin clock



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

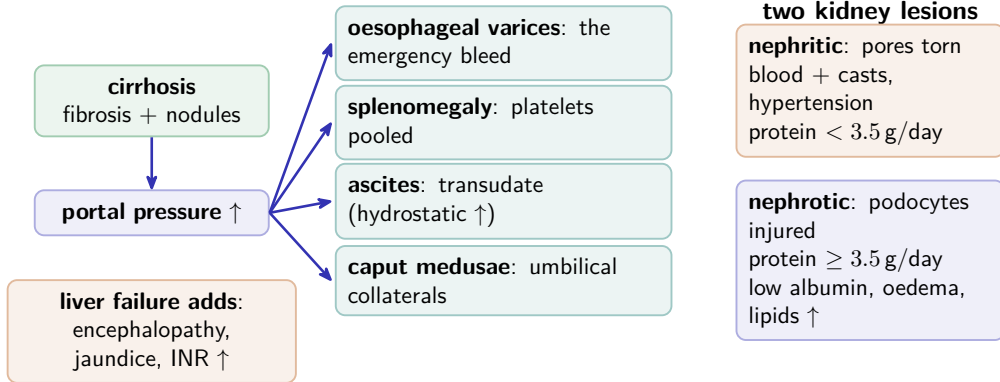
obstructive: $FEV1/FVC < 0.70$ (trapped)
 restrictive: both low, ratio ≥ 0.8
 $1.6/4.0 = 0.40 \cdot 1.8/2.0 = 0.90$

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

Liver and kidney: patterns

- 1 Portal Cirrhosis** (fibrosis + regenerative nodules) → **portal hypertension**: varices, splenomegaly (platelets pooled → 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 ≥ 3.5 g/day, hypoalbuminaemia, periorbital **oedema**, hyperlipidaemia.

Portal hypertension consequences

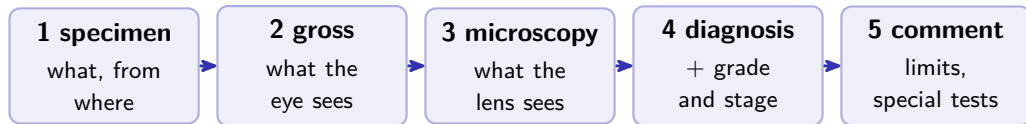


One blocked vein, four collateral results; two kidney lesions.

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** → K^+ falsely high · EDTA tube → $K^+ \uparrow$ $Ca^{2+} \downarrow$ · glucose falls ~5-7 % per hour unspun.
- 3 Report** Specimen → gross → microscopy → diagnosis (+ grade/stage) → 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.

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

$5.5 \text{ mmol/L} \times 18 \approx 99 \text{ mg/dL}$: 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 ≥ 3.5 g/day.)
- Glucose 5.5 mmol/L in mg/dL...? (≈ 99 .)