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Jun 2020 – Dec 2025

FOR THE DNB GENERAL SURGERY EXIT EXAMINATION

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Dr Ashok Bude · Dr Avni Dendu Kumari · Dr Kurunakar Jamnani

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Updated till December 2025. Answers written from Bailey & Love 28e, Sabiston 22e and Schwartz 11e
(trauma, burns, endocrine and surgical critical care only).

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The reviewers



Dr Ashok Bude

DNB (General Surgery)



Dr Avni Dendu Kumari

DNB (General Surgery)



Dr Kurunakar Jamnani

DNB (General Surgery)

From the reviewers

This book brings together the theory questions of the DNB General Surgery exit examination and answers them the way an examiner wants to read them: structured, complete and traceable to the standard textbooks.

Questions that return across sessions are merged under a single answer, and the “Asked in” line beside each one shows every paper and year it was set. That lets you spend your revision where the paper keeps coming back.

Every answer closes with its book, chapter and page references, so any point can be checked at the source. Where the textbooks are silent or have been overtaken by a newer guideline, a clearly marked box says so.

We hope it shortens your revision and steadies your writing on the day of the exam.

Dr Ashok Bude

Dr Avni Dendu Kumari

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Dr Ashok Bude
DNB (General Surgery)

Dr Avni Dendu Kumari
DNB (General Surgery)

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Reviewed by Dr Ashok Bude, Dr Avni Dendu Kumari and Dr Kurunakar Jamnani, DNB (General Surgery)

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- **One answer, many questions:** repeats and near-repeats are merged into one answer; its "Asked in" line lists every paper and year, and the badge (1×, 2×, 3×, 4×+) shows how often it was asked.
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- **Sources** at the end of each answer give book, chapter, section and printed pages.

A • Parts in this volume

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Principles of Surgery, Trauma & Burns. This sample continues with printed pages 9–38. The full answer list for this volume and the high-yield list are in the complete book. To order the full set, visit clinicapublication.com.

Basic Principles: Metabolic Response, Shock, Blood & Fluids

1.1a Surgical stress response — avoidable factors that compound it and measures that reduce it

2×

Asked in: Paper I · Dec 2022 · Q1(a) (5 marks) ; Paper I · Aug 2021 · Q1(a) (5 marks)

“Avoidable factors that compound the response to injury.”

“Factors to reduce surgical stress response.”

- **Surgical stress response:** systemic changes after surgical or accidental injury, driven by hormones, inflammatory cytokines and neural circuits; it is **graded** — the greater the injury, the greater the response.
- Several factors **prolong the acute-phase response** and keep the patient catabolic; limiting them benefits the patient.

Avoidable factors that compound the response to injury

- **Secondary triggers** that amplify or prolong catabolism: sepsis, **continuing haemorrhage**, massive transfusion, acidosis, further surgery, crush syndrome, ischaemia–reperfusion.
- **Volume loss:** baroreceptor and atrial volume-receptor input → **ADH and aldosterone** release → oliguria, sodium and water retention.
 - Made worse by **saline-rich resuscitation** → peripheral and **visceral (gastric) oedema**, delayed gastric emptying and food intake, longer stay.
- **Hypothermia:** ↑ adrenal steroids and catecholamines; even mild hypothermia → **two- to threefold rise in postoperative arrhythmias** and increased catabolism.
- **Tissue oedema:** capillary leak (cytokines, prostanoids, bradykinin, nitric oxide) impairs alveolar oxygen diffusion and renal function.
- **Systemic inflammation and tissue underperfusion:** excessive endothelial activation → compromised microcirculation, cellular hypoxia, organ failure.
- **Starvation** (including preoperative fasting): obligate need for **100 g glucose/day** for the brain → glycogen used in 24 h, then **gluconeogenesis from muscle amino acids**.
- **Immobility:** potent stimulus for **muscle wasting**; impairs meal-stimulated muscle protein synthesis.
- **Uncontrolled pain** (also stimulates ADH), anaesthesia, dehydration, acute medical illness, frailty, chronic disease and severe psychological stress.

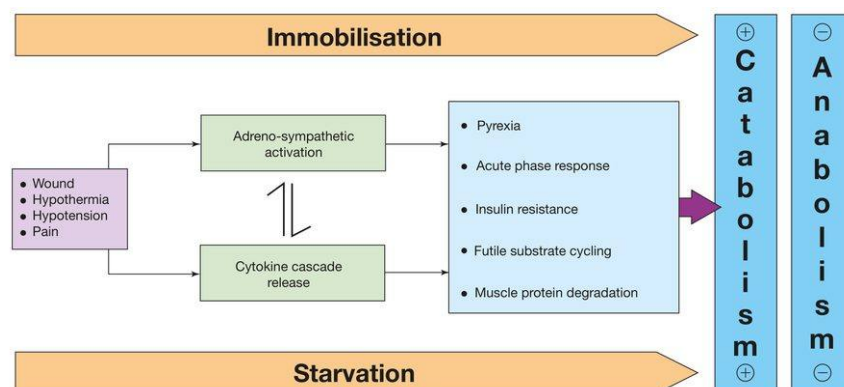


Figure 1.1a.1 — Factors that exacerbate the metabolic response to surgical injury: hypothermia, uncontrolled pain, starvation, immobilisation, sepsis and medical complications.

BL 28e Fig. 1.7, p. 9

Factors that reduce the surgical stress response

- **Before surgery:**
 - **Prehabilitation** (nutrition, exercise, mental preparation for 4–6 weeks) and correction of comorbidity, anaemia and iron deficiency; stop smoking and alcohol, preferably **6 weeks** before.

- **Avoid prolonged fasting:** clear fluids up to **2 hours** and food up to 6 hours before surgery; a **carbohydrate drink** reduces postoperative **insulin resistance**, anxiety and thirst.
- Avoid routine oral bowel preparation (dehydration).
- **During surgery:**
 - **Minimal access surgery** — smaller injury, lower inflammatory mediators; low-pressure pneumoperitoneum (8–12 mmHg) reduces IL-6 release.
 - **Afferent blockade:** thoracic **epidural/spinal** covering T10 reduces **adrenaline and cortisol** release and insulin resistance (open surgery); wound catheters, spinal diamorphine, IV lidocaine are opioid-sparing.
 - **Multimodal, opioid-sparing anaesthesia** with short-acting drugs (paracetamol, NSAIDs, ketamine, dexmedetomidine, dexamethasone).
 - **Normothermia:** forced-air warming, warmed fluids, ambient $\geq 21^{\circ}\text{C}$; avoid core temperature $< 36^{\circ}\text{C}$.
 - **Balanced fluids:** near-zero balance, weight gain $< 2\text{ kg}$; vasoactive drugs rather than fluid for anaesthetic hypotension; goal-directed therapy in high-risk patients.
- **After surgery:**
 - **Early oral/enteral feeding:** 2 L of 4% dextrose/0.18% saline gives 80 g glucose/day and is protein-sparing in the fasted patient.
 - **Early mobilisation;** avoid unnecessary bed rest, nasogastric tubes, drains and catheters.
 - **Glycaemic control** with insulin infusion in critical illness; treat sepsis promptly.
 - **β -blockers** modulate the hyperadrenergic state.

Pearls

- ✓ Avoidable factors (BL): continuing haemorrhage, hypothermia, tissue oedema, underperfusion, starvation, immobility.
- ✓ ERAS = multimodal care that makes a major operation behave like a minor one metabolically.
- ✓ Epidural + carbohydrate loading + early feeding nearly abolish insulin resistance after open colorectal surgery.
- ✓ Epidural is no longer recommended for laparoscopic surgery — PCA suffices.
- ✓ Hyperalimentation is itself a metabolic stress; feed modestly.

Sources

- BL 28e, ch. 1 “Metabolic response to injury”, §“Tissue damage and inflammation; Table 1.1 secondary triggers”, p. 3.
- BL 28e, ch. 1 “Metabolic response to injury”, §“Avoidable factors that compound the response to injury; Enhanced recovery after surgery”, pp. 8–10; Fig. 1.7, p. 9.
- SAB 22e, ch. 22 “Enhanced Recovery After Surgery (ERAS): Evidence-Based P”, §“Physiology of the effects of ERAS; preadmission, preoperative and intraoperative care elements”, pp. 78–82.

1.1b SIRS, metabolic response to injury and the neuroendocrine pathway

2x

Asked in: Paper I · Oct 2024 · Q1 (2+(3+5 marks) ; Paper I · Oct 2023 · Q1(a) (3 marks)

“What is SIRS? Discuss metabolic response to injury and explain neuroendocrine pathway.”

“Neuroendocrine response to injury.”

What is SIRS?

- **Systemic inflammatory response syndrome (SIRS):** the clinically detectable systemic inflammatory cascade that follows injury — a harmful excess of the normal inflammatory response, with high circulating cytokines and activated neutrophils.
- Causes: **sepsis**, and without infection **multiple trauma, burns, pancreatitis**, major surgery.
- Corresponds to the **hypermetabolic flow phase** after resuscitation.

Table 1.1b.1. SIRS criteria (two or more)

Variable	Criterion
Temperature	$> 38^{\circ}\text{C}$ or $< 36^{\circ}\text{C}$
Heart rate	$> 90/\text{min}$ (not on β -blockers)
Respiration	$> 20/\text{min}$ or $\text{PaCO}_2 < 32\text{ mmHg}$
White cell count	$> 12 \times 10^9/\text{L}$ or $< 4 \times 10^9/\text{L}$ (or $> 10\%$ immature neutrophils)
Sepsis (old definition)	SIRS + documented source of infection

Source: BL 28e Table 5.1, p. 59; BL 28e p. 1271; SAB 22e Table 33.5, p. 238

- Mechanism: DAMPs (heat shock proteins, HMGB1, S100, nucleic-acid fragments) → **pattern recognition receptors** (Toll-like, NOD-like) on macrophages, neutrophils, dendritic cells → inflammasomes, caspases → **IL-1, IL-6, TNF α** .
- Neutrophils adhere to remote endothelium → increased permeability → **MODS** → multiple system organ failure.
- Counter-regulation: IL-1Ra, soluble TNF receptors, IL-4, -5, -9, -13, TGF β ; excess gives **CARS** (immunosuppression, nosocomial infection); persistence gives PICS (persistent inflammation, immunosuppression and catabolism).

Metabolic response to injury

- **Graded response:** after major elective surgery — a transient, modest rise in temperature, heart rate, respiratory rate, energy expenditure and white cell count; after major trauma, emergency surgery, sepsis or burns — SIRS, hypermetabolism, marked catabolism, shock and even MODS; intensity also varies genetically.
- **Reprioritisation:** resources shift from peripheral tissues (**muscle, adipose tissue, skin**) to central tissues (**liver, immune system, wound**).
- **Phases** (Cuthbertson's ebb and flow):
 - **Ebb / catabolic phase** — from injury, ~**24–48 h**: hypovolaemia, ↓ basal metabolic rate, ↓ cardiac output, hypothermia, lactic acidosis; hormones: catecholamines, cortisol, aldosterone; conserves volume and energy.
 - **Flow / hypermetabolic phase** (= SIRS): tissue oedema, ↑ BMR, ↑ cardiac output, pyrexia, leukocytosis, ↑ O₂ consumption, ↑ gluconeogenesis.
 - **Anabolic phase:** repair and restoration of tissue; may last weeks.
- **Hypermetabolism:** energy expenditure **15–25% above predicted** in most trauma patients (more in burns).
- **Carbohydrate:**
 - Glycogenolysis, then gluconeogenesis (↑ up to 50%) → **hyperglycaemia**.
 - In shock insulin fails to rise and may fall; within days insulin rises but with **insulin resistance** (cytokines, ↓ glucose-transporter responsiveness) — proportional to injury; ~**2 weeks** after upper abdominal surgery; patients behave like type 2 diabetics.
 - Managed in ICU by **IV insulin infusion**, avoiding unduly tight control (hypoglycaemia).
- **Protein:**
 - Skeletal muscle proteolysis via the **ATP-dependent ubiquitin–proteasome pathway**; glutamine and alanine flow to liver, immune cells and wound.
 - Urinary nitrogen **14–20 g/day** in severe sepsis ≈ **500 g muscle/day**; death likely once 30–40% of body protein is lost.
 - Loss also from **respiratory muscles** (hypoventilation, chest infection) and gut (reduced motility); cardiac muscle mostly spared; clinically weakness, fatigue, critical illness myopathy.
 - Unlike simple starvation, where ketone adaptation rapidly reduces nitrogen loss, injury with sepsis causes '**auto-cannibalism**' (10–20 g N/day).
 - Catabolism cannot be fully reversed by feeding while the stress response continues; **hyperalimentation** is itself a stress — feed modestly; severe illness needs 1.2–2.0 g protein/kg/day to overcome **anabolic resistance**.
 - Prolonged proteolysis ↑ glutamine use → gut mucosal barrier breakdown → **microbial translocation**.
- **Fat:** catecholamine- and GH-driven **lipolysis** — fatty acids, glycerol and ketones as fuel.
- **Acute-phase response** (IL-6): positive reactants (CRP, fibrinogen) ↑; negative reactants (albumin) ↓, mainly from capillary escape.
- **Body composition:** extracellular water ↑ **6–10 L in 24 h** on resuscitation; total body protein falls ~15% in 10 days despite feeding; weight may paradoxically rise.
- **Immunity:** inflammation clears debris and fights infection, but prolonged stress causes immunosuppression → sepsis, which amplifies the response (**vicious catabolic cycle**).
- After **major elective surgery** weight and nitrogen balance can be maintained by epidural blockade of the stress response, early oral/enteral feeding and avoiding excess saline.

Neuroendocrine pathway

- **Afferent limb:** nociceptive neurones excited by direct injury and local inflammation → spinal cord → thalamus → **hypothalamus**.
 - Volume loss: carotid/aortic **baroreceptors** and left-atrial volume receptors add afferent input.

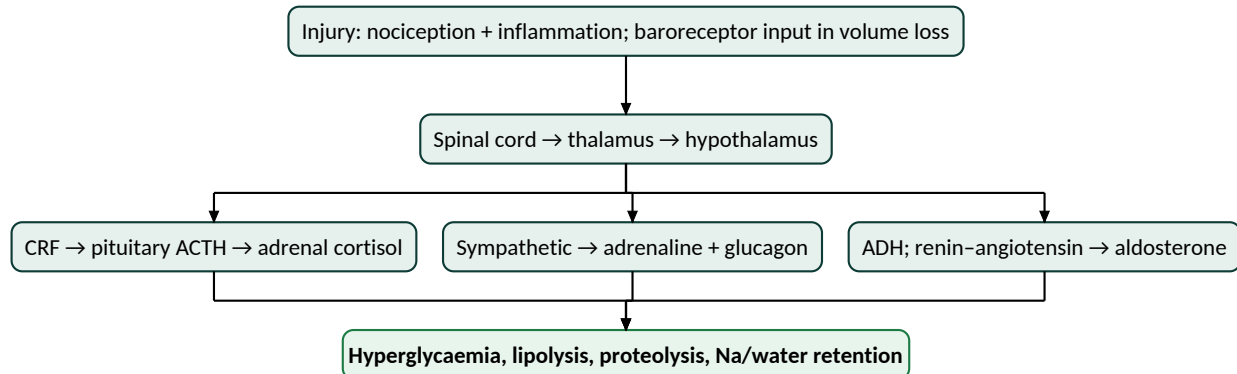
- **Magnitude** depends on the degree of tissue damage, blood loss and stimulation of somatic afferent nerves at the site of injury.
- **Hypothalamic-pituitary-adrenal axis:** hypothalamus releases **CRF** → anterior pituitary **ACTH** → adrenal cortex **cortisol** within hours (elevated 24–48 h).
 - Cortisol mobilises amino acids from skeletal muscle for healing, raises blood glucose; immunosuppressive at high levels.
- **Sympathetic activation** → adrenal medulla **adrenaline**, ↑ heart rate and BP; also stimulates **glucagon** release.
- **Salt and water:** ADH (also stimulated by pain) → water retention; ↓ pulse pressure → juxtaglomerular apparatus → **renin-angiotensin** → **aldosterone** (augmented by ACTH) → sodium retention → postoperative **oliguria**.
- Other changes: ↑ prolactin and **GH** with low IGF-1; ↓ insulin early; inactivation of peripheral thyroid hormone (↓ T3) and gonadal function (↓ testosterone).
- An infusion of **glucagon + glucocorticoid + catecholamine** reproduces much of the metabolic response.

Table 1.1b.2. Hormonal changes after injury and their effects

Hormone	Change	Main metabolic effect
Cortisol (via CRF–ACTH)	↑	Muscle catabolism, amino-acid mobilisation, hyperglycaemia; synergises with IL-6 on acute-phase response
Adrenaline / noradrenaline	↑	Glycogenolysis, lipolysis, ↑ HR and BP
Glucagon	↑	Glycogenolysis, gluconeogenesis
Growth hormone	↑	Lipolytic, insulin-antagonising, proinflammatory
ADH, aldosterone	↑	Water and sodium retention, oliguria
Insulin	↓ early, then resistance	Hyperglycaemia
IGF-1, T3, testosterone	↓	Anabolism postponed

Source: BL 28e pp. 3–5, 9; SAB 22e p. 273

Flowchart. Neuroendocrine pathway of the stress response



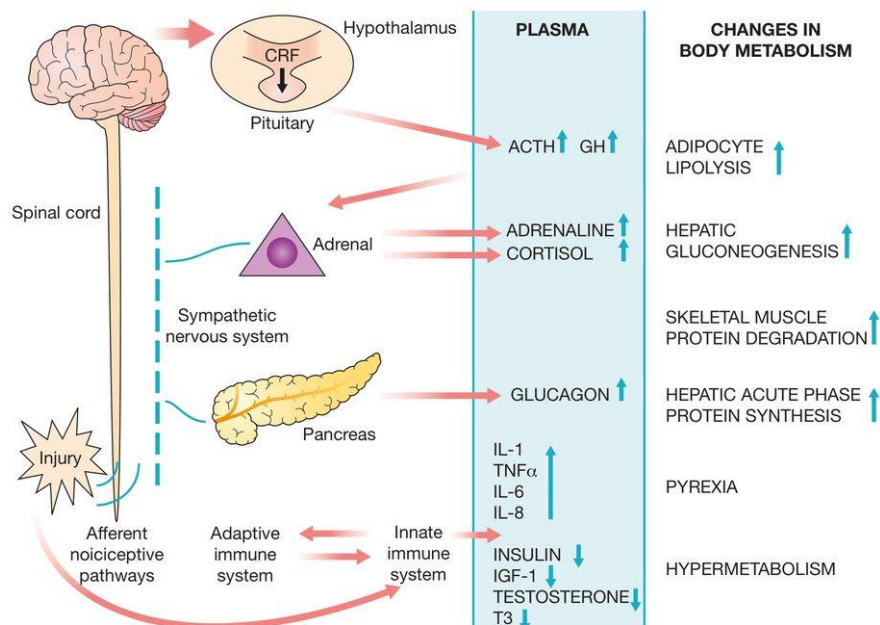


Figure 1.1b.1 — The integrated response to surgical injury (first 24–48 hours): interplay between the neuroendocrine stress response and the proinflammatory cytokine response of the innate immune system.

BL 28e Fig. 1.2, p. 4

- **Biphasic:** acute phase (hours) — high counter-regulatory hormones, beneficial for survival; chronic phase (days) — **hypothalamic suppression**, low target-hormone levels, contributing to chronic wasting.
- **Cytokine interplay:** IL-1, TNF α , IL-6, IL-8 (first 24 h) act on the hypothalamus → **pyrexia**, augment ACTH release, cause muscle proteolysis and insulin resistance; high cortisol forms a weak negative feedback.
- **Other mediators:** nitric oxide (iNOS), prostanoids (COX-2), **endothelin-1** (renal vasoconstriction), complement and kinins; **hyperglycaemia** aggravates inflammation via mitochondrial free radicals and ↑ cytokine gene expression.
- **Purpose:** provide substrates from tissue breakdown, **postpone anabolism**, optimise host defence — helpful short term, harmful if prolonged.

Pearls

- ✓ SIRS = 2 of: T >38 or <36 °C, HR >90, RR >20 (PaCO₂ <32), WBC >12 or <4 × 10⁹/L.
- ✓ Ebb (24–48 h, hypometabolic) → flow (hypermetabolic, = SIRS) → anabolic.
- ✓ Classic pathway: nociception → hypothalamus (CRF) → pituitary (ACTH) → adrenal (cortisol) + sympathetic (adrenaline, glucagon).
- ✓ Muscle wasting is via the ubiquitin–proteasome pathway.
- ✓ Postoperative oliguria = ADH + aldosterone; worsened by saline.
- ✓ Exam trap: albumin falls mainly from capillary leak, not reduced synthesis.

Sources

- BL 28e, ch. 1 “Metabolic response to injury”, §“Introduction; magnitude; mediators; neuroendocrine response; metabolic changes; managing the catabolic stress response”, pp. 1–7; Fig. 1.2, p. 4.
- BL 28e, ch. 1 “Metabolic response to injury”, §“Changes in body composition; volume loss”, pp. 8–9.
- BL 28e, ch. 5 “Surgical infection”, §“Systemic inflammatory response syndrome; Table 5.1”, pp. 58–59.
- BL 28e, ch. 72 “The pancreas”, §“Acute pancreatitis: SIRS criteria”, p. 1271.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Sepsis definitions; Table 33.5 SIRS criteria”, p. 238.
- SAB 22e, ch. 34 “Metabolism and Nutrition in Surgical Patients”, §“The metabolic response to surgical stress and critical illness; neuroendocrine response”, pp. 272–273.
- SAB 22e, ch. 22 “Enhanced Recovery After Surgery (ERAS): Evidence-Based P”, §“Stress reactions to surgery”, p. 77.

1.2a Classification of shock

1×

Asked in: Paper I · Aug 2021 · Q1(b) (5 marks)

“Classification of shock.”

- **Shock:** a systemic state of **low tissue perfusion inadequate for normal cellular respiration** → anaerobic metabolism, lactic acidosis and, if not reversed, cell death.
- Most clinically useful classification is by **initiating mechanism**; all types share systemic hypoperfusion and **may coexist** in one patient.

Classification by mechanism (BL)

- **1. Hypovolaemic / haemorrhagic** — reduced circulating volume; the **commonest** type and a component of all others.
 - Haemorrhagic: revealed or concealed blood loss.
 - Non-haemorrhagic: poor intake (dehydration), vomiting, diarrhoea, urinary loss (e.g. diabetes), evaporation, **third-spacing** (bowel obstruction, pancreatitis).
- **2. Cardiogenic** — primary pump failure: myocardial infarction, dysrhythmias, valvular disease, blunt myocardial injury, cardiomyopathy, myocardial depression from sepsis or drugs; venous hypertension with pulmonary/systemic oedema.
 - Haemodynamic criteria (SCH): SBP <90 mmHg for ≥30 min, cardiac index <2.2 L/min/m², pulmonary artery wedge pressure >15 mmHg; complicates 5–10% of acute MIs.
- **3. Obstructive** — mechanical obstruction to cardiac filling (↓ preload): **cardiac tamponade, tension pneumothorax**, massive pulmonary or air embolus.
 - Other causes: IVC obstruction (DVT, gravid uterus, neoplasm), raised intrathoracic pressure (excess PEEP); low cardiac output with **raised CVP**.
- **4. Distributive** — vasodilatation, low SVR, inadequate afterload, **high cardiac output**:
 - **Septic** — endotoxin and immune activation; microvascular maldistribution, arteriovenous shunting, impaired cellular O₂ use; later hypovolaemia and myocardial depression.
 - **Anaphylactic** — histamine release.
 - **Neurogenic** — high spinal cord injury (or spinal/epidural anaesthetic): loss of sympathetic outflow and vascular tone; bradycardia with injuries at C5 and above; “warm” shock.
 - Septic-type vasodilatory shock also follows non-infective inflammation — pancreatitis, burns, adrenal insufficiency, prolonged severe hypotension.
 - Peripheries are **warm with brisk capillary refill** despite profound shock.
- **5. Endocrine** — hypo- or hyperthyroidism, adrenal insufficiency (Addison’s or relative, as in sepsis); presents as a mix of hypovolaemic, cardiogenic or distributive patterns.

