PIPADOR REVISION

MRCOG Part 1 Revision Notes

Wound Healing

PHYSIOLOGY • LECTURE 162

VISUAL EXAMINATION REVISION NOTES

2026 Edition

MRCOG PART 1 REVISION NOTES | PHYSIOLOGY

162 Wound Healing

Phases of healing, influencing factors, abnormal scarring, sutures and obstetric wounds

4 phases	≈ 20–30%	≈ 70–80%	≈ 5–10%
Haemostasis, inflammation, proliferation, remodelling	Tensile strength at 3 weeks	Maximum strength (never 100%)	Surgical site infection after caesarean

■ KEY FACTS: Learning objectives

- Describe the phases, cells and mediators of wound healing and types of healing.
- Describe local and systemic factors affecting healing.
- Describe abnormal scarring and suture materials.
- Apply wound healing to caesarean section, perineal repair and uterine scars.

162.1 Phases of healing

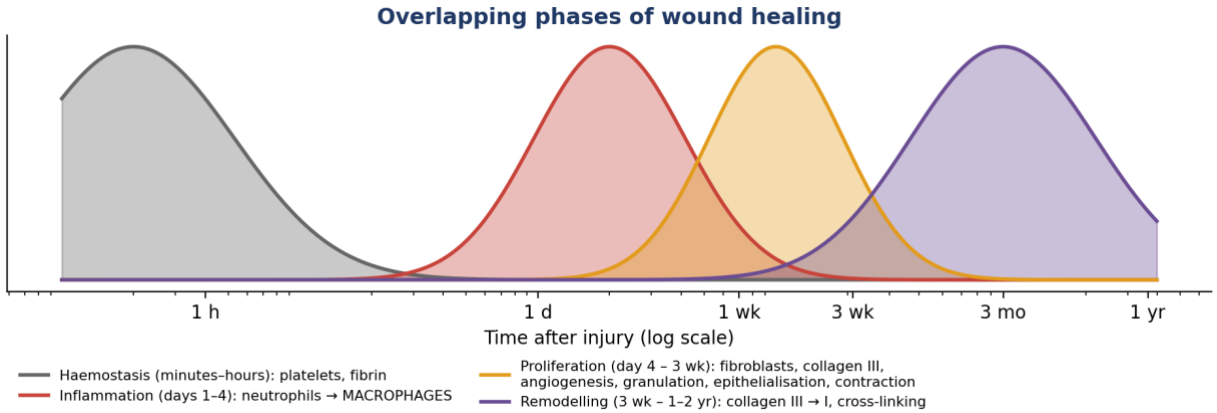


Figure 162.1 — Overlapping phases of wound healing

Phase	Timing	Key events and cells	Mediators
Haemostasis	Minutes–hours	Vasoconstriction, platelet plug, fibrin clot (provisional matrix)	PDGF, TGF-β from platelets; thrombin
Inflammation	Days 1–4	Neutrophils (24–48 h; debride, kill bacteria) → macrophages (day 2–3; essential — phagocytosis and growth-factor 'conductor')	IL-1, TNF-α, PDGF, TGF-β, VEGF
Proliferation	Day 4 – 3 weeks	Fibroblasts lay type III collagen (needs vitamin C, O_2, iron, zinc); angiogenesis → granulation tissue; re-epithelialisation (from edges/appendages); myofibroblast contraction	FGF, VEGF, EGF, KGF, TGF-β
Remodelling (maturation)	3 weeks – 1–2 years	Collagen III → I, cross-linking (lysyl oxidase — copper), MMP turnover, ↓ vascularity — scar pales and flattens	MMPs/TIMPs, TGF-β

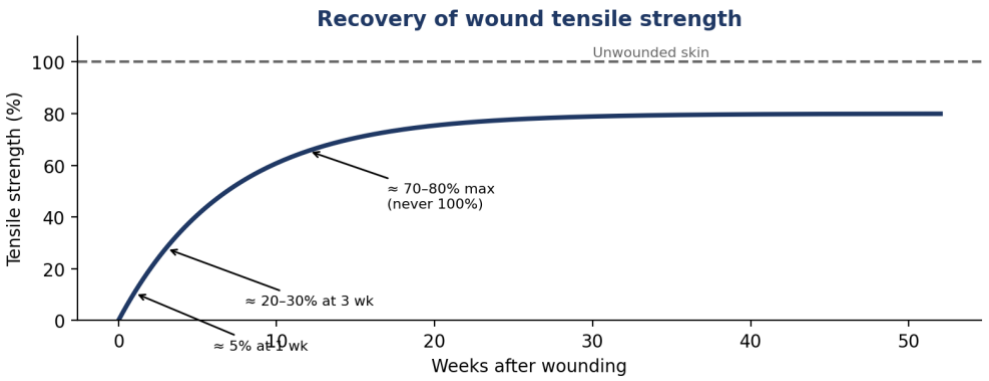


Figure 162.2 — Recovery of tensile strength

Primary intention
Edges apposed (sutured incision) — minimal granulation, fine scar