Table 1.2a.1. Cardiovascular and metabolic characteristics of shock

Variable	Hypovolaemic	Cardiogenic	Obstructive	Distributive
Cardiac output	Low	Low	Low	High
Systemic vascular resistance	High	High	High	Low
Venous pressure	Low	High	High	Low
Mixed venous saturation	Low	Low	Low	High
Base deficit	High	High	High	High

Source: BL 28e Table 2.1, p. 12

Other examinable schemes

- **Schwartz (aetiological, six types):** hypovolaemic, **septic (vasodilatory)**, neurogenic, cardiogenic, obstructive and **traumatic** shock — Blalock’s original four were hypovolaemic, vasogenic, cardiogenic and neurogenic.
- **Traumatic shock:** hypovolaemia plus soft-tissue injury/long-bone fracture → magnified proinflammatory activation (DAMPs via pattern recognition receptors); MODS and ARDS commoner than after pure haemorrhage.
- **Sabiston (four categories):** distributive, hypovolaemic, cardiogenic, obstructive; shown by anatomical level — heart (extrinsic tamponade, intrinsic failure), large/medium vessels (haemorrhage), small vessels (sepsis, neurogenic).

- **By severity (BL):**

- **Compensated** — tachycardia, cool peripheries, BP maintained, narrowed pulse pressure; occult gut/muscle hypoperfusion.
- **Mild** — tachycardia, tachypnoea, mild oliguria, anxiety; **moderate** — urine <0.5 mL/kg/h, falling BP, drowsiness; **severe** — profound hypotension, anuria, coma.
- **Unresuscitatable (irreversible)** — loss of response to fluids, inotropes and vasopressors.

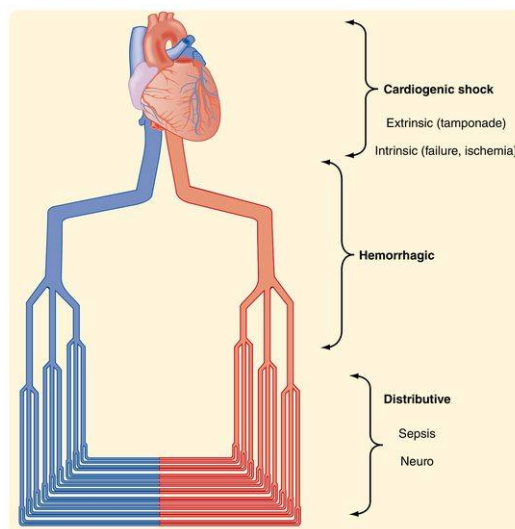


Figure 1.2a.1 — Types of shock by level of the cardiovascular system: cardiogenic (extrinsic, intrinsic), haemorrhagic, distributive (sepsis, neurogenic).

SAB 22e Fig. 33.5, p. 227

Pearls

- ✓ BL's five: hypovolaemic, cardiogenic, obstructive, distributive, endocrine.
- ✓ Hypovolaemia is the commonest and is part of every type — exclude it first.
- ✓ Distributive is the only type with high cardiac output, low SVR and high SvO₂.
- ✓ Obstructive and cardiogenic both have high venous pressure.
- ✓ Neurogenic shock: hypotension with bradycardia; spinal shock is loss of reflexes, not a haemodynamic state.

Sources

- BL 28e, ch. 2 "Shock, haemorrhage and transfusion", § "Shock: pathophysiology; classification of shock; recognition and diagnosis", pp. 11–14; Table 2.1, p. 12.
- SAB 22e, ch. 33 "Shock, Electrolytes, and Fluid", § "Physiology of shock; types of shock", pp. 226–227; Fig. 33.5, p. 227.
- SAB 22e, ch. 46 "Surgical Critical Care", § "Surgical critical care: shock; distributive shock", pp. 533–534.
- SCH 11e, ch. 5 "Shock", § "Overview; historical background; Table 5-1 classification of shock", pp. 131–132.
- SCH 11e, ch. 5 "Shock", § "Traumatic shock; septic (vasodilatory) shock; cardiogenic shock; obstructive shock; neurogenic shock", pp. 145–151.

Asked in: Paper I · Oct 2023 · Q1(b) (4 marks) ; Paper I · May 2024 · Q4(a) (5 marks)

“Classification and management of hemorrhagic shock.”

“Classification of hemorrhagic shock.”

- **Haemorrhagic shock:** hypovolaemic shock from uncontrolled bleeding; adult blood volume $\approx$ **5 L (70 mL/kg; 80 mL/kg in neonates)**.
- Haemorrhage and shock are not the same: a bleeding patient may not yet be shocked, and a shocked patient may have stopped bleeding.

Classification (ATLS classes)

Table 1.2b.1. Classes of haemorrhagic shock

Parameter	Class I	Class II	Class III	Class IV
Blood loss (%)	<15	15–30	30–40	>40
Blood loss (mL, 70 kg)	<750	750–1500	1500–2000	>2000
Pulse (beats/min)	<100	>100	>120	>140
Blood pressure	Normal	Normal (orthostatic)	Decreased	Decreased
Pulse pressure	Normal	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Urine (mL/h)	>30	20–30	5–15	Negligible
CNS	Slightly anxious	Mildly anxious	Anxious / confused	Confused / lethargic
Fluid	Crystalloid	Crystalloid	Crystalloid + blood	Crystalloid + blood

Source: BL 28e Table 2.3, p. 15; SAB 22e Table 33.1, p. 226; SCH 11e Table 5-5, p. 142

- ~15% loss is within normal compensation; BP usually falls only after **30–40%** loss; **40% loss (2 L)** is immediately life-threatening and usually needs operative control.
- **Limitations:** arbitrary, untested and **difficult to apply clinically** (BL: never applied clinically).
 - Young patients compensate well then collapse; the elderly decompensate early; β -blockers, pacemakers and pain confound the signs.
 - Tachycardia is unreliable (present in only 65% of hypotensive trauma patients); some bleeding patients show **relative bradycardia** (HR <100 with SBP <90 mmHg) — lower mortality.
 - SBP **<110 mmHg** may better define hypotension; haemoglobin is a poor early indicator (a concentration); a low Hb, when present, reflects severe loss.
 - **Shock index** (HR/SBP) and modified shock index (HR/MAP) grade severity better than HR or BP alone.

Management

- **Assume** shock is hypovolaemic and hypovolaemia is haemorrhagic until proved otherwise; **activate the major haemorrhage protocol**.
- **Airway and breathing first; large-bore IV access;** cross-match; start with **emergency group O blood**.
- **Fluid response** identifies active bleeding: **responder; transient responder** and **non-responder** are still bleeding → find and control the source.
- **Locate** the bleeding by rapid bedside tests suited to physiology (chest and pelvic radiographs, abdominal ultrasound); CT only when stable.
- **Damage control resuscitation** (while bleeding):
 - **Rapid haemorrhage control** — direct pressure, tourniquets, balloon occlusion; move to theatre or interventional radiology; **damage control surgery** (arrest haemorrhage, control sepsis, protect from further injury, nothing else).
 - **Permissive hypotension** — palpable central pulse, MAP $\sim$ 50 mmHg (SCH: SBP 80–90 mmHg in penetrating injury; $\sim$ 110 mmHg with brain injury).
 - **Avoid dilutional coagulopathy** — avoid crystalloids and colloids; balanced transfusion **1:1:1** red cells : plasma : platelets.

- **Treat coagulopathy** — **tranexamic acid** as early as possible; cryoprecipitate for low fibrinogen; platelets; guide by PT, fibrinogen, ROTEM/TEG.
- Monitor and correct Ca^{2+} , K^+ , pH, lactate, temperature; warm fluids and patient.
- **Transfusion and fluids:**
 - In severe haemorrhage restore volume with **blood products**; colloids increased mortality versus crystalloid in bleeding trauma patients.
 - Massive transfusion (>10 units in 24 h) with a **high plasma-to-red-cell ratio** improves survival; target Hb 7–9 g/dL only in stable ICU patients.
- **Time matters:** each delay in reaching theatre raises mortality (~1% per 3 minutes in the emergency department for abdominal bleeding); the actively bleeding patient cannot be resuscitated until bleeding is controlled.
- **Irreversible shock:** ongoing fluid needs and vasopressor-resistant vasodilatation despite haemorrhage control, with uncorrectable hypothermia, acidosis and coagulopathy.
- **After haemostasis** — perfusion-targeted resuscitation: adequate preload and afterload (fluids, pressors), rewarming, correction of coagulopathy, thromboprophylaxis; end points lactate, base excess, SvO_2 , urine output.

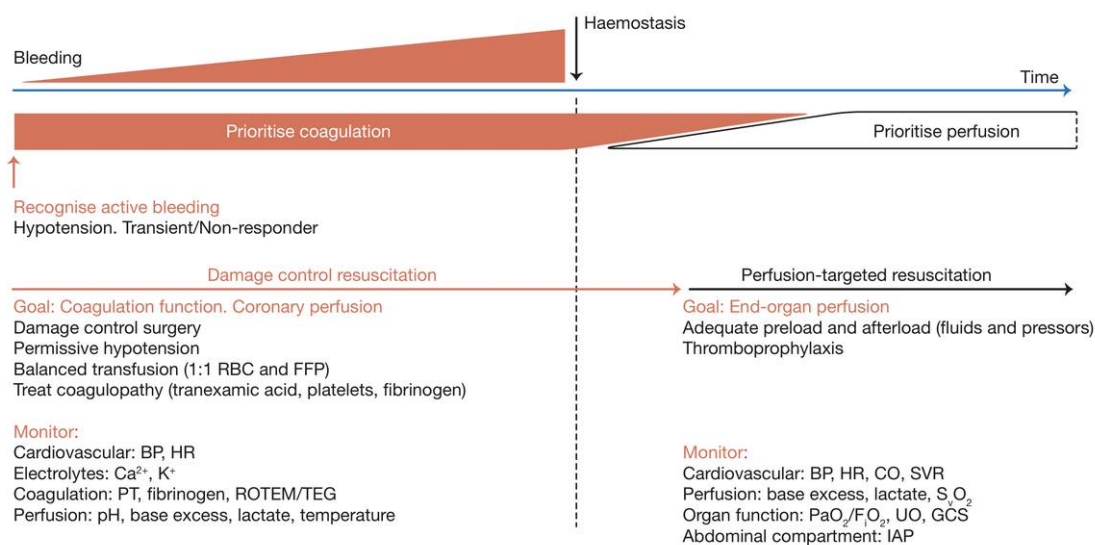


Figure 1.2b.1 — Haemorrhage resuscitation: damage control resuscitation until haemostasis, then perfusion-targeted resuscitation.
BL 28e Fig. 2.2, p. 16

Pearls

- ✓ Class III (30–40%) is when BP first falls — a ‘six-pack’ (~2 L) of blood lost.
- ✓ Haemorrhage is treated by stopping the bleeding, not by fluid or transfusion.
- ✓ DCR = rapid haemorrhage control + permissive hypotension + balanced 1:1:1 transfusion + treat coagulopathy (TXA).
- ✓ Transient and non-responders are still bleeding.
- ✓ Lethal triad: acidosis, hypothermia, coagulopathy.

Sources

- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Haemorrhage; degree of haemorrhage and classification; haemorrhage resuscitation; damage control resuscitation”, pp. 14–17; Table 2.3, p. 15; Fig. 2.2, p. 16.
- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Management of coagulopathy”, p. 23.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Physiology of shock: bleeding; ATLS classes”, pp. 226–228; Table 33.1, p. 226.
- SCH 11e, ch. 5 “Shock”, §“Hypovolemic/hemorrhagic shock: diagnosis and treatment”, pp. 141–145; Table 5-5, p. 142.

Asked in: Paper I · Dec 2025 · Q9(b) (5 marks) ; Paper I · Dec 2020 · Q6(a) (3 marks)

“Classify hemorrhage according to time and source.”

“Pathophysiology of hemorrhage.”

Pathophysiology of haemorrhage

• Compensation:

- ↓ pressure at **baroreceptors** (aortic arch, atria, carotid) → ↑ sympathetic activity, adrenaline and noradrenaline → tachycardia, ↑ contractility and **vasoconstriction**.
- Blood shunted from skin, muscle and splanchnic bed to brain, heart and kidneys.
- **Renin-angiotensin-aldosterone** and **ADH** → sodium and water retention, ↓ urine output; cortisol released.
- **Fluid shift** from interstitial and intracellular spaces into vessels → later fall in haemoglobin/haematocrit (unchanged early as whole blood is lost).

• Respiratory: acidosis and sympathetic drive → ↑ respiratory rate and minute ventilation → compensatory respiratory alkalosis.

• Capillary refill of the circulation: precapillary sphincters constrict more than postcapillary → capillary hydrostatic pressure falls → interstitial fluid recruited; hormone release is finite and is exhausted in ongoing shock.

• Metabolic: catecholamines → **hyperglycaemia**.

• Cellular: hypoperfusion → anaerobic metabolism → **lactic acidosis**; failure of Na^+/K^+ pumps, lysosomal enzyme release, cell lysis, **hyperkalaemia**.

• Microvascular: complement and leucocyte activation, free radicals, cytokines → leaky endothelium, tissue oedema.

• Decompensation: ~15% loss is compensated; BP falls after 30–40% loss; prolonged shock → unresuscitatable state, ischaemia-reperfusion injury, MODS.

• Coagulopathy:

- **Acute traumatic coagulopathy (ATC)** — tissue trauma + shock; up to **25%** of trauma patients within minutes; hyperfibrinolysis, low fibrinogen, platelet dysfunction; fourfold mortality.
 - Evolves into **trauma-induced coagulopathy** with dilution by fluids/red cells; underperfused muscle cannot generate heat → **hypothermia**, worsened by cold fluids and open body cavities; acidosis and hypothermia inhibit coagulation proteases.
 - Result: **lethal triad** (acidosis, hypothermia, coagulopathy) and a vicious circle of further bleeding; repeated volume resuscitation while bleeding → physiological exhaustion and death.
- **Arterial vs venous:** arterial bleeding drops BP early (may stop by spasm and thrombosis; a lacerated artery bleeds more than a transected one); slower venous bleeding allows compensation, so large losses and abnormal lactate precede hypotension.

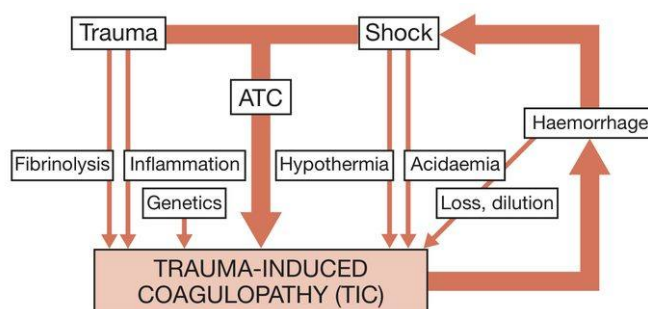


Figure 1.2c.1 — Trauma-induced coagulopathy (ATC, acute traumatic coagulopathy).

BL 28e Fig. 2.1, p. 15

Classification of haemorrhage — by time and by source

• By time:

- **Primary** — occurs immediately as a result of injury or during operation.

- **Reactionary** — delayed, **within 24 hours** (24–48 h after operation); dislodgement of clot as resuscitation normalises BP and vessels dilate, or technical failure — slipped ligature, missed vessel injury.
- **Secondary** — **7–14 days** (days to weeks) after injury; sloughing of the vessel wall from **infection**, pressure necrosis (e.g. from a drain) or malignancy; deep-seated infection eroding a vessel.
- **By source / visibility:**
 - **Revealed** — obvious external bleeding: open arterial wound, massive haematemesis from a duodenal ulcer.
 - **Concealed** — contained in a body cavity; must be suspected, actively investigated and controlled: **chest, abdomen, pelvis, retroperitoneum, limbs** (contained vascular injury, long-bone fractures); non-traumatic — occult GI bleeding, ruptured aortic aneurysm.
 - Five sites can hide shock-producing loss: external, intrathoracic, intra-abdominal, retroperitoneal, long-bone fractures.
- **By vessel:** arterial, venous or capillary bleeding.
 - Arterial: bright, pulsatile, early hypotension; venous: slower, larger loss before hypotension.
- **By need for surgery:**
 - **Surgical** — direct vessel injury amenable to suture ligation or angioembolisation.
 - **Non-surgical** — general bleeding from raw surfaces and mucous membranes due to **coagulopathy**; cannot be stopped surgically (except packing); treat by correcting clotting.
- **Degree:** classes I–IV by % blood volume lost (<15%, 15–30%, 30–40%, >40%).
- **Response to fluid:** responder, transient responder, non-responder — the last two are still bleeding.

Pearls

- ✓ Primary – at once; reactionary – within 24 h; secondary – 7–14 days (infection).
- ✓ Reactionary bleeding is unmasked when BP rises after resuscitation.
- ✓ Haemoglobin is a concentration — normal early in acute bleeding.
- ✓ ATC develops within minutes in up to 25% of trauma patients.
- ✓ Non-surgical bleeding is treated by correcting coagulopathy, not suturing.

Sources

- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Shock pathophysiology; haemorrhage: pathophysiology, definitions, degree of haemorrhage”, pp. 11–16; Fig. 2.1, p. 15.
- BL 28e, ch. 7 “Basic surgical skills”, §“Basic haemostatic methods: primary, reactionary, secondary haemorrhage”, p. 110.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Physiology of shock: bleeding; arterial vs venous bleeding; compensatory mechanisms; lethal triad”, pp. 226–230.

1.2d Monitoring of a patient in shock

1×

Asked in: Paper I · Dec 2022 · Q1(b) (5 marks)

“Monitoring of patient in shock.”

- Aim: restore **cellular and organ perfusion**; monitoring tracks cardiovascular response and perfusion and guides fluids, vasopressors and inotropes.
- **Minimum:** continuous ECG/heart rate, **pulse oximetry**, frequent non-invasive BP, **hourly urine output** (catheter).
- **Additional:** central venous pressure, invasive arterial BP, cardiac output, **base deficit and serum lactate** — for shock not rapidly corrected by a little fluid.

What to measure and targets

Table 1.2d.1. Monitoring in shock

Monitor	What it shows	Interpretation / target
Heart rate, BP (arterial line)	Cardiovascular response	Hypotension is a late sign; arterial line for continuous BP
CVP	Response to a fluid challenge (250–500 mL over 5–10 min)	No rise = empty; rise 2–5 cmH ₂ O drifting back in 10–20 min = normal; large sustained rise = high preload / cardiac insufficiency; no single “normal” value
Cardiac output (Doppler, pulse contour, indicator dilution, PA catheter)	CO, SVR, preload	Distinguishes coexisting hypovolaemic, distributive, cardiogenic shock; needed with vasopressors/inotropes
Pulse pressure variation (ventilated)	Preload responsiveness	(PPmax – PPmin)/mean × 100; best predictor of fluid response
Urine output	Renal (organ) perfusion	Best single measure of adequacy of therapy; <0.5 mL/kg/h = moderate shock
Conscious level	Cerebral perfusion	Preserved until late — poor guide
Lactate, base deficit (ABG)	Global perfusion, O ₂ debt	Base deficit >6 mmol/L → higher morbidity/mortality; time to normalise matters
Mixed venous O ₂ saturation	O ₂ delivery vs extraction	Normal 50–70%; <50% hypovolaemic/cardiogenic; >70% expected in sepsis
Regional: NIRS (StO ₂), tissue O ₂ , sublingual capnometry, gastric tonometry	Tissue-bed perfusion	StO ₂ ≤75% predicts MODS like base deficit ≥6

Source: BL 28e pp. 18–20, Table 2.4; SCH 11e pp. 152–153, 444–445

Practical points

- **Arterial line and CVP** when shock does not correct with a small amount of fluid; long narrow central lines are for **monitoring, not rapid infusion**.
- **CVP pitfalls:** ventricular compliance changes minute to minute, so CVP poorly reflects end-diastolic volume; some patients need 5 cmH₂O, others ≥15; CVP/PAOP readings of 5–20 mmHg are least informative.
- **Cardiac output:** pulmonary artery catheters are used less as Doppler, pulse waveform analysis, lithium/thermal indicator dilution and transoesophageal echo give similar data; start early in patients on vasopressors or inotropes.
- **Urine output** is hourly — not a minute-to-minute view; the level of consciousness is preserved until very late.
- **SvO₂:** true mixed venous sample from the right atrium/pulmonary artery; SVC samples read slightly higher. In sepsis, SvO₂ <70% means coexisting hypovolaemia or cardiac failure → fluids and **dobutamine** to >70%.
- **Lactate** is cleared by the liver (~50%) and kidney (~30%); admission level, peak and time to clearance are prognostic.
- **ABG** also gives pH, PaO₂, PaCO₂, HCO₃[−], carboxy- and methaemoglobin.

End points of resuscitation

- Normal pulse, BP and urine output reflect organs perfused until late; **occult hypoperfusion** (normal vital signs, persistent lactic acidosis, low SvO₂) persists in **80–85%** of trauma patients.
- Occult hypoperfusion **>12 hours** → two to three times the mortality and more infection and organ failure.
- Resuscitate to **global perfusion end points** — lactate, base deficit, SvO₂; lactate normalising within 24 h → 100% survival vs 14% if >48 h.
- **Base deficit** severity: mild 3–5, moderate 6–14, severe ≥15 mmol/L (≥15 → ~70% mortality); confounded by bicarbonate, hypothermia, hypocapnia, heparin, ethanol, ketoacidosis.
- Dynamic measures (passive leg raise, fluid challenge, PPV) have replaced static CVP for fluid responsiveness; **MAP target 65 mmHg** in septic shock.
- During active bleeding also monitor **Ca²⁺, K⁺, PT, fibrinogen, ROTEM/TEG, temperature**; after haemostasis PaO₂/FiO₂, GCS and **intra-abdominal pressure**.
- Over-aggressive crystalloid → tissue oedema, ARDS, abdominal compartment syndrome, cerebral oedema.

Pearls

- ✓ Minimum monitoring: ECG, SpO₂, BP, hourly urine output.
- ✓ CVP is read dynamically as the response to a fluid challenge.
- ✓ Urine output is the best clinical monitor of resuscitation adequacy.
- ✓ Normal vital signs ≠ resuscitated: check lactate, base deficit, SvO₂.
- ✓ Low SvO₂ in sepsis means coexisting hypovolaemia or cardiac failure.

Sources

- BL 28e, ch. 2 "Shock, haemorrhage and transfusion", §"Recognition and diagnosis of shock; clinical features", pp. 13–14.
- BL 28e, ch. 2 "Shock, haemorrhage and transfusion", §"Shock resuscitation: monitoring; cardiac output; systemic and organ perfusion; end points of resuscitation", pp. 17–20; Table 2.4, p. 19.
- SAB 22e, ch. 46 "Surgical Critical Care", §"Septic shock: hemodynamic measures of volume status", p. 534.
- SCH 11e, ch. 5 "Shock", §"Endpoints in resuscitation: lactate, base deficit, NIR spectroscopy", pp. 152–153; Table 5-11, p. 152.
- SCH 11e, ch. 13 "Physiologic Monitoring of the Surgical Patient", §"Assessing preload responsiveness; pulse pressure variation; NIRS", pp. 444–445.

1.3a Blood products (enumerate)

1×

Asked in: Paper I · Dec 2020 · Q6(b) (2 marks)

"Enumerate different blood products."

- Donated blood (UK: up to 450 mL, maximum three times a year) is screened for **hepatitis B, hepatitis C, HIV-1, HIV-2 and syphilis**, ABO/RhD grouped, **leukodepleted** and processed into components.

Blood products

- **1. Whole blood** — rarely available in civilian practice; coagulation-factor rich and, if fresh, more metabolically active; cold-stored whole blood (4 °C in CPDA, up to 35 days; low-titre group O) is used in military and some trauma practice.
- **2. Packed red blood cells** — ~**330 mL**, haematocrit **50–70%**, stored in **SAGM** at **2–6 °C** for **5 weeks** (CPD storage: 2–3 weeks); for acute blood loss, perioperative and symptomatic anaemia.
- **3. Fresh-frozen plasma (FFP)** — all coagulation factors, fibrinogen, albumin, proteins C and S, antithrombin; stored at **–40 to –50 °C**, **2-year** shelf life; first-line for coagulopathic haemorrhage; must be thawed.
- **4. Cryoprecipitate** — precipitate of FFP rich in **fibrinogen, factor VIII, factor XIII**, vWF, fibronectin; **–30 °C**, 2 years; for low fibrinogen or factor VIII deficiency; smaller volume than FFP.
- **5. Platelets** — pooled concentrate (~250 × 10⁹/L), stored on an agitator at **20–24 °C** for only **5 days**; for thrombocytopenia or platelet dysfunction with bleeding or surgery.
- **6. Prothrombin complex concentrate (PCC)** — pooled plasma concentrate of **factors II, VII, IX, X** (with proteins C and S); emergency reversal of warfarin; less volume and fewer reactions than FFP.
- **7. Autologous blood** — pre-donation up to 3 weeks before elective surgery, or intraoperative **cell salvage** (washed red cells returned).
- **8. Others:** plasma-derived or recombinant **factor VIII and IX concentrates**; **lyophilised plasma**; **cold-stored platelets** (up to 21 days).
- **Blood substitutes** (investigational): haemoglobin-based (biomimetic) and perfluorocarbon-based (abiotic) oxygen carriers.

Pearls

- ✓ Platelets: room temperature (20–24 °C), shelf life only 5 days.
- ✓ Packed cells: SAGM, 2–6 °C, 5 weeks.
- ✓ Cryoprecipitate = fibrinogen + VIII + XIII.
- ✓ PCC (II, VII, IX, X) is first-line for warfarin reversal.

Sources

- BL 28e, ch. 2 "Shock, haemorrhage and transfusion", §"Transfusion: blood and blood products; autologous blood", pp. 20–21.
- BL 28e, ch. 2 "Shock, haemorrhage and transfusion", §"Blood substitutes", p. 23.
- SAB 22e, ch. 100 "Hemostasis and Thrombosis", §"Common hemostatic medications: plasma, prothrombin complex, cryoprecipitate", pp. 1588–1589.
- SAB 22e, ch. 100 "Hemostasis and Thrombosis", §"Hemophilias: factor concentrates", p. 1590.
- SAB 22e, ch. 33 "Shock, Electrolytes, and Fluid", §"Whole blood resuscitation", p. 246.

1.3b

Complications of blood transfusion and of massive transfusion; precautions in massive transfusion

3×

Asked in: Paper I · Jun 2025 · Q1 (5+5 marks) ; Paper I · Aug 2021 · Q2(b) (5 marks) ; Paper I · Dec 2020 · Q6(c) (5 marks)

“Discuss in brief complications of blood transfusion. Describe special precautions in event of massive blood transfusion.”

“Complications of blood transfusion.”

“What are the common complications of massive blood transfusion?”

- Complications are grouped into those of a **single transfusion** and those of **massive transfusion**; repeated transfusion over long periods adds **iron overload**.
- Transfusion also carries an **immunological price** — increased morbidity and reduced survival in trauma and malignancy — so it must be judicious; restrictive trigger **Hb 7 g/dL** in most stable patients (8 g/dL in stable cardiovascular disease).

Complications of blood transfusion

- **Immunological — haemolytic:**
 - **Acute haemolytic (incompatibility) reaction** — ABO-incompatible cells meet recipient antibodies → **complement-mediated intravascular haemolysis**, potentially fatal, with multiple organ failure; minimised by strict identity checks.
 - Reactions from other antigen systems are milder and self-limiting; **RhD sensitisation** (e.g. RhD-positive FFP to an RhD-negative woman) — anti-D can later cause haemolytic disease of the fetus.
- **Immunological — non-haemolytic:**
 - **Febrile non-haemolytic reaction** — the commonest; caused by donor leucocytes; fever, chills, rigors; rare with leukodepleted blood.
 - **Allergic / anaphylactic reactions**; plasma and cryoprecipitate share the same reaction profile (haemolytic, anaphylactic, febrile, TACO, TRALI).
 - **Transfusion-related acute lung injury (TRALI)** — usually from **FFP**; higher plasma volumes also raise infection, sepsis, multiple organ failure and ARDS.
 - **Immunosuppression** — more infection; stored older red cells (storage lesion) impair immune response.
- **Infective:**
 - **Bacterial** contamination — usually faulty storage.
 - **Viral: hepatitis B, hepatitis C, HIV** (every unit tested for HBV, HCV, HIV-1, HIV-2 and syphilis); **malaria**; variant CJD (reason for leukodepletion).
- **Non-immunological:**
 - **Transfusion-associated circulatory overload (TACO)** — fluid overload; FFP volume can precipitate heart failure in elderly cardiac patients (PCC needs less volume).
 - **Air embolism; thrombophlebitis** at the infusion site.
 - **Hyperkalaemia; iron overload** with repeated transfusion — each red-cell unit carries ~**250 mg** of elemental iron (e.g. thalassaemia).

Table 1.3b.1. Approximate risk per unit of red-cell transfusion

Adverse event	Approximate risk
Febrile reaction	1 : 60
Transfusion-associated circulatory overload	1 : 100 (per recipient)
Allergic reaction	1 : 250
Transfusion-related acute lung injury	1 : 12 000
Hepatitis C	1 : 1 149 000
Hepatitis B	1 : 1 208 000 to 1 : 843 000
HIV	1 : 1 467 000
Fatal haemolysis	1 : 1 972 000

Source: SAB 22e Table 19.12, p. 16

- **Recognition and first action:** monitor every transfusion closely; **stop the transfusion immediately** if any reaction is suspected (e.g. fever, chills, rigors).

Complications of massive transfusion

- **Massive transfusion:** >10 units of packed red cells in the first 24 hours.
- **Coagulopathy:**
 - **Dilutional** — red cells and clear fluids dilute coagulation factors and platelets; worsens pre-existing acute traumatic coagulopathy.
 - **Fibrinogen** is the first factor to be depleted; hypofibrinogenaemia predicts the need for massive transfusion and death.
- **Hypocalcaemia:**
 - **Citrate** anticoagulant chelates ionised calcium (iCa^{2+} falls while total Ca may be normal), especially with rapid transfusion.
 - Ionised calcium <0.6 mmol/L makes platelets less effective.
- **Hyperkalaemia and hypokalaemia.**
- **Hypothermia:**
 - Red cells are stored at 4 °C: **2 L of cold blood** lowers core temperature by ~1 °C.
 - Hypothermia and **acidosis** inhibit coagulation proteases → **lethal triad** (acidosis, hypothermia, coagulopathy) and further bleeding.
- **Metabolic derangement** — acidosis; repeated volume cycles while bleeding cause physiological exhaustion.
- **Older stored red cells** (>22 days) in massive transfusion are associated with higher 24-hour mortality; massive red-cell transfusion is linked to **MODS**.
- **Lung injury and volume overload** — TRALI, ARDS (higher plasma volumes), TACO; also infection and sepsis.

Special precautions in massive transfusion

- **Major haemorrhage / massive transfusion protocol** activated early; every trauma hospital must have one.
- **Right blood, right patient:**
 - Group and cross-match on arrival: full cross-match up to **45 min**; **type-specific** (ABO/RhD) blood in **10–15 min**; emergency **group O** (O– to females, O+ to males) until then.
 - Two staff check patient details against prescription, unit label and serial number on the issue slip.
- **Balanced (haemostatic) resuscitation:**
 - **1:1:1** red cells : FFP : platelets, approximating whole blood; whole blood if available.
 - **Avoid crystalloids and colloids** — small volumes only to maintain BP until blood arrives.
 - Permissive hypotension (SBP 70–90 mmHg; >90 mmHg with head injury) until bleeding is controlled.
- **Antifibrinolytic: tranexamic acid 1 g IV over 10 min, then 1 g over 8 h**, as early as possible (ideally within 1 h); not started >3 h after injury.
 - Children: 15 mg/kg bolus then 15 mg/kg over 8 h.
- **Targeted component replacement:**
 - **Cryoprecipitate** for low fibrinogen; **platelets** for low count or dysfunction; **PCC** for warfarin reversal.
 - Paediatric targets (BL): Hb >80 g/L, platelets >75 × 10⁹/L, fibrinogen >1.5 g/L, APTT/PT <1.5 × normal.
- **Prevent hypothermia:**
 - **Warm all products** with in-line warmers up to the point of insertion (maximum 40 °C) — the main purpose is to avoid cooling the patient.
 - Dry and cover the patient (head cover), warm the room, close body cavities, control the bleeding.
- **Monitor frequently:**
 - **Ionised Ca²⁺ and K⁺**, ABG (pH, base excess, lactate), temperature, BP, heart rate.
 - Clotting — PT, fibrinogen and **viscoelastic tests (TEG/ROTEM)**; a cold patient is more coagulopathic than the laboratory result shows (samples are warmed to 37 °C).
- **Transfuse on clinical grounds** in active bleeding — tachycardia, hypotension, lactate — because a falling Hb is a late sign.
- **Evidence:** TXA reduced all-cause mortality (14.5% vs 16.0%, CRASH-2) but is less effective or harmful when delayed >3 h; TEG-guided resuscitation lowered mortality versus empiric MTP (33.3% vs 54.1%) in penetrating injury needing

>10 units; 1:1:1 achieved more haemostasis and fewer exsanguination deaths than 1:1:2 (PROPPR).

- **Giving set:** use normal saline, not Ringer's lactate, in the blood line (regulatory policy — calcium binds citrate); use **leukoreduced** components.
- **After haemostasis:** stop the protocol, correct residual coagulopathy, warm, and start **thromboprophylaxis**; watch for TRALI/TACO and infection.

Table 1.3b.2. Viscoelastic (TEG) guide to component replacement

TEG parameter	Reflects	If abnormal, give
R time prolonged	Coagulation factor activity (time to clot formation)	Plasma (FFP)
K time prolonged / α angle low	Fibrin cross-linking, fibrinogen	Cryoprecipitate
Maximum amplitude low	Clot strength — platelet function	Platelets (or desmopressin)
LY30 raised	Fibrinolysis	Tranexamic acid

Source: SAB 22e p. 1588; Fig. 46.1, p. 546

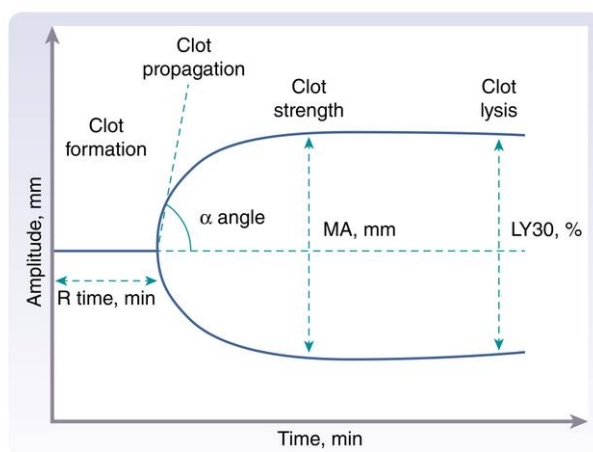


Figure 1.3b.1 — Thromboelastogram parameters: R time (clot formation), α angle (propagation), maximum amplitude (strength), LY30 (lysis).

SAB 22e Fig. 46.1, p. 546

Pearls

- ✓ Single-transfusion complications: haemolytic, febrile, allergic, infection, air embolism, thrombophlebitis, TRALI (usually FFP).
- ✓ Massive transfusion (>10 units/24 h): coagulopathy, hypocalcaemia (citrate), hyper-/hypokalaemia, hypothermia.
- ✓ Commonest reaction: febrile non-haemolytic; most feared: ABO-incompatible intravascular haemolysis.
- ✓ Precautions: MTP 1:1:1, early TXA (<3 h), warm everything, check $\text{Ca}^{2+}/\text{K}^{+}$, TEG-guided correction.
- ✓ 2 L of cold blood cools the patient by 1 °C.

Sources

- BL 28e, ch. 2 "Shock, haemorrhage and transfusion", § "Damage control resuscitation: avoid dilutional coagulopathy; treat coagulation deficits", p. 17.
- BL 28e, ch. 2 "Shock, haemorrhage and transfusion", § "Transfusion; transfusion trigger; cross-matching; complications of blood transfusion; management of coagulopathy", pp. 20–23.
- BL 28e, ch. 19 "Trauma in children", § "Trauma in children: transfusion targets; trauma-induced coagulopathy (citrate)", p. 274.
- BL 28e, ch. 24 "Postoperative care including perioperative optimisation", § "Postoperative haemorrhage: risks of transfusion", p. 323.
- BL 28e, ch. 27 "Early assessment and management of severe trauma", § "Permissive hypotension, massive transfusion protocols and tranexamic acid", pp. 355–356.
- SAB 22e, ch. 19 "Principles of Preoperative and Operative Surgery", § "Hematologic system: anemia and transfusion risk", pp. 15–16; Table 19.12, p. 16.
- SAB 22e, ch. 33 "Shock, Electrolytes, and Fluid", § "Hypothermia; coagulopathy; TEG; tranexamic acid", pp. 232–234.
- SAB 22e, ch. 33 "Shock, Electrolytes, and Fluid", § "Storage lesion of PRBCs; leukoreduction", p. 242.
- SAB 22e, ch. 33 "Shock, Electrolytes, and Fluid", § "Crystalloid choice with blood transfusion", p. 248.
- SAB 22e, ch. 33 "Shock, Electrolytes, and Fluid", § "Hypocalcemia: citrate in transfused blood", p. 263.
- SAB 22e, ch. 36 "Management of Acute Trauma", § "Prehospital transfusion; massive transfusion definition", p. 310.
- SAB 22e, ch. 46 "Surgical Critical Care", § "Hemorrhagic shock: MTP versus viscoelastic-guided resuscitation", p. 535.
- SAB 22e, ch. 46 "Surgical Critical Care", § "Transfusion strategies; viscoelastic testing", pp. 545–546; Fig. 46.1, p. 546.
- SAB 22e, ch. 100 "Hemostasis and Thrombosis", § "Plasma, PCC, cryoprecipitate: adverse reactions; TEG-guided management; trauma-induced coagulopathy (PROPPR)", pp. 1588–1590.

Asked in: Paper I · Jun 2022 · Q6 (10 marks) ; Paper I · Dec 2025 · Q3(b) (5 marks) ; Paper IV · Apr 2023 · Q4(a) (5 marks)

“Discuss different methods and materials available to control intra-operative bleeding.”

“Methods of hemostasis in surgical practice.”

“Advances in topical hemostatic therapy in surgical practice.”

Types of surgical haemorrhage

- Bleeding at operation may be **arterial, venous or capillary**.
- **Primary** — during the operation.
- **Reactionary** — 24–48 h after operation: a **slipped ligature** or a missed vessel injury that stopped temporarily from vasoconstriction and hypotension; bleeds once blood pressure recovers.
- **Secondary** — days to weeks later: usually **deep-seated infection eroding a vessel**.

General principles

- No device replaces **preoperative preparation**, careful management of **antiplatelets and anticoagulants**, and **meticulous surgical technique** with knowledge of anatomy.
- Surgical bleeding obscures the field, prolongs operating time, increases haemodynamic stress, induces **coagulopathy**; perioperative transfusion is linked to recurrence and poorer survival in some cancers.
- Avoid damage to adjacent nerves and organs, unintended vascular thrombosis and adjacent tissue injury; **plunging clamps and suturing blindly in a pool of blood** do more harm than good.
- The method chosen depends on the **site and extent of bleeding** and the pathology.
- Capillaries and small veins: **monopolar diathermy**; vessels **>1 mm** (all named vessels): ties, clips, staples, bipolar or ultrasonic devices; large vessels: **suture ligation** to prevent dislodgement.

Classification of haemostatic methods

Table 1.4a.1. Common intra-operative haemostatic techniques (BL Summary box 7.8)

Group	Methods
Mechanical	Digital pressure; ligatures; haemostatic clamps and ligating clips; vascular stapling devices; wound packing; bone wax; image-guided embolisation
Thermal	Electrosurgery (monopolar, bipolar); cryosurgery; argon beam coagulation; vessel-sealing devices
Chemical / topical	Physical: absorbable collagen, gelatin, oxidised cellulose. Biological: topical thrombin, fibrin sealant, tranexamic acid
Intravenous (systemic)	Plasma; prothrombin complex concentrate; desmopressin (DDAVP); tranexamic acid

Source: BL 28e Summary box 7.8, p. 110; SAB 22e Fig. 100.5, p. 1591

Mechanical methods

- **Pressure** — digital or bimanual compression; first manoeuvre for any bleeding.
- **Ligatures and suture ligation** — permanent suture traditionally; absorbable sutures carry no increased bleeding or reoperation risk. **Endoloops** give burst pressures close to a hand-tie.
- **Clips and staplers** — vascular staple loads tolerate lower but still supraphysiological burst pressures.
- **Packing** — tamponade; in **damage control** temporary abdominal packing is followed by ICU resuscitation and delayed definitive haemostasis.
- **Inflow occlusion** — **Pringle manoeuvre** (digital or soft clamp on the portal triad) reduces hepatic arterial and portal inflow but not back-bleeding from the IVC and hepatic veins.
- **Plug** — holes from penetrating liver injury plugged with silicone tubing or a Sengstaken–Blakemore tube.
- **Bone wax** — physical barrier for bone bleeding.
- **Image-guided (angio)embolisation** — for bleeding not controlled at operation.

Thermal (energy) methods

- **Electrosurgery** — high-frequency AC (500 000–3 000 000 Hz):
 - **Coagulation mode**: interrupted, higher-voltage current (on 6%, off 94%) → dehydration and protein denaturation; stops bleeding by distorting the vessel wall, coagulating plasma proteins and stimulating the clotting cascade.
 - **Monopolar**: active pencil + return plate. **Bipolar**: current only between forceps limbs — near the recurrent laryngeal nerve or in patients with pacemakers.
- **Advanced bipolar vessel sealing** (e.g. LigaSure) — pressure + energy fuses collagen and elastin; seals vessels **up to 7 mm**, withstands **3 × normal systolic pressure**; impedance feedback stops energy when the seal is complete (2–4 s).
- **Harmonic (ultrasonic) scalpel** — blade vibrates at **20 000–50 000 Hz**; seals by protein denaturation from vibration, not heat; less smoke.
- **Combination devices** — ultrasonic + bipolar in one instrument; seal vessels up to 7 mm faster, no smoke.
- **Argon beam coagulator** — monopolar current carried along an argon jet that also blows blood away; shallow penetration; for **raw solid-organ surfaces** (liver, spleen, kidney, lung); ineffective for large vessels; risk of **argon gas embolism**.
- **Saline-cooled radiofrequency dissectors** — irrigation keeps surface temperature <105 °C, avoiding eschar and delayed bleeding during liver or kidney transection.

Topical haemostatic agents (materials)

- **Adjuncts** to mechanical and thermal methods, for **microvascular bleeding or diffuse ooze**.
- **Passive (physical)** agents give a scaffold for fibrin deposition and absorb up to **40 times their weight** in blood (tamponade); they need near-normal clotting factors and platelets.
- **Active (biological)** agents supply thrombin or fibrin and work even when the patient's own clotting is abnormal.

Table 1.4a.2. Topical haemostatic agents: mechanism and limitations

Agent (example)	Mechanism	Points of note
Gelatin (Gelfoam, Surgifoam) — porcine skin	Activated on contact with blood; matrix for platelet aggregation and clot	Often combined with topical thrombin
Collagen / microfibrillar collagen (Avitene)	Increases platelet function, activates intrinsic pathway	Works in heparinised patients; less effective in thrombocytopenia ; resorbs in 4–6 weeks
Oxidised (regenerated) cellulose (Surgicel)	Platelet adhesion; low pH causes coagulative necrosis; swells → tamponade	Antimicrobial ; breaks down over several weeks
Bone wax	Physical barrier (tamponade)	Non-absorbable; foreign-body infection risk
Caustic (silver nitrate, zinc chloride)	Protein precipitation and vessel occlusion	Superficial wounds only
Topical thrombin (bovine, human, recombinant)	Fibrinogen → fibrin; activates V, VIII, XI	Bovine thrombin can cause antibody-mediated coagulopathy
Fibrin sealant (Tisseel, Artiss)	Fibrinogen + thrombin + calcium + factor XIII ± TXA/aprotinin; forms clot regardless of patient's pathway	Reabsorbed in ~2 weeks; not for high-volume arterial or venous bleeding (clot dislodges → embolism)

Source: SAB 22e pp. 1590–1591; BL 28e p. 113

Advances in topical haemostatic therapy

- **Flowable agents** (FloSeal, Surgiflo) — gelatin scaffold (passive) combined with **topical thrombin** (active): multifactorial action.
- **Microporous polysaccharide haemospheres (MPH)** — hydrophilic spheres bind platelets and clotting factors and form a matrix for the fibrin clot.
- **Ostene** — **absorbable** alternative to bone wax; does not impair bone healing or cause inflammation.
- **Synthetic sealants** — **cyanoacrylate** and **polyethylene glycol** polymers polymerise on contact into an adhesive seal.
- **BioGlue** (glutaraldehyde + bovine albumin) — forms an adherent scaffold sealing tissue or synthetic grafts; used on **larger vessels and anastomoses**.
- **Fibrin-impregnated collagen fleece** and fibrin glue — on the raw cut surface of the liver remnant.

- **Fibrin sealant** — better than manual compression for anastomotic bleeding from **PTFE vascular grafts**.

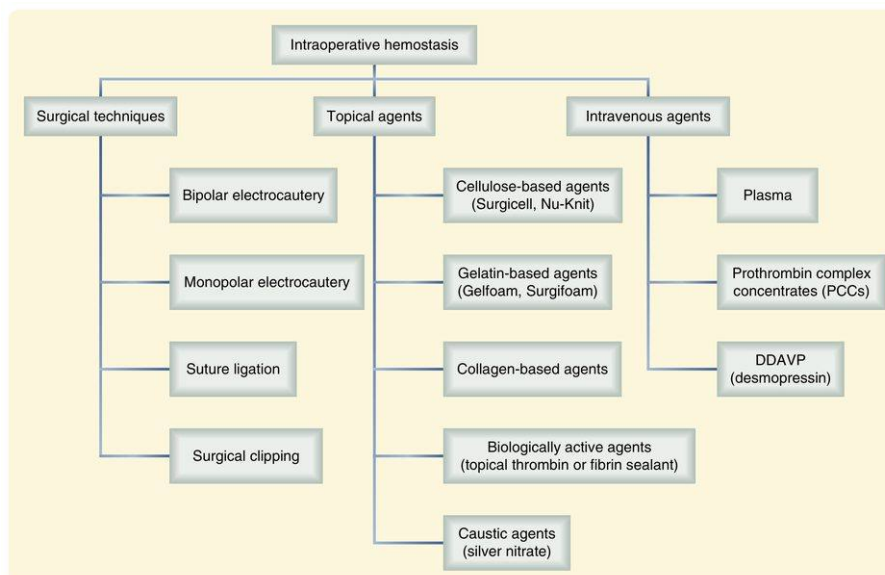


Figure 1.4a.1 — Options to consider for intraoperative hemostasis.

SAB 22e Fig. 100.5, p. 1591

Systemic (intravenous) measures and blood conservation

- **Tranexamic acid** — binds plasminogen, prevents fibrin breakdown; for hyperfibrinolysis (raised LY30 on TEG); risk of thromboembolism and seizures.
- **Desmopressin (DDAVP)** — releases endothelial vWF and raises factor VIII; for haemophilia, vWD, liver or renal disease; watch for **hyponatraemia**.
- **Prothrombin complex concentrate** — first line for emergency reversal of vitamin K antagonists.
- **Cryoprecipitate** — fibrinogen (first factor depleted in acute bleeding). **FFP** — first line for coagulopathic haemorrhage. **Platelets** — thrombocytopenia or platelet dysfunction.
- **Low central venous pressure anaesthesia** and **thromboelastography**-guided correction reduce loss in liver resection.
- **Cell saver** — intra-operative collection and return of washed red cells.

Haemostasis in minimal access surgery

- Blood obscures the view and **absorbs light**, so bleeding is harder to control endoscopically; a controlled conversion can quickly become uncontrolled.
- Ultrasonic and tissue-fusion devices, some combining 3–4 instrument functions, give a **clean smoke-free field**, fewer instrument exchanges and shorter operations.
- **Endoloops** and endoclips replace hand-ties; even small pools of blood or clot absorb light and impair the view — suction below a fluid level or in short bursts to keep the pneumoperitoneum.
- **CUSA** (ultrasonic aspirator) fragments high-water-content parenchyma (liver) but spares vessels and ducts, which are then clipped or sealed — it has no sealing ability itself.

Pearls

- ✓ Haemorrhage: primary (on table), reactionary (24–48 h, slipped ligature), secondary (days, infection).
- ✓ Vessels >1 mm need ties, clips, staples or sealing devices — not monopolar alone.
- ✓ LigaSure / combination devices seal vessels up to 7 mm.
- ✓ Passive topical agents need working clotting; fibrin sealant and thrombin do not.
- ✓ Fibrin sealant is not for brisk arterial bleeding — clot dislodges and embolises.
- ✓ Argon beam: raw organ surfaces; danger of gas embolism into large veins.

Sources

- BL 28e, ch. 7 “Basic surgical skills”, §“Basic haemostatic methods and principles of electrosurgery; advanced vessel-sealing devices; topical haemostatic agents”, pp. 110–113.
- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Transfusion: blood products, autologous blood”, p. 21.
- BL 28e, ch. 10 “Principles of minimal access surgery”, §“Minimal access surgery: control of bleeding and haemostasis”, p. 165.