Secondary intention
Open wound fills with granulation; contraction; slower, larger scar

Tertiary (delayed primary)
Contaminated wound left open 3–5 days, then closed

162.2 Factors affecting healing

Local	Systemic
Infection (> 10^5 organisms/g), haematoma/seroma, tissue ischaemia (tight sutures, poor blood supply), tension, foreign body, dead space, radiotherapy, previous scarring, oedema, poor surgical technique	Obesity (BMI ≥ 30 — avascular fat, tension, haematoma), diabetes (hyperglycaemia impairs neutrophils, microvascular disease), smoking (vasoconstriction, CO), corticosteroids (↓ inflammation — vitamin A partially reverses), malnutrition (protein, vitamin C, zinc), anaemia, age, immunosuppression, chemotherapy, jaundice, uraemia

162.3 Abnormal healing and scars

	Hypertrophic scar	Keloid
Extent	Within wound margins	Beyond wound margins (claw-like)
Timing	Within weeks; may regress	Months later; does not regress
Risk	Tension, burns, infection	Darker skin, family history, sternum/earlobes/shoulders; ↑ in pregnancy
Collagen	Type III, organised parallel to surface	Disorganised thick type I/III bundles
Treatment	Pressure, silicone, steroid injection	Intralesional steroids, silicone, excision + adjuncts (high recurrence)

Other complications: **dehiscence** (fascial — 'pink fluid' day 5–8; risk factors above), incisional hernia, chronic wounds, sinus formation (non-absorbable suture), **necrotising fasciitis** (group A streptococcus, polymicrobial).

162.4 Obstetric and gynaecological applications

Situation	Key points
Caesarean wound SSI prevention (NICE NG125/NG192)	Antibiotic prophylaxis before skin incision; chlorhexidine–alcohol skin preparation; avoid routine hair removal (clip if needed); close subcutaneous fat if ≥ 2 cm; suture vs staples (sutures fewer complications); consider negative-pressure dressings in high-risk (obese) women; normothermia, glycaemic control

Situation	Key points
Perineal tears (RCOG GTG 29)	1st: skin; 2nd: perineal muscles; 3a < 50% EAS; 3b > 50% EAS; 3c IAS torn; 4th: anal mucosa. Repair in theatre (OASI) with good light/analgesia; broad-spectrum antibiotics and laxatives; physiotherapy; follow-up
Perineal healing	Highly vascular — heals quickly; continuous rapidly-absorbed polyglactin for 2nd-degree repair (less pain); infection and breakdown ↑ with haematoma, obesity, smoking
Uterine scar	Myometrium heals by fibrosis; niche/isthmocele (abnormal bleeding, scar ectopic); uterine rupture in VBAC ≈ 0.5% (RCOG GTG 45) — higher with classical/T-incisions, induction with prostaglandins, short inter-delivery interval
Endometrium	Regenerates from basalis each cycle (scarless); damage → Asherman's syndrome
Fascia	Regains ≈ 50% strength at 6 weeks — mass closure with slowly absorbable/non-absorbable suture (suture length:wound length ≈ 4:1)

Suture	Type	Absorption / use
Polyglactin 910 (Vicryl)	Absorbable, braided	≈ 60–90 days (strength ≈ 3 wk); Vicryl Rapide ≈ 10–14 days (perineal skin)
Poliglecaprone (Monocryl)	Absorbable, monofilament	≈ 90–120 days; subcuticular skin
Polydioxanone (PDS)	Absorbable, monofilament	≈ 180 days; fascial closure, EAS repair (or polyglactin)
Polypropylene (Prolene), nylon	Non-absorbable, monofilament	Skin (removed), fascia, vascular; low reactivity
Silk	Non-absorbable, braided	High tissue reaction — rarely used

Braided sutures handle well but harbour bacteria (capillarity); monofilaments have less infection risk but more memory/knot slippage.

162.5 Must-know summary

■ EXAM TIP

- Haemostasis → inflammation (neutrophils then **macrophages — essential**) → proliferation (fibroblasts, **type III collagen**, angiogenesis) → remodelling (**III → I**).
- Tensile strength ≈ **20–30% at 3 weeks**, max **≈ 70–80%** of normal.
- Vitamin C (hydroxylation), zinc, iron, oxygen and copper (cross-linking) are needed for collagen.
- Keloid extends **beyond** the margins (darker skin); hypertrophic stays within.
- OASI: 3a < 50% EAS, 3b > 50%, 3c IAS, 4th anal mucosa.

■ MNEMONIC / MEMORY AID

Phases — 'Healing Is Pretty Remarkable': Haemostasis, Inflammation, Proliferation, Remodelling. **Delayed healing — 'DIDN'T HEAL'**: Diabetes, Infection, Drugs (steroids), Nutrition, Tension, Hypoxia, Excess (obesity), Anaemia, Low temperature/radiation.