- BL 28e, ch. 10 “Principles of minimal access surgery”, §“Minimal access surgery: evacuation of blood clots”, p. 171.
- BL 28e, ch. 29 “Torso and pelvic trauma”, §“Liver injury: the four Ps”, p. 380.
- BL 28e, ch. 29 “Torso and pelvic trauma”, §“Damage control surgery”, p. 386.
- BL 28e, ch. 69 “The liver”, §“Liver resection: blood loss and transfusion”, p. 1212.
- SAB 22e, ch. 19 “Principles of Preoperative and Operative Surgery”, §“Hemostasis; surgical adhesives; energy devices; argon beam coagulator”, pp. 31–34.
- SAB 22e, ch. 19 “Principles of Preoperative and Operative Surgery”, §“Damage control: temporary abdominal packing”, p. 36.
- SAB 22e, ch. 100 “Hemostasis and Thrombosis”, §“Treatment of coagulopathy; surgical management; topical and biologically active agents”, pp. 1589–1591; Fig. 100.5, p. 1591.

1.4b

Operating on a patient with haemophilia A — peri-operative measures

1×

Asked in: Paper III • May 2024 • Q8(b) (5 marks)

“Discuss the various measures to be taken while operating on a patient with haemophilia A.”

- **Haemophilia A:** X-linked recessive deficiency of **factor VIII**; isolated **prolonged aPTT**; severity depends on baseline factor level — from prolonged bleeding after minor injury to spontaneous bleeding into muscles, joints or the brain.

Pre-operative measures

- **Multidisciplinary planning** — haematologist, anaesthetist and surgeon; elective surgery only where laboratory and therapeutic resources are available after operation.
- **History** — haemarthrosis, deep haematomas, large palpable ecchymoses, bleeding after prior procedures or dental extraction, current antiplatelets/anticoagulants.
- **Choice of approach** — coagulation disorders are particularly dangerous in **minimal access surgery**, where options for haemostasis are more limited; patient must be fit for open conversion.
- **Gene transfer therapy** (viral vectors) has not yet produced sustained factor rises; long half-life factors reduce prophylactic infusion frequency.
- **Tests** — platelet count, PT/INR, aPTT, factor VIII level; mixing study.
- **Inhibitor screen one week before surgery** in patients with multiple prior factor treatments — up to **25% of severe haemophilia A** develop factor VIII inhibitors.
- **Factor correction by severity:**
 - **Mild (factor VIII >5%): desmopressin (DDAVP)** on the day of surgery raises factor VIII **up to fivefold within 90 min**; measure levels before and after; redose daily or twice daily; watch fluid retention and **hyponatraemia** (elderly).
 - **Severe: factor VIII concentrate** (plasma-derived or recombinant) **10–20 min before incision**; avoiding repeated dosing reduces inhibitor formation.
 - **Inhibitors present:** bypassing agents — **activated recombinant factor VII** or **activated prothrombin complex concentrate**.
- **Emicizumab** (bispecific antibody bridging activated IX and X) — unaffected by FVIII inhibitors; used for prophylaxis with or without inhibitors.
- **Cryoprecipitate**, PCC and plasma (cryoprecipitate is rich in factor VIII) — the traditional replacement products before targeted concentrates.

Intra-operative measures

- **Multiple venous access** so blood is sampled away from infusion sites.
- **Meticulous haemostasis:** ligatures, clips, bipolar or vessel-sealing devices; topical agents for ooze — **fibrin sealant and thrombin clot independent of the patient’s pathway**.
- **Tranexamic acid** to inhibit fibrinolysis.
- **Thromboelastography** — prolonged R-time reflects low factor levels.
- **Universal precautions** — haemophiliacs were exposed to infected blood products (HIV); main risk to staff is needle-stick injury.

Post-operative measures

- Continue **factor replacement / DDAVP** with serial factor VIII levels.
- **Analgesia without aspirin or NSAIDs.**

- Watch for **haematoma** — reopen and evacuate if it threatens skin, causes infection or compresses the airway in the neck.
- Monitor **sodium and fluid balance** while on DDAVP.

Table 1.4b.1. Haemophilia A: perioperative factor strategy

Situation	Agent	Key point
Mild (FVIII >5%)	Desmopressin	Up to 5-fold rise in 90 min; hyponatraemia
Severe	FVIII concentrate	10–20 min before incision; levels before and after
Inhibitors	rFVIIa or activated PCC	Bypass the intrinsic pathway; screen 1 week pre-op
Prophylaxis (± inhibitors)	Emicizumab	Not affected by FVIII inhibitors

Source: SAB 22e pp. 16–17, 1589–1590

Pearls

- ✓ Haemophilia A = factor VIII deficiency, X-linked, isolated long aPTT.
- ✓ Mild: DDAVP; severe: factor VIII concentrate 10–20 min before incision.
- ✓ Screen for inhibitors 1 week before surgery; inhibitors → rFVIIa or aPCC.
- ✓ No aspirin/NSAIDs; plan with haematology.
- ✓ DDAVP side effect: hyponatraemia.

Sources

- SAB 22e, ch. 19 “Principles of Preoperative and Operative Surgery”, §“Inherited coagulopathy: perioperative management of haemophilia”, pp. 16–17.
- SAB 22e, ch. 100 “Hemostasis and Thrombosis”, §“Haemophilia”, p. 1584.
- SAB 22e, ch. 100 “Hemostasis and Thrombosis”, §“TEG in haemophilia; DDAVP, TXA; haemophilias; surgical management; postoperative haematomas”, pp. 1588–1591.
- BL 28e, ch. 21 “Preoperative care including the high-risk surgical patient”, §“Preoperative care: bleeding disorders”, p. 292.
- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Cryoprecipitate”, p. 21.
- BL 28e, ch. 5 “Surgical infection”, §“Human immunodeficiency virus”, p. 59.
- BL 28e, ch. 10 “Principles of minimal access surgery”, §“Perioperative planning for minimal access surgery”, p. 168.

1.5a Fluid and electrolyte distribution in the body

1x

Asked in: Paper I • Dec 2020 • Q2(a) (4 marks)

“Fluid and electrolyte distribution in the body.”

Total body water

- Water forms **50–70% of body weight** (about **60%** in adults).
- Higher in **males**, in those with greater **lean body mass**, and at the **extremes of age**; lower with more body fat.
- Measured clinically by **bioimpedance spectroscopy**; intravascular volume by tracer dilution (potassium-40 or albumin).

Compartments

- **Intracellular fluid (ICF)** — the largest: **30–40% of body weight, two-thirds of total body water**.
- **Extracellular fluid (ECF)** — interstitial fluid + plasma:
 - **Intravascular (plasma): 5–7% of body weight, one-sixth of total body water**.
 - **Interstitial:** stores large volumes; recirculated by **lymphatics** back to the intravascular space.
- **Non-circulating water:** fixed water in **bone and dense connective tissue**; water secreted into **CSF**, intraocular, synovial, renal and GI fluids.
- Water **shifts rapidly** between compartments — large volumes can be drawn from the ICF into the circulation.

Electrolyte distribution

Table 1.5a.1. Principal ions of the body fluid compartments

Compartment	Principal cations	Principal anions	Notes
Intracellular	Potassium, magnesium	Phosphates, proteins	K ⁺ pumped in, Na ⁺ pumped out (Na ⁺ /K ⁺ -ATPase)
Interstitial	Sodium	Chloride, bicarbonate	Low protein; more inorganic anions than plasma
Plasma	Sodium	Chloride, bicarbonate, proteins (organic anions)	Higher total cations than interstitium (Gibbs-Donnan)

Source: SAB 22e p. 255

- **Normal plasma values** (mmol/L): sodium 135–145, chloride 95–105, potassium 3.5–5.3, bicarbonate 24–32, calcium 2.2–2.6, magnesium 0.8–1.2; osmolality 275–295 mOsmol/L.
- **Potassium** — the main intracellular ion; serum level is small, so **small serum changes reflect large intracellular changes**.
- **Calcium** — the most abundant electrolyte; **99% in bone**, 1% in blood as protein-bound, anion-bound and ionised (active) fractions.
- **Magnesium** — second most prevalent intracellular cation; **<1% of body stores in ECF**; plasma Mg is 30% protein-bound, 10% anion-bound, 60% ionised.
- **Gibbs-Donnan equilibrium**: the product of any diffusible cation-anion pair is equal on both sides of a semipermeable membrane.

Forces that maintain the distribution

- **Osmotic pressure**: water crosses cell membranes freely and follows osmotically active particles (about **290–310 mOsm**); the **number** of particles counts, not their size.
- **Sodium** is the major determinant of ECF osmotic pressure; **glucose and urea** also add to effective osmolality.
- **Colloid osmotic (oncotic) pressure** of plasma proteins holds water in the vascular space against the interstitium.
- Serum sodium concentration does **not** indicate ECF volume — a severe ECF deficit may coexist with low or high sodium.

Redistribution after surgery and injury

- **ADH and aldosterone** conserve sodium and water in the extracellular space; saline-rich resuscitation worsens it → peripheral and **visceral oedema**.
- **Capillary leak** in systemic inflammation moves fluid, proteins and electrolytes into the tissues; **intracellular volume falls** to refill the ECF.
- After severe sepsis or major trauma the **ECF expands by 6–10 L within 24 h**; patients can be **intravascularly depleted yet total-body overloaded** (endotheliopathy).

Pearls

- ✓ TBW ≈ 60% body weight: ICF two-thirds, plasma one-sixth.
- ✓ ICF: K⁺, Mg²⁺, phosphate, protein. ECF: Na⁺, Cl⁻, HCO₃⁻.
- ✓ Sodium governs ECF osmolality; plasma proteins govern oncotic pressure.
- ✓ 99% of calcium is in bone; >99% of magnesium is intracellular.
- ✓ Serum Na⁺ tells concentration, not volume.

Sources

- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Perioperative fluid management: body water”, pp. 255–256.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Electrolytes: potassium”, p. 260.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Electrolytes: calcium”, p. 262.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Electrolytes: magnesium”, p. 265.
- SAB 22e, ch. 34 “Metabolism and Nutrition in Surgical Patients”, §“Fluids and electrolytes”, p. 280.
- BL 28e, ch. 25 “Nutrition and fluid therapy”, §“Composition of plasma (Table 25.2)”, p. 333.
- BL 28e, ch. 1 “Metabolic response to injury”, §“Changes in body composition; volume loss; tissue oedema”, pp. 8–9.

Asked in: Paper IV · Jun 2022 · Q2(b) (5 marks)

“Concept of state of zero fluid balance.”

Definition

- **Zero (near-zero) fluid balance:** fluid is given so that **whole-body water balance stays near zero** — intake matches losses — with **no net weight gain** after elective surgery (**weight gain kept <2 kg**).
- A key element of **ERAS**: optimise intravascular volume, preserve cardiac output and tissue perfusion **without overload**; extra fluid is given only to replace **blood loss**.

Normal daily balance

Table 1.5b.1. Estimated daily fluid balance, healthy 70-kg adult (temperate climate)

Intake	Litres	Output	Litres
Beverages	1.2	Urine	1.5
Water in food	1.0	Insensible (skin, lungs)	0.9
Metabolic water (oxidation)	0.3	Faeces	0.1
Total	2.5	Total	2.5

Source: BL 28e Table 25.1, p. 331

- Maintenance requirement $\approx$ **2.2 L or 30 mL/kg per day**; sodium **0.9–1.2 mmol/kg/day**, potassium **1 mmol/kg/day**.
- Insensible losses rise with **fever, exertion, heat and tracheostomy**; faecal losses rise with diarrhoea, high-output stoma or fistula — these must be added.

Why aim for zero balance

- **Metabolic response to surgery:** ADH and aldosterone cause **post-operative oliguria** and retention of **sodium and water**; saline-rich fluids exaggerate it.
- **Harms of overload** (positive balance):
 - **Peripheral and visceral oedema** (gut wall) → delayed gastric emptying, **ileus**, delayed feeding, longer stay.
 - Release of **natriuretic peptides** and **glycocalyx damage**.
 - **Pulmonary oedema** and **intra-abdominal hypertension**.
- **Harms of under-filling** (negative balance):
 - **Hypoperfusion**, gut **mucosal acidosis**, **anastomotic leak**, impaired GI function.
- Restricting balanced crystalloids to avoid net weight gain **reduces complications and length of stay**.

How it is achieved

- **Pre-operative:**
 - Avoid prolonged fasting; **carbohydrate drink** before surgery.
 - Overnight IV fluids are unnecessary before elective major surgery.
- **Intra-operative:**
 - **Balanced crystalloid** (lactated Ringer rather than 0.9% saline) for maintenance; colloid boluses give no benefit over balanced crystalloids.
 - Treat anaesthetic (e.g. epidural) **hypotension with vasoactive drugs, not fluid boluses**.
 - Replace **blood loss** in addition.
 - **High-risk patients: goal-directed fluid therapy** — repeated boluses up to **250 mL** to optimise **stroke volume** on the Frank–Starling curve.
 - Avoid **very restrictive or liberal** regimens — aim for **euvolaemia**.
- **Post-operative:**
 - **Early oral intake** (solid diet from day 0); patients with minimal blood loss, litres of crystalloid already given and adequate urine output do not need IV maintenance.
 - Do not give reflex 1-L boluses for urine output <0.5 mL/kg/h — **oliguria is <400 mL/24 h (≈ 0.24 mL/kg/h)**; such boluses cause overhydration.

- Adjust maintenance to the individual, not a fixed rate.

Monitoring

- **Body weight before and after surgery** — best simple estimate of total water balance; daily weight thereafter.
- **Intake–output chart** including drains, fistulae, nasogastric tube and stool; pulse, blood pressure, CVP where available.
- **Serum electrolytes, haematocrit, BUN : creatinine ratio** (>20 prerenal); $\text{PaO}_2/\text{FiO}_2$ ratio reflects lung water.

Pearls

- ✓ Zero balance = intake equals output; no net weight gain (<2 kg).
- ✓ Daily turnover ≈ 2.5 L; maintenance ≈ 30 mL/kg/day.
- ✓ Overload $\rightarrow$ gut oedema, ileus, pulmonary oedema; under-filling $\rightarrow$ leak, mucosal acidosis.
- ✓ Vasopressors for anaesthetic hypotension; GDFT for high-risk cases.
- ✓ Weigh the patient — the simplest balance monitor.

Sources

- BL 28e, ch. 1 “Metabolic response to injury”, §“Metabolic response: volume loss; salt and water retention”, pp. 8–9.
- BL 28e, ch. 25 “Nutrition and fluid therapy”, §“Fluid and electrolyte replacement: daily fluid balance”, pp. 331–333; Table 25.1, p. 331.
- SAB 22e, ch. 22 “Enhanced Recovery After Surgery (ERAS): Evidence-Based P”, §“ERAS: intraoperative fluid therapy”, pp. 81–82.
- SAB 22e, ch. 22 “Enhanced Recovery After Surgery (ERAS): Evidence-Based P”, §“ERAS colorectal pathway: fluid therapy”, p. 84.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Perioperative fluid management: maintenance fluids”, pp. 255–257.

1.6 Body fluid changes (classification) and hyperkalaemia — aetiology, clinical features and management

2x

Asked in: Paper I · Oct 2024 · Q5 (2+(2+2+4 marks) ; Paper I · Jun 2022 · Q3(a) (5 marks)

“Classify the types of body fluid changes. Describe etiology, clinical manifestation and treatment of hyperkalemia.”

“Clinical features and management of hyperkalemia.”

NOT COVERED IN SOURCE

Classification of body fluid changes: searched BL 28e and SAB 22e full text for 'body fluid changes', 'fluid disorders / disturbances / abnormalities / imbalance', 'volume changes / deficit / excess', 'concentration changes', 'composition(al) changes', 'classification + fluid / electrolyte', 'third space'; BL index (pp. 1655–1669: fluid balance, fluid replacement, electrolytes, hyperkalaemia); chapters BL 1, 2 and 25, SAB 33 (Shock, electrolytes and fluid) and SAB 46 (Surgical critical care). Neither book gives a named classification of body fluid changes. The grouping the books use for these disorders — volume, sodium (osmolality), other electrolytes and acid–base — is given below as closely related material.

Types of body fluid changes (grouping used in the books)

Table 1.6.1. Body fluid changes grouped as the books describe them

Group	Disorders	Examples / thresholds
Volume (water) changes	Hypovolaemia (deficit); hypervolaemia (excess)	Deficit: haemorrhage, vomiting, diarrhoea, NG suction, fistulae, drains, burns, pancreatitis, obstruction, sepsis. Excess: iatrogenic over-infusion $\rightarrow$ pulmonary oedema, intra-abdominal hypertension
Sodium / osmolality (concentration) changes	Hyponatraemia; hypernatraemia	Hypo: mild 130–138, moderate 120–130, severe <120 mEq/L — classified by volume status (hypo-, eu-, hypervolaemic). Hyper: >145; severe >160 mEq/L
Other electrolyte (composition) changes	Potassium, calcium, magnesium, phosphate disorders	Hypokalaemia <3.5, hyperkalaemia >5.0 mmol/L; hypocalcaemia total <8.4 mg/dL
Acid–base changes	Metabolic / respiratory acidosis and alkalosis	Normal pH 7.35–7.45, PaCO_2 35–45 mmHg, HCO_3^- 22–26 mEq/L

Source: SAB 22e pp. 255–262, 544

- **Volume changes:** loss or gain of water and salt in the ECF; surgical patients are often **intravascularly depleted yet total-body overloaded**; it is safer to assume hypo- or hypervolaemia than normovolaemia.

- **Sodium (concentration) changes:** governed largely by **ADH**; hyponatraemia is assessed by volume status, as renal or extrarenal; hypernatraemia causes cellular dehydration.
- **Other electrolyte changes:** potassium (main intracellular ion), calcium (99% in bone), magnesium (mainly intracellular), phosphate.
- **Acid–base changes:** primary metabolic or respiratory disorder with compensation by the other system.
- Serum sodium reflects **concentration, not volume** — a severe ECF deficit can occur with low or high sodium, so volume and concentration are assessed separately.

Hyperkalaemia — definition

- **Serum potassium >5.0 mmol/L** (normal plasma 3.5–5.3 mmol/L).
- **>6 mmol/L:** resting membrane potential is perturbed and normal depolarisation and repolarisation are impaired.
- **6–7 mmol/L:** dangerous hyperkalaemia.
- Potassium is the main intracellular ion, kept inside cells by **Na⁺/K⁺-ATPase**, which needs ATP; when ATP fails (shock), the gradient is lost.
- **Balance:** intake 50–100 mmol/day; kidneys excrete 20–400 mmol/day; the **renin–angiotensin–aldosterone axis** is the key regulator of clearance, and hyperkalaemia itself stimulates aldosterone release.
- Small changes in serum potassium reflect **large intracellular changes**.

Aetiology

- **Reduced renal excretion** (commonest):
 - **Acute renal dysfunction or failure** — the **commonest cause in hospitalised patients**.
 - **Chronic renal failure** — at least **20% of normal renal function** is needed to respond to aldosterone and keep potassium normal; tubular defects add to it.
 - **Renal failure 2–14 days after major burns** (with metabolic acidosis, azotaemia).
 - **Delayed graft function** after renal transplantation.
- **Mineralocorticoid deficiency or resistance:**
 - **Adrenal insufficiency (Addison's)** — hyperkalaemia with hyponatraemia, hypotension and hypoglycaemia.
 - With normal renal function, **aldosterone, renin and cortisol** levels separate deficiency from resistance.
- **Drugs acting on the tubule** (usually contributory):
 - **Triamterene, spironolactone, β -blockers, ciclosporin, tacrolimus.**
- **Release of intracellular potassium** (cell injury overwhelms renal clearance):
 - **Shock** — ischaemic cell death and Na⁺/K⁺ pump failure release potassium, with acidosis.
 - **Sepsis; ischaemia–reperfusion** after revascularisation (severity depends on ischaemic time, 4–6 h); **rhabdomyolysis**.
 - **Tumour lysis syndrome** — with hypocalcaemia, hyperphosphataemia and hyperuricaemia.
- **Suxamethonium:**
 - In disuse atrophy, prolonged bed rest, denervation, **severe burns**, direct muscle trauma or rhabdomyolysis it causes **severe hyperkalaemia and cardiac arrest**.
- **Increased load:**
 - **Potassium infusions; massive transfusion;** blood transfusion.
- **Spurious (pseudo-) hyperkalaemia** — **haemolysed sample** releases potassium; repeat before drastic treatment.

Clinical manifestations

- **Cardiac arrhythmia** — the primary clinical problem; can be lethal (**cardiac arrest**).
- **ECG** (graded by severity):
 - **Peaked T waves** — tall, narrow and symmetric (Fig. 33.21A).
 - **T wave higher than R wave** — dangerous range (6–7 mmol/L).
 - **Loss of P wave and broad slurring of QRS** — arrest is pending.
- **Myocardial depression** with acidaemia in severe shock → poor cardiac output.
- In **CKD**, hyperkalaemia can cause **intra-operative death**; a normal potassium is a prerequisite for operation.
- Features of the **cause:** oliguria/renal failure, rhabdomyolysis or reperfusion injury, burns, tumour lysis, adrenal crisis.

Confirming the diagnosis and cause

- **Serum potassium** — repeat if haemolysis is suspected.
- **ECG** — decides urgency of treatment.
- **Urea, creatinine, urine output** — renal failure.
- **Arterial blood gas** — acidaemia (bicarbonate works best when acidaemia is present).
- **Aldosterone, renin, cortisol** — mineralocorticoid deficiency when renal function is normal.

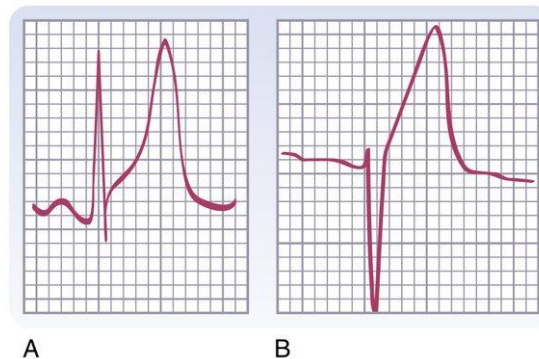


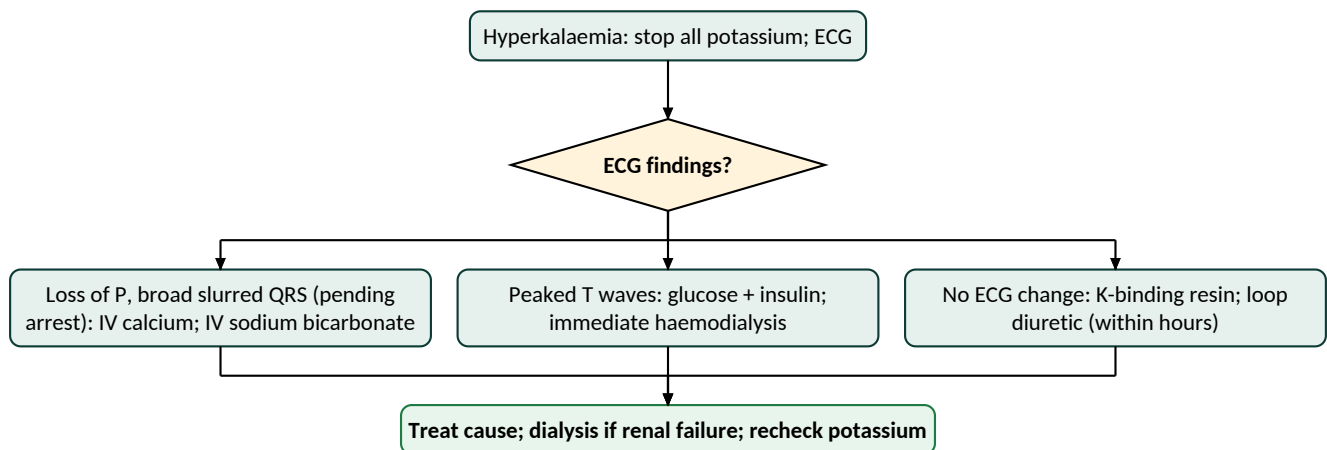
Figure 1.6.1 — Electrocardiographic changes. (A) Hyperkalaemia: the T wave is tall, narrow and symmetric. (B) Acute myocardial infarction: the T wave is tall but broad based and asymmetric.

SAB 22e Fig. 33.21, p. 261

Treatment

- **First: stop all potassium infusions.**
- Rapidly rising potassium is an **immediate threat** — every surgical unit should have an established protocol.
- Treatment urgency is set by the **ECG** (flowchart): pending arrest → immediate therapy; peaked T → prompt therapy; biochemical only → effective therapy within hours.
- **Renal replacement therapy** is indicated for AKI complicated by severe hyperkalaemia, acidosis or fluid overload.

Flowchart. Treatment of adult hyperkalaemia (SAB Box 33.5)



Agents — action and dose

- **Membrane stabilisation:**
 - **IV calcium** — **first therapy**; immediately reduces arrhythmia risk by antagonising the depolarising effect of K^+ .
 - Dose: **10 mL of 10% calcium chloride over 10 min** or **10 mL of 10% calcium gluconate over 3–5 min**.
- **Shift K^+ into cells:**
 - **Glucose + insulin:** **50 mL of 50% dextrose + 10 units regular insulin IV**; increases Na^+/K^+ -ATPase activity; **monitor glucose**.
 - **Sodium bicarbonate 50–100 mEq IV over 10–20 min:** protons leave cells and K^+ enters; benefit proportional to pre-existing **acidaemia**.
- **Remove K^+ from the body:**
 - **Haemodialysis** — most reliable in acute renal failure; continuous filtration clears K^+ more slowly.

- **Sodium polystyrene sulfonate** (cation-exchange resin) orally or rectally with 20% sorbitol; rectal route effective because colonic mucus carries much potassium — for chronic renal hyperkalaemia.
- **Loop diuretic** — promotes renal kaliuresis.
- **Fludrocortisone** (9 α -fludrocortisone) when aldosterone deficiency is the cause.
- **Prevention in surgical practice:**
 - CKD: **dialysis the day before or on the day of surgery**; treat $K^+ > 6$ mmol/L before elective surgery.
 - Give **bicarbonate before reperfusion** of an ischaemic limb; correct acidosis and K^+ released after revascularisation.
 - Avoid **suxamethonium** in burns, denervation, disuse atrophy and muscle trauma.
 - Early post-transplant hyperkalaemia: glucose + insulin first; early dialysis often needed.

Current guideline

- **Severity:** mild K^+ 5.5–5.9, moderate 6.0–6.4, **severe ≥ 6.5 mmol/L**; urgent 12-lead ECG for $K^+ \geq 6.0$; continuous monitoring for ≥ 6.5 or ECG changes.
- **Protect the heart** (ECG changes): IV calcium 6.8 mmol — **30 mL 10% calcium gluconate over 10 min** or 10 mL 10% calcium chloride over 5 min (chloride preferred in peri-arrest).
- **Shift: 10 units soluble insulin in 25 g glucose IV** (severe 1B; moderate 2C); if pre-treatment glucose < 7.0 mmol/L follow with 10% glucose 50 mL/h for 5 h; glucose checks over 6 h.
- **Nebulised salbutamol 10–20 mg** as an adjunct only — never alone for severe hyperkalaemia (1A); IV bicarbonate not routine (2C).
- **Remove:** sodium zirconium cyclosilicate for severe (1B) and to be considered for moderate (1B); patiromer an option (1C); **calcium resonium no longer routine** (2B); urgent dialysis for patients on haemodialysis (1A).
- **Recheck K^+** at 1, 2, 4, 6 and 24 h (within 2 h after treating severe hyperkalaemia).

Recommendation grade: as given per recommendation in brackets (GRADE 1A–2C)

UK Kidney Association — Clinical Practice Guideline: Management (Treatment) of Acute Hyperkalaemia in Adults (2020, updated 2023 and July 2026). The textbook predates this.

Pearls

- ✓ Hyperkalaemia = $K^+ > 5.0$ mmol/L; > 6 disturbs membrane potential; 6–7 dangerous.
- ✓ Commonest cause in hospital: acute renal failure.
- ✓ ECG sequence: peaked T $\rightarrow$ T taller than R $\rightarrow$ loss of P, broad QRS $\rightarrow$ arrest.
- ✓ Order of treatment: stop $K^+ \rightarrow$ IV calcium $\rightarrow$ insulin–glucose / bicarbonate $\rightarrow$ remove K^+ (dialysis, resin, diuretic).
- ✓ Always exclude a haemolysed sample.
- ✓ Suxamethonium in burns or denervation can cause fatal hyperkalaemia.

Sources

- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Body water; maintenance fluids; sodium; potassium: hypokalaemia, hyperkalaemia; calcium; tumour lysis syndrome”, pp. 255–263; Fig. 33.21 and Box 33.5, p. 261.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Magnesium; phosphate”, pp. 265–267.
- SAB 22e, ch. 19 “Principles of Preoperative and Operative Surgery”, §“Patients with renal disease: preoperative dialysis”, p. 13.
- SAB 22e, ch. 43 “Burns”, §“Burns: renal failure”, p. 498.
- SAB 22e, ch. 103 “Peripheral Occlusive Disease”, §“Acute limb ischaemia: reperfusion”, p. 1673.
- SAB 22e, ch. 46 “Surgical Critical Care”, §“Acid–base regulation; acute kidney injury and renal replacement therapy”, pp. 544–545.
- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Shock: cellular pathophysiology”, p. 11.
- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Shock: decompensation”, p. 13.
- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Complications of massive transfusion”, p. 22.
- BL 28e, ch. 24 “Postoperative care including perioperative optimisation”, §“Postoperative care: risks of blood transfusion”, p. 323.
- BL 28e, ch. 21 “Preoperative care including the high-risk surgical patient”, §“Preoperative care: renal failure”, p. 291.
- BL 28e, ch. 23 “Anaesthesia and pain relief”, §“Muscle relaxants: suxamethonium”, p. 307.
- BL 28e, ch. 25 “Nutrition and fluid therapy”, §“Composition of plasma”, p. 333.
- BL 28e, ch. 57 “The adrenal glands and other abdominal endocrine disorders”, §“Adrenal insufficiency”, p. 898.
- BL 28e, ch. 88 “Kidney transplantation and the principles of transplantation”, §“Renal transplant: early postoperative course”, p. 1605.

1.7a Types of metabolic acidosis

1x

Asked in: Paper I · Jun 2020 · Q3(a) (5 marks)

“Describe the types of metabolic acidosis.”

Definition

- **Metabolic acidosis:** a **primary reduction in plasma bicarbonate (HCO_3^-)** with fall in pH; pH and HCO_3^- move in the **same direction**.
- Three mechanisms:
 - **Addition of acid** buffered by $\text{HCO}_3^- \rightarrow$ an unmeasured organic anion is created.
 - **Loss of HCO_3^-** .
 - **Dilution** of HCO_3^- by non-bicarbonate fluid (e.g. large-volume 0.9% saline).
- **Normal values:** pH 7.35–7.45; PaCO_2 4.5–6 kPa (35–50 mmHg); standard bicarbonate 22–28 mmol/L; chloride 98–107 mmol/L.

Classification by anion gap

- **Anion gap (AG) = $[\text{Na}^+] - [\text{Cl}^-] - [\text{HCO}_3^-]$** ; normal **10–16 mmol/L**.
- Acid added $\rightarrow$ organic anion accumulates $\rightarrow$ **high (raised) AG acidosis**.
- HCO_3^- lost or diluted $\rightarrow$ **chloride rises** to keep electroneutrality $\rightarrow$ **normal-AG (hyperchloraemic) acidosis**.

Table 1.7a.1. Types of metabolic acidosis and their causes

Type	Mechanism	Causes (surgical examples)
High anion gap	Acid gain; unmeasured anion retained	MUDPILES: methanol, uraemia, diabetic ketoacidosis, paracetamol, isoniazid, lactic acidosis, ethanol/ethylene glycol, salicylates. Surgical: lactic acidosis of shock , mesenteric ischaemia, strangulated bowel; renal failure (e.g. after major burns); euglycaemic ketoacidosis with SGLT2 inhibitors peri-operatively
Normal anion gap (hyperchloraemic)	HCO_3^- loss or dilution; Cl^- rises	Diarrhoea and other HCO_3^- -rich GI losses; large-volume 0.9% saline ; renal tubular acidosis ; urinary diversion into bowel (ileal conduit, neobladder); mafenide acetate over large burns (carbonic anhydrase inhibition)

Source: SAB 22e p. 544; BL 28e pp. 11, 978, 1519

High anion gap acidosis — key points

- **Lactic acidosis** — underperfused cells switch to anaerobic metabolism; lactic acid accumulates and produces systemic acidosis; measured as **lactate and base deficit**.
- **Uraemic acidosis** — acute or chronic renal failure, e.g. renal failure 2–14 days after major burns with hyperkalaemia and azotaemia; in acute renal failure a **base deficit >6 mmol/L** predicts a poor outcome.
- **Toxins** — methanol, ethylene glycol, ethanol, salicylates, paracetamol, isoniazid.
- **Ketoacidosis** — SGLT2 inhibitors are stopped before surgery because of the risk of **euglycaemic diabetic ketoacidosis**.

Normal anion gap acidosis — key points

- **GI bicarbonate loss:** pancreatic juice (HCO_3^- 115 mmol/L), bile (35) and ileal fluid (30) are bicarbonate-rich.
- **Saline-induced:** massive 0.9% saline dilutes HCO_3^- ; it alters the base deficit and confuses interpretation but is a different state from lactic acidosis.
- **Mafenide acetate** (burn dressing) inhibits carbonic anhydrase — acidosis when applied over large surfaces, so it is kept for small full-thickness burns.
- **Urinary diversion:** bowel segments in the urinary tract cause hyperchloraemic acidosis that may decrease **bone density** — corrected with oral **sodium bicarbonate**.

Mixed disorders and compensation

- **Respiratory compensation:** hyperventilation lowers PaCO_2 ; expected $\Delta\text{PaCO}_2 = 1.2 \times \Delta\text{HCO}_3^-$; a different value means a mixed disorder.

- **Delta gap** = (normal AG – calculated AG) – (normal HCO_3^- – calculated HCO_3^-): **>+6 → coexisting metabolic alkalosis; <-6 → coexisting normal-AG acidosis.**
- Effects: mild acidemia is tolerated (aids O_2 unloading); worsening acidosis **depresses myocardial contractility**, blunts catecholamine response and is part of the **lethal triad** with hypothermia and coagulopathy.

Pearls

- ✓ Metabolic acidosis = primary fall in HCO_3^- .
- ✓ $\text{AG} = \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$; normal 10–16 mmol/L.
- ✓ High AG: MUDPILES — lactate is the commonest surgical cause.
- ✓ Normal AG: diarrhoea/fistula, saline, RTA, urinary diversion.
- ✓ Check compensation: $\Delta\text{PaCO}_2 = 1.2 \times \Delta\text{HCO}_3^-$.

Sources

- SAB 22e, ch. 46 “Surgical Critical Care”, §“Acid–base regulation: metabolic acidosis”, p. 544.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Lactate and base deficit; lethal triad”, pp. 230–231.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Normal saline and hyperchloraemic acidosis”, p. 248.
- SAB 22e, ch. 19 “Principles of Preoperative and Operative Surgery”, §“Preoperative management of SGLT2 inhibitors”, p. 20.
- SAB 22e, ch. 43 “Burns”, §“Burns: topical antimicrobials (mafenide)”, p. 493.
- SAB 22e, ch. 43 “Burns”, §“Burns: renal failure”, p. 498.
- SAB 22e, ch. 121 “Urologic Surgery”, §“Urinary diversion: metabolic complications”, p. 2081.
- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Shock: cellular pathophysiology”, p. 11.
- BL 28e, ch. 25 “Nutrition and fluid therapy”, §“Composition of gastrointestinal secretions (Table 25.3)”, p. 333.
- BL 28e, ch. 60 “The thorax”, §“Arterial blood gases: normal values (Table 60.2)”, p. 978.
- BL 28e, ch. 74 “The small intestine”, §“Acute mesenteric ischaemia: investigation”, p. 1313.
- BL 28e, ch. 83 “The urinary bladder”, §“Urinary diversion: complications”, p. 1519.

1.7b Lactic acidemia — definition and management

1×

Asked in: Paper I • Jun 2020 • Q3(b) (5 marks)

“Define lactic acidemia and discuss its management.”

Definition

- **Lactic acidemia**: blood lactate above normal — **normal lactate <2 mmol/L** (BL also quotes <2.5 mmol/L).
- Lactic acid is the product of **anaerobic metabolism**: pyruvate, the end product of glycolysis, is converted to lactate when oxygen is lacking.
- When enough tissue is underperfused, accumulated lactic acid produces a **systemic (high anion gap) metabolic acidosis — lactic acidosis.**
- Lactate is also an **active metabolite** (“lactate shuttle”) — used as fuel by heart, brain, liver and kidney; **adrenaline / catecholamine** release raises lactate without hypoxia. It signifies **tissue distress.**
- Blood level depends on **production versus clearance**; the **liver** metabolises most lactate, so liver disease raises it.

Causes

Table 1.7b.1. Causes of raised lactate in surgical patients

Group	Examples
Tissue hypoperfusion	All forms of shock ; occult hypoperfusion (normal vital signs, persistent lactic acidosis); mesenteric ischaemia; strangulated obstruction
Sepsis	Septic shock: lactate >2 mmol/L despite no hypovolaemia
Impaired clearance	Liver failure; liver graft dysfunction
Drugs and toxins	Metformin ; propofol infusion syndrome (>4 mg/kg/h for >48 h); cyanide in smoke inhalation (ATP depletion)
Metabolic	Refeeding syndrome; catecholamine excess

Source: BL 28e pp. 11, 19–20, 340, 1313, 1615; SAB 22e pp. 53, 229–230, 491, 531, 534

Assessment

- **Serial arterial lactate and base deficit** — sensitive for diagnosing shock and monitoring response; measure until normal.

- **Base deficit >6 mmol/L** → much higher morbidity and mortality; base deficit 8 → 25% mortality in older trauma patients.
- **Mixed venous O₂ saturation:** <50% = inadequate delivery (hypovolaemic, cardiogenic); >70% in sepsis.
- **Occult hypoperfusion for >12 h** carries **2–3 times the mortality**.

Management

- **1. Treat the cause — restore perfusion:**
 - **Volume first:** correct preload with IV fluids (and blood if bleeding) through short wide-bore cannulae before pressors or inotropes.
 - **Haemorrhage:** damage control resuscitation — haemostasis takes priority, since lactic acidosis cannot be corrected while bleeding continues.
 - **Sepsis:** Sepsis Six — fluids, IV antibiotics, oxygen and urine output; cultures, blood count, lactate; bundle within 6 h when lactate >4 mmol/L.
 - **Distributive shock:** vasopressors (noradrenaline); low output: **dobutamine** to mixed venous saturation >70%.
 - **Source control:** resect ischaemic or strangulated bowel; **damage control surgery** if lactate >5 mmol/L, pH <7.2, temperature <34 °C.
- **2. Resuscitate to perfusion end points:**
 - Aim for **normalisation of lactate, base deficit and pH** (with haemorrhage control).
 - Lactate **<2 mmol/L** → resuscitated, fit for definitive surgery; **>3 mmol/L** → under-resuscitated: further resuscitation or damage control.
 - Avoid aggressive crystalloid → oedema, ARDS, abdominal compartment syndrome.
- **3. Remove specific causes:**
 - Stop **metformin** before surgery; stop **propofol** (PRIS); **hydroxocobalamin** for cyanide.
- **4. Sodium bicarbonate — not routine:**
 - Treating the pH alone shows **no benefit**; rapid injection can **worsen intracellular acidosis** (CO₂ diffuses into cells).
 - Some give it in **dire coagulopathic bleeding**; the 50-mL ampoule (1 mEq/mL) acts as hypertonic sodium and raises BP.
- **5. Renal replacement therapy** for AKI with severe acidosis.

Pearls

- ✓ Normal lactate <2 mmol/L; >4 mmol/L triggers the sepsis bundle; >5 mmol/L favours damage control.
- ✓ Lactic acidosis = high anion gap acidosis of hypoperfusion.
- ✓ Treatment is the cause: restore perfusion, control bleeding and sepsis.
- ✓ Bicarbonate is not routine — it treats a number.
- ✓ Persistent lactate with normal vital signs = occult hypoperfusion.

Sources

- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Shock: cellular pathophysiology”, p. 11.
- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Clinical features of shock (Table 2.2)”, p. 14.
- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Damage control resuscitation”, p. 16.
- BL 28e, ch. 2 “Shock, haemorrhage and transfusion”, §“Fluid therapy; vasopressors; base deficit and lactate; end points of resuscitation”, pp. 18–20.
- BL 28e, ch. 5 “Surgical infection”, §“Sepsis bundle and Sepsis Six”, p. 59.
- BL 28e, ch. 25 “Nutrition and fluid therapy”, §“Refeeding syndrome”, p. 340.
- BL 28e, ch. 27 “Early assessment and management of severe trauma”, §“Venous lactate”, p. 358.
- BL 28e, ch. 29 “Torso and pelvic trauma”, §“Indications for damage control surgery”, p. 386.
- BL 28e, ch. 46 “Burns”, §“Burns: smoke and cyanide poisoning”, p. 667.
- BL 28e, ch. 69 “The liver”, §“Functions of the liver”, p. 1194.
- BL 28e, ch. 74 “The small intestine”, §“Acute mesenteric ischaemia”, p. 1313.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“End points of resuscitation”, p. 227.
- SAB 22e, ch. 33 “Shock, Electrolytes, and Fluid”, §“Lactate and base deficit; lethal triad; bicarbonate”, pp. 229–231.
- SAB 22e, ch. 20 “Anesthesiology Principles, Pain Management, and Sedation”, §“Perioperative diabetes: metformin”, p. 53.
- SAB 22e, ch. 43 “Burns”, §“Inhalation injury: cyanide”, p. 491.
- SAB 22e, ch. 46 “Surgical Critical Care”, §“Propofol-related infusion syndrome”, p. 531.
- SAB 22e, ch. 46 “Surgical Critical Care”, §“Sepsis and septic shock”, p. 534.
- SAB 22e, ch. 46 “Surgical Critical Care”, §“Metabolic acidosis; renal replacement therapy”, pp. 544–545.
- SAB 22e, ch. 91 “Small Intestine”, §“Strangulated obstruction”, p. 1335.

How to use these notes

- **One answer, many questions:** repeats and near-repeats are merged into one answer; its "Asked in" line lists every paper and year, and the badge (1×, 2×, 3×, 4×+) shows how often it was asked.
- **Red box — NOT COVERED IN SOURCE:** the point is not in the three textbooks; the box names the searches made and the answer gives the closest related book material.
- **Amber box — Current guideline:** the only material not taken from the textbooks; it names the body, document and year, and states that the textbook predates it.
- **Figures** are real figures cropped from the books, with the book figure number and printed page; **schematics** are simplified exam diagrams drawn for these notes and are labelled as such.
- **Sources** at the end of each answer give book, chapter, section and printed pages.

A • Parts in this volume

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22	Arterial Disease	19	25	321
23	Venous & Lymphatic Disease	13	23	365
24	Thorax, Heart & Diaphragm	13	15	392

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Plastic & Reconstructive Surgery

14.1a

Split-thickness skin graft — ways to resurface a wound, principles, harvesting and causes of graft failure

2×

Asked in: Paper IV · Dec 2025 · Q1 (3+4+3 marks) ; Paper I · Dec 2022 · Q10(a) (5 marks)

“What are the various ways to resurface a wound? Discuss how will you raise split thickness skin graft.

What are the causes of failure of such a graft?”

“Principles of split thickness skin graft.”

1. Ways to resurface a wound

- Choice is **patient-centred**: the obsolete **reconstructive ladder** (simplest method first) has given way to the **reconstructive elevator** — pick the technique that gives the best long-term result for that patient, skills and resources.
- Guiding principle: replace **“like with like”** after analysing what is missing (skin, fat, muscle, nerve); simply “closing the hole” is not enough.
- Skin closure must always be **without tension**.
- **Matching the method to the wound**:
 - **acute major burns** → STSG, almost always, to restore skin early and preserve life;
 - **face after tumour excision** → local flap, superior to a graft in contour, skin quality and colour match;
 - **pressure sore** → local skin-and-muscle flap to obliterate dead space and move the scar away from the pressure point;
 - **open lower-limb fracture** → free tissue transfer, since local tissue is lacking — healthy vascularised cover over fracture and metalwork.
- **Skin substitutes**: readily available in quantity with **no donor-site defect**, but expensive and technique-sensitive.
- **Tissue expansion**: skin elongates by **creep and stress relaxation**; angiogenesis increases flap vascularity — gives skin of similar quality to that being replaced.

Table 14.1a.1. Options for wound closure and resurfacing

Option	Principle	Typical use
Primary closure	Direct, tension-free apposition of edges	Wounds that close without tension
Secondary intention	Healing from the base: contraction and epithelialisation	Wounds with poor healing potential (leg and pressure ulcers)
Tertiary (delayed primary) closure	Closure once the open wound is clean and granulating	Contaminated wounds, fasciotomy wounds
Negative-pressure wound therapy	Draws edges together, removes exudate, promotes granulation	Adjunct before definitive closure; securing grafts
Split-thickness skin graft	Epidermis + part of dermis, revascularised from the bed	Large defects, burns, flap donor sites
Full-thickness skin graft	Epidermis + whole dermis	Small facial defects, syndactyly, contracture release
Composite graft	Skin + cartilage or fat	Nasal ala (helical root graft), eyebrow
Dermal matrices / skin substitutes	Engineered dermal ± epidermal replacement	Burns, chronic wounds; thin graft on top
Allograft / xenograft	Cadaver or porcine skin as temporising cover	Extensive burns, necrotising fasciitis
Local, regional or pedicled flap	Tissue with its own blood supply	Face, pressure sores, poor beds
Tissue expansion	Subcutaneous silicone balloon inflated weekly	Scalp, burn alopecia, like-for-like skin
Free flap	Microvascular anastomosis to recipient vessels	Open lower-limb fractures, head and neck

Source: BL 28e Summary box 3.4, pp. 31–32; pp. 62, 684–688

2. Principles of split-thickness skin grafting

- **Definition:** a graft of **epidermis and a variable amount of dermis** (Thiersch graft), transferred **without its blood supply**.
- **Survival depends on the recipient bed:** it must be healthy and well vascularised so that **angiogenesis into the graft** can occur.
- The graft supplies extracellular matrix, keratinocytes, fibroblasts and growth factors and excludes bacteria (“dynamic reciprocity”).
- **Thickness** (thin, deeper, thick STSG):
 - in burns usually **0.30–0.38 mm**;
 - **the thinner the graft, the greater the contraction** and functional disability, but the faster the donor site heals.
- **Donor site heals by secondary intention** from the remaining dermis and adnexa within about **2 weeks**; it can be re-harvested, but each harvest thins it — the donor site is not an infinite resource.
- **Indications:**
 - defects too large for primary closure where secondary healing is inappropriate or too slow;
 - **burns** (deep dermal and full-thickness after excision) — life-saving early cover;
 - free-flap donor sites (radial forearm, fibula); lining of defects under an obturator or orbital prosthesis;
 - patients unfit for a long flap operation; delayed closure over a granulating or matrix-prepared bed.
- **Unsuitable beds** (poor vascularity):
 - exposed bone, cartilage or tendon; irradiated tissue — and grafts tolerate post-operative radiotherapy poorly.
- **Limitations:** contraction, poor colour and thickness match, mesh-pattern scarring, fragile under shear — never “normal” skin.
- **Donor-site morbidity:** pain (reduced by meshing to limit donor area), temporary hyperpigmentation; repeated harvesting can leave a deep wound that itself needs grafting.
- **Massive burns** (donor skin short): very thin grafts for quick re-harvest, wide meshing, **Meek** micrografts (3 × 3 mm squares), and widely meshed autograft under allograft cover.

3. How to raise (harvest) a split-thickness skin graft

- **1. Prepare the recipient bed:** excise all necrotic tissue down to **punctate bleeding**; meticulous haemostasis.
- **2. Choose the donor site:** usually the **thigh** (lateral thigh); large areas can be harvested.

- **3. Infiltrate** the donor site (and burn eschar) with **tumescent dilute adrenaline 1:500 000–1:1 000 000** to reduce bleeding.
- **4. Tension the skin:** the donor skin is stretched flat (hands and a board) ahead of the dermatome — the correct method of providing skin tension.
- **5. Cut the graft** with a **power dermatome or graft knife** set to the required depth — gives a **consistent thickness**.
- **6. Dress the donor site at once:** sterile non-adherent dressing — **alginate** (haemostatic calcium salts) suits the exudating donor site; heals in about 2 weeks.
- **7. Prepare the graft:**
 - leave as an **intact sheet** where function or appearance matter (face, neck, hands, over joints, breasts);
 - or **mesh** it through bladed rollers — expands cover and lets blood and serum escape (less haematoma/seroma); ratio $\leq 2:1$ for cosmesis, 1:3 up to 1:9 in extensive burns (worse mesh scarring).
- **8. Apply and fix:** lay on the bed in close contact; fix with **sutures, staples or glue** (fibrin sealant aids adherence); apply in the position of maximal stretch.
- **9. Secure against shear:** **tie-over bolster** or **NPWT dressing**; “rolling” sheet grafts to express fluid.
- **10. Aftercare:** immobilise and **splint** (hand in the position of function, knee in extension, axilla in abduction), elevate the limb; supervised movement after about **5 days**; monitor fluid leaks and change outer dressings.

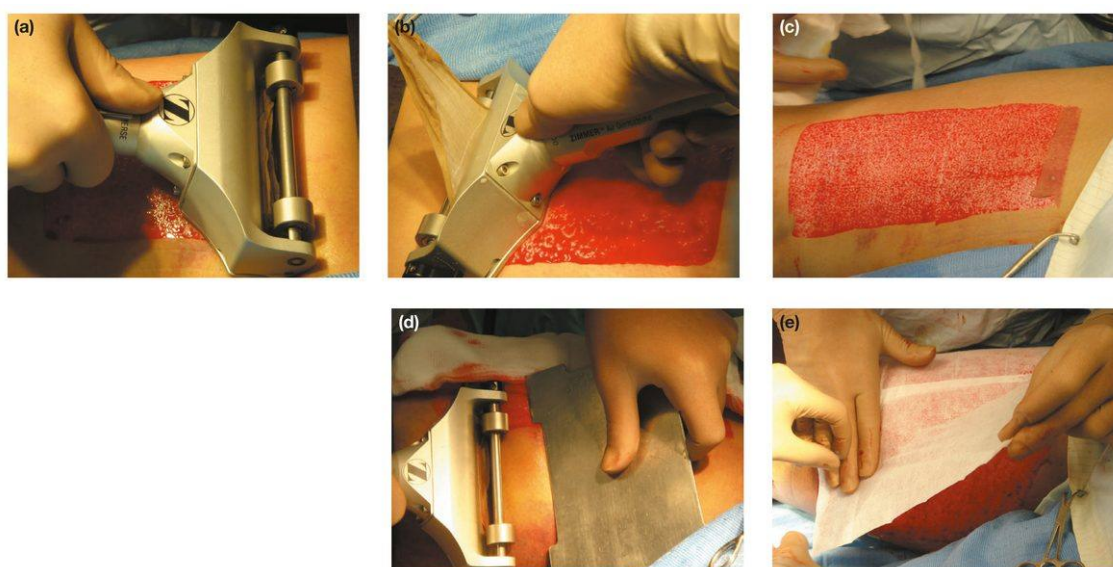


Figure 14.1a.1 — Power dermatome harvest of a split-thickness skin graft, with the correct method of providing skin tension (a–d) and applying a sterile dressing (e).

BL 28e Fig. 47.8, p. 687

4. Causes of failure of a split-thickness skin graft

Table 14.1a.2. Causes of skin-graft failure and their prevention

Cause	Mechanism	Prevention
Inadequate bed preparation (commonest in burns)	Residual necrotic tissue — no vascular bed for ingrowth	Excise to punctate bleeding / healthy tissue
Shearing forces	Movement disrupts the graft from the bed before vessels link up	Bolster or tie-over, NPWT, splinting, immobilisation
Haematoma or seroma	Fluid lifts the graft off the bed	Meticulous haemostasis, meshing, rolling, pressure dressing
Infection	Graft lysis — especially group A β-haemolytic Streptococcus	Adequate excision, topical antimicrobials at surgery
Avascular or irradiated bed	Exposed bone, cartilage, tendon; irradiated tissue cannot revascularise the graft	Choose a flap (vascularised tissue) instead
Patient factors	Smoking, diabetes, malnutrition impair healing	Optimise before elective surgery

Source: BL 28e p. 686; SAB 22e pp. 495–496, 809, 867–869

Pearls

- ✓ STSG = epidermis + part of dermis; FTSG (Wolfe) = epidermis + whole dermis.
- ✓ Graft survives by revascularisation from the bed — the bed decides take.
- ✓ Thinner graft → more contraction but quicker donor healing.
- ✓ Commonest causes of loss: poor bed, shear, haematoma/seroma, infection (group A Streptococcus).
- ✓ Sheet grafts on face, hands, neck and joints; mesh elsewhere.
- ✓ Never graft bare bone, tendon, cartilage or irradiated tissue — use a flap.

Sources

- BL 28e, ch. 3 “Wound healing and tissue repair”, §“Wound management: reconstruction options for wound closure”, pp. 30–32; Summary box 3.4, p. 32.
- BL 28e, ch. 3 “Wound healing and tissue repair”, §“Scar contractures”, p. 36.
- BL 28e, ch. 5 “Surgical infection”, §“Surgical infection: delayed primary closure”, p. 62.
- BL 28e, ch. 46 “Burns”, §“Burns: surgical treatment; skin grafts and skin substitutes”, pp. 674–676.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Wound healing; wound dressings; reconstructive techniques; grafts”, pp. 683–688; Fig. 47.8, p. 687.
- SAB 22e, ch. 23 “Wound Healing”, §“Wound healing: split-thickness skin grafting”, p. 105.
- SAB 22e, ch. 43 “Burns”, §“Burns: excision and grafting”, pp. 495–496.
- SAB 22e, ch. 67 “Head and Neck Cancer”, §“Head and neck reconstruction: skin graft”, p. 809.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Goals of reconstruction; preoperative considerations; radiation”, pp. 866–869.

14.1b

Split-thickness (partial-thickness) versus full-thickness skin grafts

1×

Asked in: Paper II · May 2024 · Q10(b) (5 marks)

“Differentiate between partial thickness and full thickness skin grafts.”

Definitions

- **Split-thickness (partial-thickness) skin graft (STSG, Thiersch):** epidermis + a variable amount of dermis; graded thin, deeper and thick.
- **Full-thickness skin graft (FTSG, Wolfe):** epidermis + the entire dermis.
- Both are transferred **without a blood supply** and depend on a well-vascularised bed; both carry a complete epidermis — the difference lies in the **dermis**.

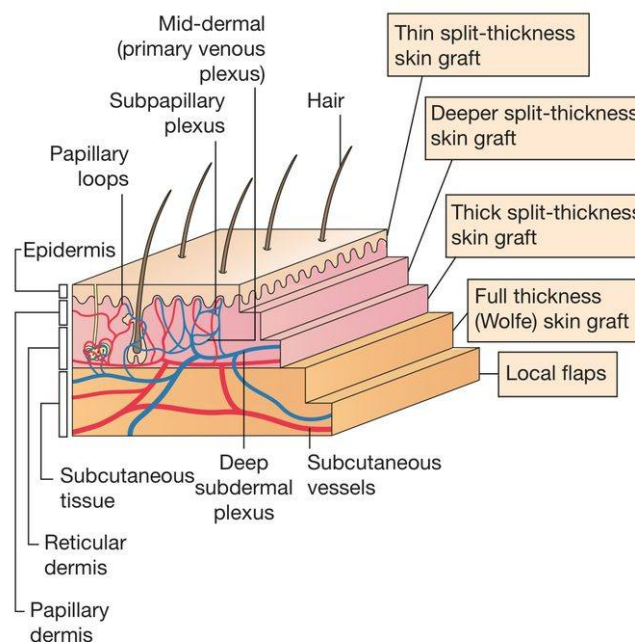


Figure 14.1b.1 — Schematic anatomy of the skin and its relationship to harvesting skin grafts of varying thickness and raising local flaps.

BL 28e Fig. 47.7, p. 686

Differences

Table 14.1b.1. Split-thickness versus full-thickness skin graft

Feature	Split-thickness graft	Full-thickness graft
Composition	Epidermis + part of dermis	Epidermis + whole dermis
Thickness	Variable; burns 0.30–0.38 mm	Full dermal thickness
Harvest	Dermatome or graft knife at a set depth	Excised; area limited by primary closure of the donor site
Donor sites	Thigh (commonest); large areas available	Supraclavicular, groin crease, post-auricular (Wolfe)
Donor-site healing	Secondary intention from residual dermis in ~2 weeks; can be re-harvested	Closed primarily; cannot be used more than once
Size of defect	Large defects, e.g. major burns	Small defects in cosmetically important areas
Contraction	Marked secondary contraction (more with thinner grafts)	Retains elasticity — least secondary contraction
Appearance	Poor colour/thickness match; mesh pattern; never “normal” skin	Better texture; closer to native skin; best scar outcome
Typical uses	Burns, flap donor sites, granulating wounds, obturator lining, unfit patients	Facial defects (nose, temple) after skin-cancer excision, syndactyly release, burn contracture release

Source: BL 28e pp. 31, 36, 675, 686–687; SAB 22e pp. 496, 809

Key points

- **Dermis decides behaviour:** the near-complete dermal layer explains the superior function and appearance of FTSG.
- **Contraction:** the thinner the graft, the greater the contraction and functional disability — so for **contracture release** an FTSG (or a flap) is preferred over an STSG.
- **Supply versus quality:** STSG gives quantity (large areas, re-harvestable donor site) at the cost of quality; FTSG gives quality but only in small amounts.
- Thicker STSG does not always scar better and slows donor healing — standard-thickness STSG is used for acute burn cover.
- **Choosing between them:**
 - large area or poor general condition, speed of cover paramount → STSG;
 - small defect where appearance and low contraction matter (face, syndactyly, burn contracture release) → FTSG;
 - neither when the bed is avascular — then a flap is needed.
- **Composite grafts** add another tissue (cartilage, fat): e.g. helical-root skin–cartilage graft for the nasal ala.

Pearls

- ✓ STSG = Thiersch; FTSG = Wolfe.
- ✓ FTSG contracts least; thin STSG contracts most.
- ✓ STSG donor heals by itself in ~2 weeks; FTSG donor must be sutured.
- ✓ Face → FTSG (post-auricular, supraclavicular); burns → STSG (thigh).
- ✓ Neither takes on bare bone, tendon, cartilage or irradiated tissue.

Sources

- BL 28e, ch. 3 “Wound healing and tissue repair”, §“Wound management: skin grafts and flaps”, p. 31.
- BL 28e, ch. 3 “Wound healing and tissue repair”, §“Scar contractures”, p. 36.
- BL 28e, ch. 46 “Burns”, §“Burns: skin grafts”, p. 675.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Grafts: split-thickness, full-thickness and composite skin grafts”, pp. 686–688; Fig. 47.7, p. 686.
- SAB 22e, ch. 43 “Burns”, §“Burns: full-thickness grafts and scarring”, p. 496.
- SAB 22e, ch. 67 “Head and Neck Cancer”, §“Head and neck reconstruction: split- and full-thickness skin grafts”, p. 809.

Asked in: Paper IV · Dec 2020 · Q5(a) (5 marks)

“Musculocutaneous flaps.”

Definition and vascular basis

- **Flap:** block of tissue with its own **innate blood supply** transferred from a donor site to a defect; the **pedicle** is the base that carries the blood supply.
- **Musculocutaneous (myocutaneous) flap:** a muscle carried with its overlying skin — one of the *composition* classes of flaps (cutaneous, fasciocutaneous, musculocutaneous, muscle, osseocutaneous...).
- The **skin paddle is supplied by musculocutaneous perforators** that pass from the muscle through the deep fascia to the subdermal plexus (Taylor’s **angiosome** concept: every tissue block is fed by a source vessel via perforators).
- The **motor nerve always runs with a vascular pedicle**, often the main source of the flap circulation.
- A **dominant pedicle** can sustain the whole muscle; a **minor pedicle** sustains only a portion.

Mathes and Nahai classification of muscle flaps

Table 14.2a.1. Mathes and Nahai (1981) classification of muscle (and musculocutaneous) flaps

Type	Vascular pattern	Examples
I	Single vascular pedicle	Tensor fascia lata, gastrocnemius
II	One dominant + one or more minor pedicles; cannot survive on the minor pedicle(s) alone	Gracilis, biceps femoris, sternocleidomastoid, soleus, trapezius
III	Two dominant pedicles	Gluteus maximus, pectoralis minor, rectus abdominis, serratus anterior, temporalis
IV	Segmental pedicles	Flexor hallucis longus, sartorius, tibialis anterior
V	One dominant pedicle + several smaller segmental pedicles; can survive on the minor pedicles alone	Latissimus dorsi, pectoralis major

Source: BL 28e p. 690; Fig. 47.24, p. 697

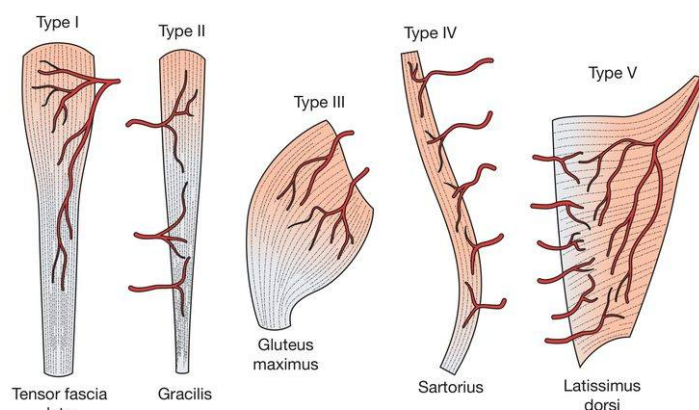


Figure 14.2a.1 — The Mathes and Nahai classification of muscle flaps.

BL 28e Fig. 47.24, p. 697

Common musculocutaneous flaps and their uses

- **Pectoralis major:** advancement flap on the **thoracoacromial artery**, turnover flap on the **internal mammary artery**, or myocutaneous flap; upper/lower sternal defects; the pedicled myocutaneous pectoralis flap patches pharyngeal defects and is the **gold standard for closing large pharyngocutaneous fistulas**.
- **Latissimus dorsi:** workhorse flap of large size and mobility on the **thoracodorsal vessels**; very large arc of rotation (neck, shoulder, anterior and posterior chest wall); can be folded posteromedially on its intercostal perforators; breast reconstruction (alone or with an implant) and, as a free flap, limb salvage.
- **Rectus abdominis — TRAM / VRAM:** TRAM for breast reconstruction; VRAM on the **deep inferior epigastric vessels** for the irradiated perineum and pelvis after colorectal resection (extended VRAM reaches 30–40 cm from its pedicle, up to 10 cm wide); can be turned upward on the superior epigastric vessels for anterior chest wall defects.

- **Anterolateral thigh:** septocutaneous or musculocutaneous on the **lateral circumflex femoral artery**, depending on the course of the perforators.
- **Gracilis:** pelvic dead space and perineum; free (neurotised) functional transfer for facial reanimation.

Advantages and indications

- Covers defects **without a vascularised bed** — exposed tendon, cortical bone, prosthesis or hardware — where a graft would fail.
- **Bulk** obliterates dead space (deep pelvis, large fistula) and seals tissue of tenuous quality.
- **Well-vascularised muscle** aids infection control and wound healing; brings fresh tissue into irradiated fields.
- Pedicled flaps need a shorter operation and suit patients unfit for free tissue transfer or in whom a free flap has failed.

Drawbacks

- **Donor-site morbidity:** TRAM/VRAM → hernia, bulge, abdominal wall laxity (mesh often used); the muscle-sparing DIEP flap has largely replaced the TRAM.
- Muscle **atrophy** after its motor nerve is divided; bulky flaps may need debulking 6–12 months later.
- Insensate muscle flaps are prone to **pressure ulceration**.
- Pedicled regional flaps are limited by reach and, in head and neck cancer, fail more often than free flaps.
- Previous surgery may sacrifice the pedicle (thoracotomy → thoracodorsal artery; internal mammary used for CABG → pectoralis turnover flap).

Monitoring

- Colour, warmth and turgor; **pressure on the skin paddle gives the capillary refill time**; pinprick for bleeding.
- Hand-held or **implantable Doppler** — the latter especially for buried muscle flaps with no skin paddle.

Pearls

- ✓ Skin of a musculocutaneous flap lives on perforators from the underlying muscle.
- ✓ Mathes–Nahai: I single, II dominant + minor, III two dominant, IV segmental, V dominant + segmental.
- ✓ Latissimus dorsi and pectoralis major = type V; rectus abdominis = type III; gracilis = type II.
- ✓ Pectoralis major flap: gold standard for large pharyngocutaneous fistula.
- ✓ TRAM donor site → hernia/bulge; DIEP spares the muscle.

Sources

- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Flaps: classification; muscle and musculocutaneous flaps”, p. 690.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Skin anatomy: angiosome concept”, p. 683.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Mathes and Nahai classification”, p. 697; Fig. 47.24, p. 697.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Microsurgery; flap monitoring”, pp. 699–700.
- BL 28e, ch. 58 “The breast”, §“Breast reconstruction”, pp. 938–939.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Pharyngocutaneous fistula”, p. 871.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Breast reconstruction; trunk and chest wall”, pp. 872–874.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Pelvis and perineal reconstruction: VRAM”, p. 879.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Foot: insensate and bulky flaps”, p. 886.
- SAB 22e, ch. 67 “Head and Neck Cancer”, §“Head and neck reconstruction: regional and free flaps”, pp. 809–810.
- SAB 22e, ch. 37 “The Difficult Abdominal Wall”, §“Mesh infection: vascularised muscle”, p. 367.

Asked in: Paper II · Apr 2023 · Q7(a) (5 marks)

“Classification of various types of skin flaps used in plastic surgery reconstructive procedures.”

Definition

- **Flap:** block of tissue with an **innate blood supply** transferred from a donor site to reconstruct a defect; the **pedicle** is its base carrying the blood supply.
- Unlike a graft it can cover a defect **without a vascularised bed** (exposed tendon, cortical bone, prosthesis).
- Flaps are classified by blood supply, proximity to the defect, method of transfer and tissue content — summarised as the **five Cs**.

The five Cs classification

Table 14.2b.1. Five Cs classification of flaps

C	Basis	Types
1. Circulation	Blood supply	Random pattern — no dominant vessel; axial pattern — dominant feeding vessel
2. Composition	Tissue carried	Cutaneous, fasciocutaneous, fascial, musculocutaneous, muscle, osseocutaneous, osseous, omentum/bowel
3. Contiguity	Proximity to defect	Local (shares a side with the defect), regional (near but not adjacent), distant (pedicled or free)
4. Contour	Method of transfer	Advancement, transposition, rotation, interpolation, waltzing, crane principle, free
5. Conditioning	Delay	Partial elevation and resetting before definitive transfer improves blood supply and allows a larger flap

Source: BL 28e p. 690

By circulation

- **Random pattern:** no named vessel; e.g. gluteal fold flap, Atasoy and Kutler V-Y fingertip flaps, cross-finger and thenar flaps (delayed random pattern).
- **Axial pattern:** designed on a named vessel; e.g. pedicled groin flap, forehead flap on the supratrochlear artery, Singapore flap, homodigital island flap on the digital artery.
- **Perforator flap:** skin and fat islanded on a single perforator with its venae comitantes (e.g. anterolateral thigh, DIEP); may be **propellered** 180° into an adjacent defect.
- **Venous flow-through flap:** vein provides both inflow and outflow; thin and pliable but prone to congestion.
- **Chimeric flap:** several independent flaps, each with its own pedicle, linked to one source vessel (e.g. subscapular system: scapular, parascapular, latissimus dorsi, serratus anterior).

By composition — subclassifications

- **Fasciocutaneous** (Cormack and Lamberty):
 - **A** multiple perforators (Pontén flap); **B** single perforator along the flap axis (scapular, parascapular).
 - **C** segmental perforators from one source vessel (radial forearm, lateral arm); **D** as C but raised osteomyofasciocutaneous (free fibula).
- **Muscle / musculocutaneous** (Mathes and Nahai): types I–V by pattern of vascular pedicles (e.g. tensor fascia lata I, gracilis II, gluteus maximus III, sartorius IV, latissimus dorsi V).

By contour (method of movement) — local flaps

- **Advancement:** simple rectangular ($\pm$ Burow's triangles excised at the base), **V-Y**, hatchet (back cut aids advancement), Moberg (bipedicle) flap of the thumb.
- **Transposition:** swung about a pivot point; donor defect grafted or closed primarily — **rhomboid** (parallelogram-shaped), **bilobed** (second smaller flap closes the donor site of a convex defect), **Z-plasty** (two interposed triangular flaps).
- **Rotation:** curved flap rotated into the defect, often with a back cut (scalp; pilonidal sinus).

- **Bipedicle:** “bucket-handle” flap supplied from both ends (lower eyelid).
- **Staged pedicle division:** forehead flap to the nose — pedicle divided at a second stage.

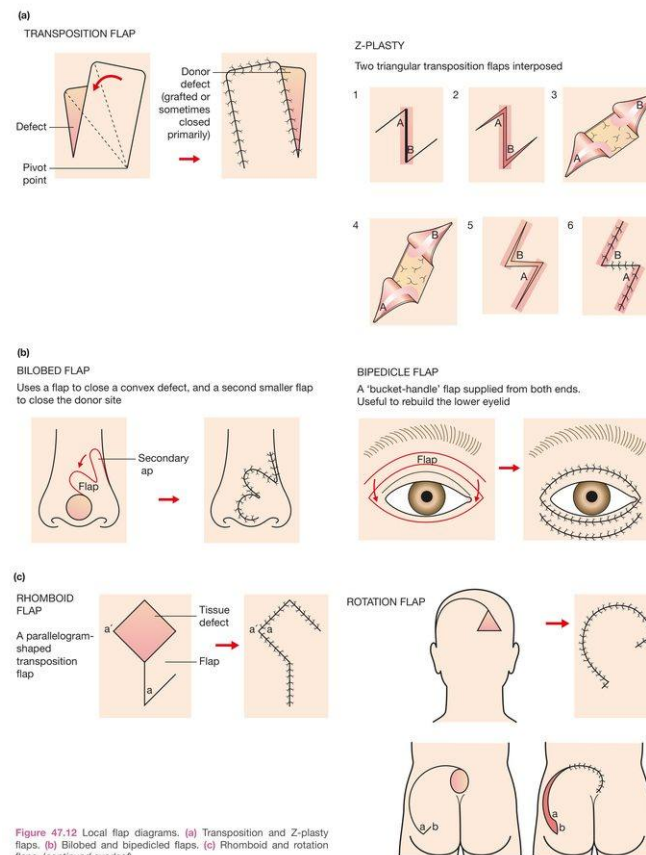


Figure 47.12 Local flap diagrams. (a) Transposition and Z-plasty flaps. (b) Bilobed and bipedicle flaps. (c) Rhomboid and rotation flaps. (continued overleaf)

Figure 14.2b.1 — Local flap diagrams. (a) Transposition and Z-plasty flaps. (b) Bilobed and bipedicle flaps. (c) Rhomboid and rotation flaps.

BL 28e Fig. 47.12, p. 691

By contiguity and distance

- **Local:** bilobed nasal flap; **regional:** forehead flap, pedicled medial plantar artery flap, pedicled medial sural artery perforator flap.
- **Distant pedicled:** groin flap to the hand (pedicle divided at 3 weeks), cross-leg flap.
- **Free flap:** detached and revascularised by microvascular anastomosis to recipient vessels — anterolateral thigh, DIEP, radial forearm, free fibula.

Pearls

- ✓ Five Cs: Circulation, Composition, Contiguity, Contour, Conditioning.
- ✓ Random = no dominant vessel; axial = named vessel; perforator = islanded on one perforator.
- ✓ Cormack–Lamberty A–D for fasciocutaneous; Mathes–Nahai I–V for muscle flaps.
- ✓ Delay improves the blood supply so that a larger flap can be raised.
- ✓ Bilobed flap — convex nasal defect; bipedicle — lower eyelid.

Sources

- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Flaps: five Cs; Cormack and Lamberty; Mathes and Nahai”, p. 690.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Local flap diagrams”, pp. 691–692; Fig. 47.12, pp. 691–692.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Local, regional, distant and perforator flaps”, pp. 693–697.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Chimeric and venous flow-through flaps; microsurgery”, p. 699.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Pedicled groin flap; angiosome”, p. 683.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Trunk: adjacent tissue transfer”, p. 877.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Perineal reconstruction: random and axial pattern flaps”, pp. 881–882.
- SAB 22e, ch. 119 “Hand Surgery”, §“Local options for flap coverage of finger defects (Table 119.5)”, p. 1999.

Asked in: Paper II · Jun 2020 · Q10 (10 marks)

“Principles of reconstruction in head and neck cancer surgery.”

Aims of reconstruction

- Restore **form and function** — speech, chewing, swallowing, **oral continence**, airway — and aesthetics; simply “closing the hole” is no longer acceptable.
- Reconstruction is an **ever-present consideration in surgical planning**; its availability allows resection of tumours that would otherwise be unresectable.
- Decisions about reconstruction **must not influence the ablative procedure** (oncological clearance comes first).
- Defects of the upper aerodigestive tract need **reliable wound coverage while maximising function**; mucosal and airway defects need resilient tissue that resists saliva and bacteria.

General principles

- **Defect analysis** — identify which structures are missing (skin, mucosa, fat, muscle, bone, nerve) and plan a component to replace each.
- **Replace like with like** — the single most important principle.
- **Use the simplest method that meets the aims** — the reconstruction ladder (Figure 53.7).
- **Vascularised tissue** for a previously irradiated recipient site; grafts take poorly on exposed bone, irradiated tissue, cartilage and tendon.
- **Always have an alternative** — first-, second- and third-line options for a defect that grows at surgery or a donor pedicle resected with tumour; a mandibular defect may need both bone and soft-tissue flaps if tumour reaches the buccal surface or floor of mouth.
- **Bony defects** that compromise three-dimensional structure need bone replacement — fibula, scapula or iliac crest.
- The **oral cavity is a unique site** — hard and soft tissues, dentition, bathed in saliva, opening of the aerodigestive tract, rich motor and sensory innervation — so both bone and soft tissue are replaced with similar tissue.
- **Donor selection** weighs pedicle length, area of harvestable tissue, tissue composition, vessel diameter (donor and recipient), donor-site morbidity and suitability for **two-team operating**.
- **Anticipate adjuvant radiotherapy** (xerostomia, scar contracture, impaired healing, osteoradionecrosis; tissue atrophy) — incorporate extra bulk, “overcorrect” volume; radiotherapy does not correlate with free-flap failure.
- **Fit the oncological timeline**: adjuvant radiation usually starts 4–6 weeks after surgery, so a slow-healing, radiation-intolerant skin graft may be a worse choice than a “more complicated” free flap that heals promptly.
- **Aesthetic units** treated as a whole; incisions hidden in relaxed skin tension lines and natural creases; access incisions planned jointly with the ablative surgeon.
- **Timing**: immediate reconstruction when a vital structure (e.g. pharynx) must be restored — planes accessible, bed not fibrotic; maxillectomy defects may be left unreconstructed (or obturated) initially to allow debridement and monitoring.
- **Secondary procedures** (debulking, liposuction) about 3–6 months later or after adjuvant therapy.

- Healing by secondary intention
- Primary closure
- Skin grafting
- Local flaps
- Regional flaps
- Microvascular free tissue transfer

Figure 14.3a.1 — The reconstruction ladder.

BL 28e Fig. 53.7, p. 823

Patient assessment and optimisation

- **Frailty** (e.g. modified frailty index mFI-5) predicts complications better than age; age alone is not an exclusion.

- **Smoking** → tissue necrosis, poor healing, thrombosis; cessation for **≥4 weeks** is optimal (cessation programmes cut wound-healing complications by 52%); nicotine replacement aids adherence.
- **Nutrition:** prealbumin <10 mg/dL or albumin <2.7 g/dL = moderate–severe malnutrition → preoperative oral or tube supplements; correct HbA1c.
- Patients unfit for prolonged anaesthesia (age, peripheral arterial disease, cardiopulmonary disease), after a failed free flap, or in salvage surgery with few recipient vessels → **regional pedicled flap**.

Options — the reconstruction ladder applied

Table 14.3a.1. Reconstructive options in head and neck cancer

Option	Use	Limitations
Secondary intention / primary closure	Small defects; small posterior mandibular defects may be left unreconstructed	Not for large or composite defects
Split-thickness skin graft	Defect too large for primary closure but too small for a free flap; lining over which an obturator or orbital prosthesis sits; flap donor sites; medically unfit patients	Contracture; poor colour and thickness match; fails on bone, cartilage, tendon, irradiated beds
Full-thickness skin graft	Small defects in cosmetically important areas (nose, temple)	Limited size
Local flaps	Small defects: facial artery myomucosal (FAMM), nasolabial, buccal fat pad, tongue and palatal flaps — safe owing to the rich head and neck blood supply	Limited tissue volume
Regional pedicled flaps	Temporalis, platysma; pectoralis major, deltopectoral, latissimus dorsi — shorter operation, versatile, easy to harvest	Higher failure than free flaps; reach limited by the pedicle
Microvascular free flap	Gold standard for most defects — any shape, size, tissue composition	Long anaesthesia; microsurgical expertise; recipient vessels needed
Prosthesis / obturator	Maxillectomy; in selected patients the simplest effective method	Free flap gives better function and aesthetics than obturation

Source: BL 28e pp. 822–823, 825, 828; SAB 22e pp. 805, 809

Choice of reconstruction by defect

- **Tongue / floor of mouth:** radial forearm free flap (RFFF) — thin, pliable, non-hirsute, long pedicle (18 cm), where bulk is not needed; anterolateral thigh (ALT) — bulk to seal the oral cavity against the soft palate in speech and swallowing, multiple skin paddles.
- **Segmental mandible:** composite free flap is the gold standard; **fibula** commonest (bone up to 14 cm), also DCIA/iliac crest, scapula, composite RFFF.
 - Anterior reconstruction is always more challenging and particularly important for function; small posterior defects may be left unreconstructed.
 - Follow the natural mandibular curve with the flap's curvature to reduce osteotomies; in the edentulous, slightly shorten the span to avoid a class III (prominent chin) appearance.
 - Free bone can become a nidus of persistent infection after radiotherapy.
- **Maxilla** — aims: restore facial contour, **separate sinonasal cavities from the mouth**, soft-palate competence, dental rehabilitation; Brown and Shaw classification guides choice (Figure 53.12):
 - Class I–II: local flaps, RFFF or obturator; composite flap for anterior and IIc defects.
 - Class III–IV (orbital and cheek support lost): composite free flap (e.g. thoracodorsal system).
- **Pharynx:** vascularised patch or onlay (pedicled pectoralis major myocutaneous, RFFF, ALT) reduces pharyngocutaneous fistula; total pharyngolaryngectomy — jejunum, ALT or gastric transposition.
- **Complex composite defects:** chimeric flaps with independently mobile bone and soft tissue (subscapular / thoracodorsal system).

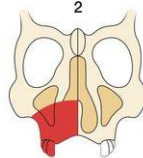
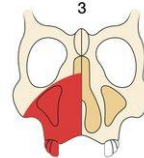
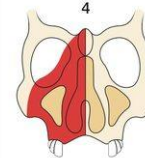
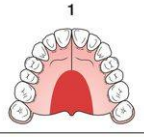
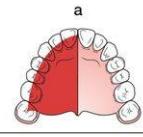
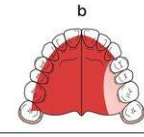
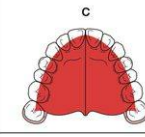
Vertical component				
Horizontal component				
Local flap				
Pedicle flap				
Obturator				
Soft-tissue FF				
Composite FF				

Figure 14.3a.2 — Maxillectomy defect classification and proposed reconstructions (FF, free flap).

BL 28e Fig. 53.12, p. 827

Peri-operative principles

- **Airway:** low threshold for tracheostomy after bilateral or previous neck dissection, large resection with reconstruction, difficult airway; consider for segmental mandibulectomy, lateral floor-of-mouth resection with reconstruction and previous irradiation — a bulky flap and oedema threaten the airway, and a tracheostomy secures access for an emergency return to theatre.
- **Recipient vessels:** neck dissection exposes them for microvascular anastomosis; ensure the carotid artery is well covered at closure.
- **Pharyngocutaneous fistula** risk (previous radiation, malnutrition, postoperative hypothyroidism) is reduced by vascularised tissue over the pharyngeal closure; the pectoralis major muscle flap is the gold standard for closing a large fistula.
- **Flap monitoring:** every 1–2 h in the first days — Doppler, capillary refill, temperature, scratch test; keep the patient “wet, warm and comfortable”.
- **Flap failure:** success >95%; most failures within **48 h**, usually **venous** thrombosis.
 - Avoid pedicle kinking by head position and compression by tracheostomy ties or oxygen masks.
 - Failure → emergency re-exploration (salvage ~50%); venous congestion → leeches; a second free flap succeeds in 85–94%.
- **Adjuncts:** virtual surgical planning with cutting guides (shorter operation, greater accuracy); zygomatic implants / zygomatic implant perforator flaps for dental rehabilitation after maxillectomy.
- **Rehabilitation:** speech and swallowing therapy; about 17% of free-flap patients remain gastrostomy-dependent.

Pearls

- ✓ Like with like; simplest method that meets the aims; vascularised tissue in irradiated fields; always a back-up plan.
- ✓ Reconstruction must never compromise the resection.
- ✓ Free flap = gold standard; regional pedicled flaps for the unfit, failed free flap or salvage.
- ✓ RFFF for thin defects; ALT for bulk (tongue); fibula for segmental mandible.
- ✓ Maxilla: Brown–Shaw class guides local flap/obturator vs composite free flap.
- ✓ Most free-flap failures occur within 48 h and are venous.

Sources

- BL 28e, ch. 53 “Oral cavity cancer”, §“Oral cavity cancer: key surgical decisions; reconstruction; mandible and maxillary reconstruction”, pp. 821–825; Fig. 53.7, p. 823; Tables 53.5–53.6.
- BL 28e, ch. 53 “Oral cavity cancer”, §“Maxillectomy defect classification; zygomatic implants; virtual surgical planning”, pp. 827–828; Fig. 53.12, p. 827.
- BL 28e, ch. 52 “The pharynx, larynx and neck”, §“Pharyngeal reconstruction”, pp. 791–792.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Microsurgery; flap monitoring”, pp. 699–700.
- SAB 22e, ch. 67 “Head and Neck Cancer”, §“Head and neck cancer: reconstruction in surgical planning”, p. 805.
- SAB 22e, ch. 67 “Head and Neck Cancer”, §“Head and neck cancer: reconstruction; flap failure”, pp. 809–810.
- SAB 22e, ch. 67 “Head and Neck Cancer”, §“Gastrostomy dependence”, p. 812.
- SAB 22e, ch. 67 “Head and Neck Cancer”, §“Sinonasal cancer: maxillectomy defects”, p. 817.

14.3b

Local flaps for reconstruction of the lip

2×

Asked in: Paper IV · Aug 2021 · Q3(a) (6 marks) ; Paper IV · Jun 2022 · Q10(a) (5 marks)

"Describe the various local flaps used for reconstruction of the lip."

"Local flaps for reconstruction of lip."

NOT COVERED IN SOURCE

Named local lip flaps: searched BL 28e and SAB 22e full text and the BL index for 'Abbe', 'Estlander', 'Karapandzic', 'Bernard', 'Burow lip', 'Gillies fan / fan flap', 'cross-lip', 'lip switch', 'Webster', 'Johanson step', 'vermillionectomy / lip shave', 'mucosal advancement', 'lip reconstruction / reconstruction of the lip', 'lip cancer / carcinoma of the lip', 'wedge excision', 'vermillion', 'commissure'; chapters BL 47 (Plastic and reconstructive surgery), BL 50 (Cleft lip and palate), BL 53 (Oral cavity cancer), SAB 66 (Benign diseases of head and neck), SAB 67 (Head and neck cancer), SAB 69 (Plastic surgical considerations in cancer surgery). Neither book describes the lip-specific flaps (lip-switch, fan, commissure or circumoral advancement flaps) or defect-size rules for the lip. The closely related material the books do hold — lip anatomy, the principles that govern local reconstruction in the face and mouth, and the local flap movements — follows, labelled as such.

Related material: lip anatomy relevant to reconstruction

- **Extent:** from the junction of facial skin and **vermilion border** to where upper and lower lips meet with the mouth closed.
- **Oral commissures** (lateral-most points): their size and position matter for **oral competence and mouth opening**.
- **Muscle chains of the face:**
 - **Bilabial chain** — orbicularis oris (oblique and horizontal heads in the upper lip; lower-lip part).
 - **Nasolabial** (transverse nasalis, levator labii superioris alaeque nasi, levator labii superioris) and **labiomental** (depressor anguli oris, depressor labii inferioris, mentalis).
- **Muscular continuity** is the basis of lip repair — restoring normal or near-normal anatomy promotes normal function.
- Lip SCC drains to levels **IA, IB, II** (lower lip) and **IB, II and perifacial nodes** (upper lip) — relevant when a neck dissection accompanies lip resection.

Related material: principles of local reconstruction in the lip region

- Aim: restore **speech, chewing, swallowing and oral continence** as well as appearance; reconstruction must not influence the resection margin.
- **Replace like with like;** use the **simplest method** on the reconstruction ladder (secondary intention → primary closure → skin graft → local → regional → free flap).
- **Local flaps in the head and neck are safe and predictable** owing to the extensive and consistent blood supply.
- **Vermilion border alignment:** even a **1-mm step** is noticeable at a distance; line up the vermilion edges first and close in layers (deep absorbable sutures, then skin), without tension.
- **Aesthetic units** reconstructed as a whole; incisions in relaxed skin tension lines and natural creases.
- Irradiated or previously operated beds need **vascularised tissue**; always plan an alternative.

Related material: local flap movements available (Bailey & Love classification by contour)

- **Advancement:** tissue moved straight into the defect; Burow's triangles excised at the base help it slide; **V–Y** variant — e.g. V-to-Y nasolabial advancement flap after excision of an alar-groove BCC, hatchet flap with a back cut, Atasoy and Kutler V–Y fingertip flaps.
- **Transposition:** flap swung about a **pivot point** into an adjacent defect; donor site closed primarily or grafted — rhomboid flap, bilobed flap (second smaller flap closes the donor site), Z-plasty.
- **Rotation:** curved flap rotated into the defect, often with a back cut (scalp, pilonidal defects).
- **Bipedicle:** "bucket-handle" flap supplied from both ends (lower eyelid).
- **Axial local and regional flaps of the face and mouth:** forehead flap on the supratrochlear artery (pedicle divided at a second stage); nasolabial, facial artery myomucosal (FAMM), buccal fat pad and tongue flaps for small oral

defects.

- **Circulation:** random pattern (no dominant vessel) versus axial pattern (named vessel); **delay** (partial elevation and resetting) improves blood supply so that a larger flap can be raised.
- Defects too large for local tissue in the oral cavity → **regional** (temporalis, platysma, pectoralis major, deltopectoral) or **free flaps** (RFFF, ALT).

Pearls

- ✓ Lip function depends on orbicularis oris continuity and commissure position.
- ✓ Vermilion: a 1-mm misalignment shows — align it first.
- ✓ Head and neck local flaps are reliable because of the rich blood supply.
- ✓ Climb the ladder only as far as needed; like with like.

Sources

- SAB 22e, ch. 66 “Benign Diseases of Head and Neck”, §“Oral cavity: lip”, p. 793.
- SAB 22e, ch. 67 “Head and Neck Cancer”, §“Oral cavity cancer: lymph node drainage of the lips”, p. 812.
- SAB 22e, ch. 66 “Benign Diseases of Head and Neck”, §“Cuts to lips: vermilion alignment”, p. 802.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Goals of reconstruction”, p. 866.
- SAB 22e, ch. 119 “Hand Surgery”, §“Local options for flap coverage of finger defects”, p. 1999.
- BL 28e, ch. 50 “Developmental abnormalities of the face, mouth and jaws:”, §“Cleft lip: muscle chains of the face; muscular reconstruction”, pp. 742–745.
- BL 28e, ch. 3 “Wound healing and tissue repair”, §“Wound closure: vermilion border”, p. 36.
- BL 28e, ch. 31 “Maxillofacial trauma”, §“Facial soft-tissue injuries: vermilion border”, p. 409.
- BL 28e, ch. 53 “Oral cavity cancer”, §“Oral cavity cancer: reconstruction; reconstruction ladder”, pp. 822–823.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Flaps: classification; local flap diagrams; local flaps”, pp. 690–696.

14.3c

Flaps commonly used in oral malignancy

1×

Asked in: Paper II · Jun 2025 · Q3(a) (5 marks)

“Describe commonly used flaps in cases of oral malignancy.”

Principles of flap choice

- Replace **like with like** (soft tissue, bone or both) using the simplest adequate rung of the reconstruction ladder; vascularised tissue for irradiated fields.
- **Free tissue transfer is the gold standard**; local or regional flaps where comorbidity, a failed free flap or salvage surgery precludes it.

Local and regional flaps

- **Local** (small defects not needing bulk or bone): **facial artery myomucosal (FAMM)**, **nasolabial**, **buccal fat pad**, **tongue** and **palatal** flaps.
- **Regional, head and neck: temporalis** (also for maxillectomy defects), **platysma**.
- **Pectoralis major myocutaneous** (thoracoacromial artery): robust muscle, seals tenuous tissue and fills dead space; widely used before microvascular techniques were refined and still applied for its versatility.
- **Deltopectoral** and **latissimus dorsi** (thoracodorsal vessels) flaps from more distant sites.
- Regional flaps: shorter operation, easy harvest — but **higher failure rate than free flaps** and reach limited by the pedicle.

Free soft-tissue flaps

- **Radial forearm free flap (RFFF)** — the quintessential oral and oropharyngeal flap:
 - Radial artery, its two venae comitantes and the cephalic vein; long pedicle (**18 cm**), vessels free of atherosclerosis.
 - Thin, pliable, non-hirsute skin, $\pm$ fascia, brachioradialis or radius.
 - **Allen test** (or Doppler of ulnar flow) before harvest; non-dominant arm; donor site skin-grafted, wrist splinted in extension for about 1 week.
- **Anterolateral thigh (ALT) flap** — used when the defect is too large for an RFFF:
 - Descending branch of the **lateral circumflex femoral artery** with venae comitantes; pedicle 12 cm.
 - Perforators are septocutaneous or musculocutaneous and are mapped with a Doppler probe; skin paddle up to 16 × 8 cm; multiple paddles or muscle possible.
 - Donor site usually closed primarily; may be too bulky in patients with much subcutaneous fat.

- **Alternatives:** rectus abdominis, latissimus dorsi, medial sural artery perforator, thoracodorsal artery perforator and lateral arm flaps (lateral arm used for partial glossectomy).

Composite (bone-containing) free flaps

Table 14.3c.1. Composite flap donor sites for oral cavity reconstruction

Flap	Bone length	Strengths	Drawbacks
Fibula (peroneal vessels)	14 cm	Most used globally; long bone; thin skin paddle; good for implants; two-team operating	Vessels may be atherosclerotic; skin paddle occasionally unreliable
DCIA / iliac crest	12 cm	Greatest bone volume; best for implants; internal oblique muscle paddle	Short pedicle; vessels may be small; donor morbidity
Scapula	10 cm	Large vessels spared from atherosclerosis; two soft-tissue flaps or chimera with latissimus dorsi	Short pedicle; poor for two-team operating
Composite RFFF	12 cm (low volume)	Long pedicle; reliable skin	Little bone volume; not suitable for implants; lacks bulk

Source: BL 28e Table 53.6, p. 824; SAB 22e pp. 809–810

- **Fibula harvest:** leave **6–8 cm** of fibula at each end (ankle stability, common peroneal nerve); identify skin perforators; **CT angiography** first to confirm foot perfusion.
- Composite free flaps are the **gold standard for segmental mandibular defects**; chimeric flaps (thoracodorsal system) when bone and soft tissue must move independently.

Choice by subsite

- **Tongue, floor of mouth, buccal and retromolar mucosa:** RFFF where bulk is not wanted; ALT where bulk is needed to seal the oral cavity against the soft palate in speech and swallowing.
- **Mandible:** fibula first; reconstruction of anterior defects is particularly important (and challenging).
- **Maxilla:** class I–II — local flaps, RFFF or obturator; class III–IV — composite free flap; temporalis flap or obturator also used after maxillectomy.



Figure 14.3c.1 — Radial forearm free flap used to reconstruct a left lateral tongue defect — thin, pliable tissue suits a modest soft-tissue defect.

BL 28e Fig. 53.8, p. 824

Pearls

- ✓ Small defects: FAMM, nasolabial, buccal fat pad, tongue, palatal flaps.
- ✓ Regional workhorse: pectoralis major myocutaneous flap.
- ✓ Soft tissue: RFFF (thin) vs ALT (bulk).
- ✓ Bone: fibula first; DCIA for bone volume; scapula for chimeric needs.
- ✓ Allen test before RFFF; CT angiography before fibula.

Sources

- BL 28e, ch. 53 “Oral cavity cancer”, §“Oral cavity cancer: reconstruction; soft-tissue and composite flaps; mandible reconstruction”, pp. 822–825; Fig. 53.8, p. 824; Tables 53.5–53.6.
- BL 28e, ch. 53 “Oral cavity cancer”, §“DCIA free flap”, p. 827.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Microsurgery: ALT pedicle”, p. 699.

- SAB 22e, ch. 67 “Head and Neck Cancer”, §“Head and neck cancer: regional and free flaps”, pp. 809–810.
- SAB 22e, ch. 67 “Head and Neck Cancer”, §“Maxillectomy defects: temporalis flap”, p. 817.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Head and neck reconstruction; pharyngocutaneous fistula”, pp. 869–871.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Pectoralis major flap pedicles”, p. 874.

14.4 Bio-engineered skin substitutes and their current status

2×

Asked in: Paper IV · Dec 2021 · Q4(a) (5 marks) ; Paper IV · Dec 2025 · Q10(a) (5 marks)

“Describe various bio-engineered skin substitutes and their current status in patient care.”

Definition and rationale

- **Skin substitutes:** engineered dressings designed to facilitate wound healing by **replicating as many key functions of skin as possible**; they replace the epidermis, the dermis or both, with a cellular or acellular matrix.
- **Tissue engineering:** application of engineering and life-science principles to develop biological substitutes that restore, maintain or improve function.
- **Why needed:** autograft is limited by donor-site morbidity, pain, scarring and donor-site availability — at **>50% TBSA** full-thickness burn the burn exceeds the donor area.
- **How they work:** protective dressing that limits bacterial colonisation and fluid loss, and an active scaffold that draws in fibroblasts, keratinocytes, endothelial cells and macrophages → growth factors → angiogenesis, matrix deposition, re-epithelialisation; the substitute is replaced by native tissue (Figure 45.11).
- **Described by** five characteristics: cellularity, material (allograft/xenograft/synthetic), layering, replaced region, permanence.

Types

Table 14.4.1. Bio-engineered skin substitutes by layer replaced

Type	Product — composition	Main use
Epidermal (cellular)	Epicel — cultured epidermal autograft (patient keratinocyte sheets on a carrier); MySkin (spray); ReCell — autologous skin-cell suspension prepared in theatre, expands 80:1	Massive burns with very limited donor sites; ReCell for partial-thickness burns or over meshed autograft
Dermal (mostly acellular)	Integra — bovine collagen–chondroitin-6-sulphate matrix with outer silastic sheet; AlloDerm — cryopreserved acellular cadaveric human dermis; NovoSorb BTM — biodegradable polyurethane foam with sealing membrane; Kerecis — acellular cod-fish skin	Full-thickness burns; exposed tendon, bone, joint capsule; complex and diabetic foot wounds; AlloDerm in breast reconstruction
Bilayer	Apligraf — living bilayer of human fibroblasts and keratinocytes; StrataGraft — allogeneic keratinocyte line over dermal fibroblasts in collagen gel; Biobrane — acellular silicone, nylon mesh, porcine collagen	Apligraf: venous and diabetic ulcers; StrataGraft: burns; Biobrane: superficial burns, donor sites

Source: BL 28e p. 688; SAB 22e pp. 494–496, 522–523; SCH 11e pp. 259–260

- **Biological temporary covers** used alongside: cadaver allograft and xenograft (pig, fish skin) — rejected eventually; xenograft is cheaper and works as well as allograft for temporary cover.

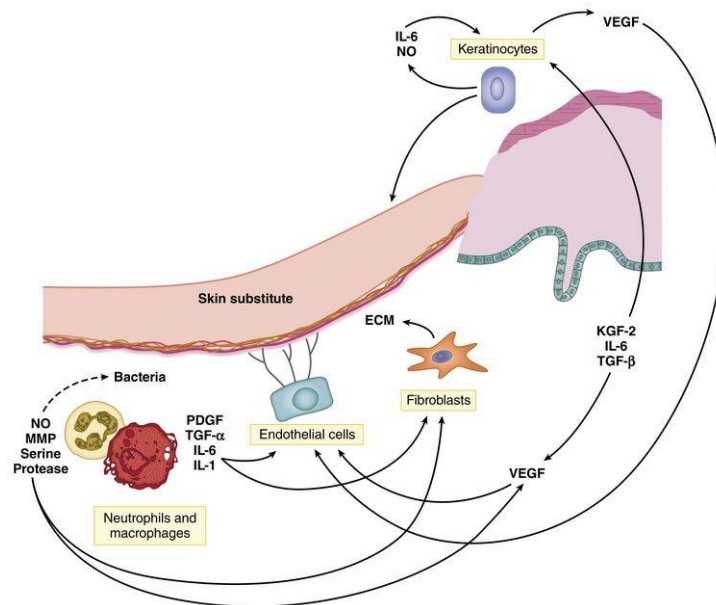


Figure 14.4.1 — The effect of skin substitutes in the wound bed: cellular or acellular substitutes induce an influx of endogenous cells whose cytokines and growth factors drive angiogenesis, matrix deposition and re-epithelialisation.

SAB 22e Fig. 45.11, p. 522

Current status in patient care

- **Burns** — the first clinical use of tissue engineering:
 - **Integra / BTM**: dermal matrix engrafts as a neodermis; silicone layer removed at about **2–3 weeks** and replaced by a thin autograft — “active” temporisation that improves thin meshed grafts; Integra remains in successful use; BTM used for full-thickness and electrical burns.
 - **Biobrane / Suprathel**: superficial second-degree burns and split-skin donor sites, applied **within 72 h**; adherent, painless, no dressing changes.
 - **Cultured epithelial autografts**: truly massive burns (>80% TBSA); Epicel is a humanitarian device for burns >30% TBSA — but 2–3 weeks to grow, **50–75% take**, fragile, scar-prone, SCC risk and expensive; conventional grafting gave shorter stay and fewer reconstructions.
 - **ReCell**: approved, supported by two phase III randomised trials; donor-sparing.
 - **StrataGraft** (FDA approved 2021): outcomes equivalent to autograft without a donor site, but about \$40/cm² and **not yet standard of care**.
- **Chronic wounds**: Apligraf licensed for **diabetic and venous ulcers**; Integra and BTM for wounds unsuitable for grafting; BTM contraindicated in **infected or necrotic** wounds.
- **Limitations**:
 - High cost; meticulous technique needed to avoid failure.
 - No antimicrobial activity → risk of invasive infection; Integra needs **two operations**.
 - Engineered skin lacks normal features (e.g. colour); a perfect permanent synthetic substitute remains elusive; over 70 products exist and the wound and patient decide the best fit.
- **Future**: cultured autologous skin with the patient’s own keratinocytes and fibroblasts, mesenchymal stem cells and exosomes, gene-corrected cells (epidermolysis bullosa); cost–benefit must be shown.

Pearls

- ✓ Skin substitutes replace epidermis, dermis or both; cellular or acellular.
- ✓ Integra = bovine collagen–GAG dermis + silastic “epidermis”; autograft at ~2–3 weeks.
- ✓ Cultured epithelial autograft: massive burns, poor take (50–75%), fragile.
- ✓ Apligraf (living bilayer): venous and diabetic ulcers.
- ✓ Autograft remains the gold standard; substitutes are costly and lack antimicrobial action.

Sources

- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Grafts: allograft, xenograft and skin substitutes”, p. 688.
- BL 28e, ch. 46 “Burns”, §“Burns: use of skin grafts and skin substitutes”, pp. 676–677.
- BL 28e, ch. 46 “Burns”, §“Burns: electrical injury; recent advances”, pp. 679–680.
- BL 28e, ch. 4 “Tissue engineering and regenerative therapies”, §“Tissue engineering: skin”, p. 43.

- SAB 22e, ch. 45 “Management of Chronic Wounds”, §“Management of chronic wounds: tissue replacements; bioengineering”, pp. 521–524; Fig. 45.11, p. 522; Table 45.6, p. 523.
- SAB 22e, ch. 43 “Burns”, §“Burns: synthetic and biologic dressings; excision and grafting”, pp. 494–496.
- SAB 22e, ch. 50 “Regenerative Medicine and Its Applications to Surgery”, §“Regenerative medicine: skin”, p. 614.e8.
- SAB 22e, ch. 23 “Wound Healing”, §“Wound healing: dressings table”, p. 108.
- SCH 11e, ch. 8 “Burns”, §“Burns: skin substitutes”, pp. 259–260.

14.5

Cleft lip and palate — LAHSHAL classification and principles of management and surgery

2×

Asked in: Paper IV · Apr 2023 · Q2 (3+7 marks) ; Paper II · Dec 2021 · Q6(a) (5 marks)

“Describe ‘LAHSHAL’ system of classification. Briefly discuss the principles of surgery for the clinical conditions described by this classification.”

“Enumerate principles of management of cleft lip and palate.”

NOT COVERED IN SOURCE

LAHSHAL coding rules: searched BL 28e and SAB 22e full text and the BL index for ‘LAHSHAL’, ‘LAHS’, ‘Kernahan’, ‘striped Y’, ‘incomplete cleft’, ‘submucous cleft’, ‘bifid uvula’, ‘Veau’, ‘orofacial cleft’, ‘cleft palate’; chapters BL 50 (Cleft lip and palate), SAB 66 (Benign head and neck), SAB 117 (Pediatric surgery); SAB has no cleft chapter. BL prints the LAHSHAL diagram (the segments and the side) but does not describe the notation for writing the code. The diagram, the book’s definitions of complete and incomplete cleft palate and the Veau classes are given below.

1. The LAHSHAL classification

- **Purpose:** cleft lip and/or palate is heterogeneous; a classification defines the **extent** and **laterality** (left / right / bilateral) of the cleft — of diagnostic and, increasingly, **prognostic** value.
- **Two phenotypes** underlie every code:
 - **Isolated cleft palate.**
 - **Cleft lip with or without the alveolus** (tooth-bearing part of the jaw) **or palate.**
- **LAHSHAL code** (Fig. 50.2a): each letter is one anatomical segment that may be cleft, recorded for the **right** and **left** sides:
 - **L** = lip; **A** = alveolus — paired, right and left.
 - **H** = hard palate; **S** = soft palate — the midline segments of the palate.
- Clefts occur at the **points of fusion of facial processes**; the segments map onto the embryology (table below).
- Single system advocated, but even with one system clinicians may **interpret findings inconsistently**; UK national audit collects outcome data for **unilateral cleft lip and palate (UCLP)** for inter-centre comparison.

Table 14.5.1. LAHSHAL segments — origin and the cleft each describes

Segment	Embryological origin	Cleft in this segment
L — Lip	Median nasal process (philtral part) + maxillary processes (rest of lip)	Failure of fusion of frontonasal and maxillary processes → unilateral or bilateral cleft lip
A — Alveolus	Primary palate (anterior to the incisive foramen), from the median nasal process	Cleft alveolus; dental anomalies concentrate here (lateral incisor)
H — Hard palate	Secondary palate (behind the incisive foramen), from the maxillary processes	Incomplete: still attached to nasal septum and vomer; complete: septum and vomer separated from the palatine processes
S — Soft palate	Secondary palate, most posterior part	Failure of fusion or descent of the palatal shelves; levator fibres run anteroposteriorly into the hard-palate edge

Source: BL 28e pp. 740–743

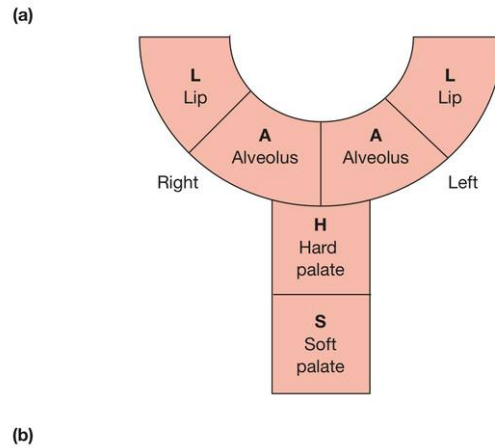


Figure 14.5.1 — (a) The LAHSHAL code. (b) The Veau classification system (classes I–IV).

BL 28e Fig. 50.2, p. 741

- **Distribution:** cleft lip alone **15%**; cleft lip and palate **45%**; isolated cleft palate **40%**; incidence about **1 in 600** live births.
- Cleft lip $\pm$ palate is commoner in **males**, isolated cleft palate in **females**; UCLP affects the **left side in 60%**.

2. The conditions the code describes — anatomy that surgery must correct

- **Unilateral cleft lip:** muscle rings disrupted on one side $\rightarrow$ asymmetric lip and nose, involving external nasal cartilages, septum and premaxilla; nasal skin displaced onto the lip, labial skin retracted, vermilion and mucosa altered.
- **Bilateral cleft lip:** greater, usually symmetrical disruption $\rightarrow$ **flared nose**, **protrusive premaxilla**, and a muscle-free **prolabium**.
- **Cleft palate:** levator veli palatini (key muscle of velopharyngeal closure) runs anteroposteriorly and inserts into the posterior edge of the hard palate instead of forming a transverse sling; in a complete cleft the palatal fibromucosa is reduced, while maxillary and gingival fibromucosa are unchanged.
- No true tissue **hypoplasia** (except rare conditions such as holoprosencephaly) — the tissues are **displaced, deformed and underdeveloped**.

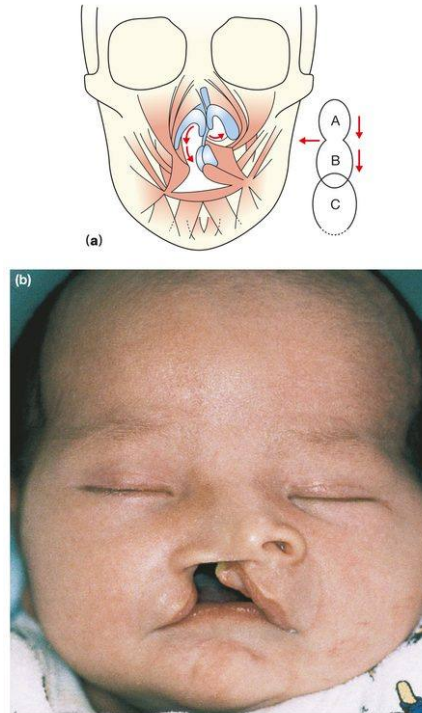


Figure 14.5.2 — (a) Disruption of the nasolabial (A) and bilabial (B) muscle chains in unilateral (right) cleft lip; C, labiomental. (b) Unilateral cleft lip before muscular reconstruction.

BL 28e Fig. 50.4, p. 743

3. Principles of management

- **Aim:** facilitate **normal development and well-being**; outcomes measured are **speech, facial growth, general well-being and dental health**.
- **Multidisciplinary cleft team**, from diagnosis onwards:
 - Cleft coordinator; **clinical nurse specialist** (first contact — feeding, airway, readiness for surgery).
 - Paediatrician; **speech and language therapist**; ENT/audiology; paediatric dentist; **orthodontist**; clinical psychologist; cleft surgeon.
- **Antenatal diagnosis:** cleft lip is usually seen on the **20-week anomaly scan**; isolated cleft palate cannot be diagnosed on routine scanning → referral to the cleft team.
- **Screen every child for other anomalies:** clefts occur in over 300 syndromes (commonly **Stickler, DiGeorge, Down, Apert, Treacher Collins**); isolated cleft palate is the most often syndromic → genetic counselling where indicated.
- **Neonatal feeding:**
 - Expressed breast milk; **soft squeezable bottles** and modified teats; feeding plates in some countries.
 - **Nasoalveolar moulding (NAM)** narrows the cleft and shapes the nose pre-operatively — long-term benefit conflicting.
- **Airway — Pierre Robin sequence** (isolated cleft palate, retrognathia, glossoptosis):
 - Intermittent obstruction managed **conservatively**; prone nursing.
 - Severe: supplemental oxygen, **nasopharyngeal airway**, tracheostomy; labioglossopexy (less common); mandibular distraction lacks consensus.

4. Principles of surgery

- **Goal:** normal or near-normal anatomy of lip, nose and palate → normal function → normal growth of lip, nose, palate and facial skeleton.
- **Muscle reconstruction is key** — of lip, nose and soft palate; most surgeons hold the muscle repair more important than the (eponymous) skin incision.
- **Staged from anterior to posterior** (lip → soft palate), timed to development; surgery in infants is **time sensitive**.
- **Limited intervention:** one to three planned operations in childhood, depending on cleft type; outcomes audited annually.

Table 14.5.2. Surgical protocol by age (UK protocol, popularised in Norway)

Age	Procedure	Principle
3–6 months	Cleft lip/nose repair + anterior palate closure	Single-layer vomer mucosal flap for the anterior palate; muscle repair of the lip
6–12 months	Definitive cleft palate repair — intravelar veloplasty (IVVP)	Levator muscles dissected free and sutured in the midline to recreate the muscular sling ; closure during early speech development
1–7 years	Speech surgery if needed: palate re-repair or pharyngoplasty ; lip revision if needed	For velopharyngeal incompetence shown on videofluoroscopy / nasendoscopy
7–12 years	Alveolar bone grafting (ABG)	Early secondary 5–7 years (lateral incisor root); late secondary 8–11 years when the canine root is one-half to two-thirds formed
After ~14–15 years	Major nasal revision — open septorhinoplasty with septal or conchal cartilage grafts	Only minor nasal adjustment in the growing child
16–17 (F) / 17–19 (M) years	Orthognathic surgery — maxillary advancement or bimaxillary osteotomy	Corrects maxillary hypoplasia when facial growth is complete

Source: BL 28e pp. 745–747

5. Follow-up and secondary care

- **Hearing:** Eustachian tube dysfunction → **otitis media with effusion** in palatal clefts; regular audiological screening (hearing underpins speech).
- **Speech:**
 - **Velopharyngeal incompetence (VPI)** → air escape and **hypernasality**.
 - **Articulation errors** — compensatory for VPI or from jaw/dental abnormalities.
- **Dental:** delayed development and eruption, **hypodontia/hyperdontia** at the cleft (maxillary lateral incisor); prevention with dietary advice, fluoride and fissure sealants.
- **ABG objectives:**
 - Bony support for adult teeth and their eruption into the arch.
 - **Stabilise the premaxilla** in bilateral clefts; close the residual alveolar cleft.
- Short pre-graft orthodontic expansion in fewer than 50% of UCLP; marked hypodontia → no ABG, teeth replaced by denture, adhesive bridge or implant (after skeletal maturity).
- **Orthodontics** in two phases: mixed dentition (8–10 years, to prepare for ABG) and permanent dentition (12–18 years, definitive alignment ± orthognathic preparation).
- **Lip revision** for misaligned vermilion or asymmetry; **nasal revision** for alar base malposition, poor tip projection or septal deviation into the non-cleft nostril — residual nasal deformity reflects incomplete nasolabial muscle-ring reconstruction.

Pearls

- ✓ LAHSHAL = Lip, Alveolus, Hard palate, Soft palate, recorded for right and left sides.
- ✓ Incisive foramen divides primary (lip, alveolus) from secondary (hard, soft) palate.
- ✓ Distribution: CL 15%, CLP 45%, CP 40%; UCLP left-sided in 60%.
- ✓ Lip at 3–6 months, palate at 6–12 months (IVVP — levator sling), ABG at canine root $\frac{1}{2}$ – $\frac{2}{3}$ formed, orthognathics after growth.
- ✓ Muscle repair matters more than the skin incision.
- ✓ Exam trap: isolated cleft palate is missed on the routine anomaly scan and is the type most often syndromic.

Sources

- BL 28e, ch. 50 “Developmental abnormalities of the face, mouth and jaws:”, §“Cleft lip and palate: classification; aetiology; embryology and pathogenesis; clinical anatomy”, pp. 740–743; Fig. 50.2, p. 741; Fig. 50.4, p. 743.
- BL 28e, ch. 50 “Developmental abnormalities of the face, mouth and jaws:”, §“The cleft multidisciplinary team and primary management; principles of cleft surgery”, pp. 744–745.
- BL 28e, ch. 50 “Developmental abnormalities of the face, mouth and jaws:”, §“Early and late childhood care; alveolar bone grafting; secondary/revision surgery”, pp. 746–747.
- BL 28e, ch. 50 “Developmental abnormalities of the face, mouth and jaws:”, §“Management of craniofacial anomalies: neonatal management”, p. 739.

Asked in: Paper IV · Oct 2024 · Q10(a) (5 marks)

“Role of microvascular surgery in today's surgical practice.”

Principle

- **Microsurgery:** a subspecialty using **magnification, precision tools and surgical techniques** to anastomose small blood vessels and coapt nerves; typical suture diameter **0.01–0.03 mm**.
- **Free flap:** tissue with its vascular pedicle is detached and transferred to a distant site; its vessels are joined under the microscope to vessels near the recipient site.
- Tissue can be transplanted to **every region of the body** — no longer limited to grafts that must revascularise from the wound bed or to pedicled flaps limited by size, length or reach; needs only an intact source-vessel course and a suitable **recipient vessel**.
- **Chimeric flaps** (several flaps on one source pedicle, e.g. lateral circumflex femoral or subscapular system) reconstruct complex composite defects.

Role in current practice

Table 14.6.1. Applications of microvascular surgery

Field	Role	Examples
Oncological reconstruction — head and neck	Superior functional and aesthetic results; restores speech, swallowing and airway; bone for 3-D defects	Anterolateral thigh, radial forearm; free fibula for mandible; fibula, scapula, iliac crest
Breast reconstruction	Autologous breast after mastectomy — natural look and feel, higher satisfaction	DIEP (deep inferior epigastric artery), TRAM, gluteal and thigh perforator flaps
Trauma and osteomyelitis	Limb salvage instead of amputation; wounds of any size covered; vascularised bone restores continuity	Gracilis or latissimus dorsi muscle flaps; fibula strut after excising osteomyelitic tibia
Functional reconstruction	Free functional muscle reanimation	Face (facial palsy); upper limb (brachial plexus palsy)
Replantation and hand	Replant amputated digits and limbs; reconstruct missing fingers	Free vascularised, sensate toe-to-hand transfer
Supermicrosurgery (0.3–0.8 mm vessels, single fascicles)	Fingertip reconstruction (formerly amputation); physiological lymphoedema surgery	Lymphaticovenous anastomosis; prophylactic lymphovenous bypass (LYMPHA); vascularised lymph node transfer
Vascularised composite allotransplantation	Transplant of composite tissue	Hand and face transplants

Source: BL 28e pp. 699–701; SAB 22e pp. 866, 869, 872–873

- Reconstruction makes **otherwise unresectable tumours resectable;** in the head and neck and extremities many tumours cannot be resected without free tissue transfer.
- In a field destined for **adjuvant radiotherapy**, or already irradiated (friable, prone to vasospasm and dehiscence), a free flap brings well-vascularised tissue and resists radiation better than a skin graft.
- Prophylactic lymphovenous bypass after axillary dissection lowers lymphoedema from **30–40% to 4–13%.**

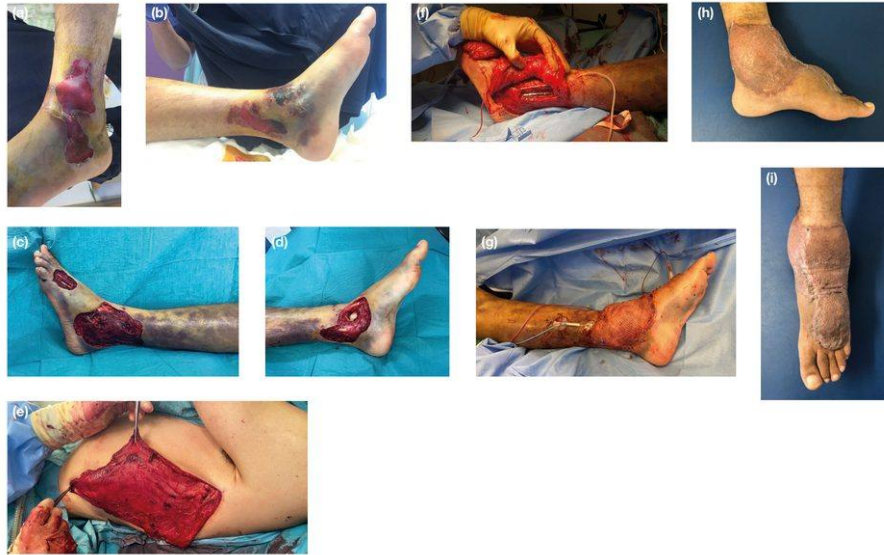


Figure 14.6.1 — Limb salvage: degloving injury of foot and ankle (a, b); after debridement, exposed tendons and joint (c, d); latissimus dorsi and serratus anterior harvested as two free flaps (e); meshed split-skin graft over the muscle flaps (f, g); normal ambulation at 6 months (h, i).

BL 28e Fig. 47.26, p. 698

Technique and post-operative monitoring

- Anastomoses **hand-sewn** with microinstruments under the operating microscope; **mechanical couplers** increasingly used for veins (faster, technically easier).
- Patient kept “**wet, warm and comfortable**” (often in a high-dependency unit): hyperdynamic circulation, warming device, good analgesia to limit catecholamines.
- Flap checked for **colour, warmth, turgor, capillary refill**, pinprick bleeding, hand-held or implantable **Doppler**; most valuable in the first **48 hours**.
- ~**5%** of free flaps need re-exploration; **>60%** of these are salvaged — the earlier, the better; two-thirds of failures are **venous**, one-third arterial.
- Venously congested flaps without a correctable obstruction: **leech therapy** (hirudin) with prophylactic quinolone and haemoglobin monitoring.

Outcomes and limitations

- Free-flap survival **>95%** in elective cases (DIEP/TRAM breast); slightly lower in polytrauma and head and neck reconstruction.
- **Contraindicated** in active smokers and patients with blood clotting disorders; donor-site morbidity higher with BMI **>35**.
- Drawbacks: **donor-site morbidity**, longer operating and recovery time.
- Technically feasible is not always best: a below-knee amputation may give better quality of life than free-flap salvage of a complex open tibiofibular fracture.
- Expanding role: **robotic** assistance (tremor elimination, 3-D view) in head and neck reconstruction; growing vascularised composite allotransplantation.

Pearls

- ✓ Microsurgery = anastomosis of vessels and nerves under magnification; free flap needs a recipient vessel.
- ✓ Workhorse free flaps: ALT and radial forearm (head and neck), DIEP (breast), fibula (mandible).
- ✓ Free flap = limb salvage instead of amputation; vascularised tissue for irradiated beds.
- ✓ Monitoring: first 48 h; failure mostly venous; explore early (>60% salvage).
- ✓ Supermicrosurgery: lymphaticovenous anastomosis for lymphoedema.

Sources

- BL 28e, ch. 3 “Wound healing and tissue repair”, §“Skin grafts and flaps: free flap”, p. 31.
- BL 28e, ch. 47 “Plastic and reconstructive surgery”, §“Microsurgery; flap monitoring; leech therapy; future directions”, pp. 698–701; Fig. 47.26, p. 698.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Goals of reconstruction”, p. 866.
- SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, §“Timing of reconstruction; radiation; head and neck”, pp. 868–869.

— SAB 22e, ch. 69 “Plastic Surgical Considerations in Cancer Surgery”, § “Breast reconstruction; lymphatic surgery”, pp. 872–873.

Skin & Soft Tissue Lesions

15.1a Premalignant lesions of the skin

2x

Asked in: Paper II · Aug 2021 · Q7(b) (6 marks) ; Paper II · Jun 2022 · Q5(b) (5 marks)

“Briefly discuss the premalignant lesions of the skin.”

“Premalignant lesions of skin.”

Concept

- **Premalignant skin lesions** carry an increased risk of becoming skin cancer; they fall into precursors of **squamous cell carcinoma (SCC)**, precursors of **melanoma**, and **intraepidermal adenocarcinoma** (extramammary Paget's disease).
- Current thinking places actinic keratosis, cutaneous horn and keratoacanthoma on a **continuum of squamous lesions** (like cervical intraepithelial neoplasia), some of which regress.
- **Basal cell carcinoma has no precursor lesion.**

Table 15.1a.1. Premalignant lesions of the skin — what each leads to

Lesion	Leads to	Malignant potential
Actinic (solar) keratosis	SCC	Up to 20% (BL); 0.01–0.6% per year, up to 2.5% over 4 years (SAB)
Cutaneous (keratin) horn	SCC	Underlying SCC in 10%
Keratoacanthoma	“Keratoacanthoma-like SCC”	Regarded as a self-healing SCC
Bowen's disease / erythroplasia of Queyrat	Invasive SCC	Already SCC in situ
Chronic scars, sinuses, ulcers	SCC (Marjolin's ulcer)	Higher metastatic potential than ordinary SCC
Atypical (dysplastic) naevus	Melanoma	>5 lesions: relative risk ×6; FAMMM syndrome: ~100%
Giant congenital pigmented naevus	Melanoma (also sarcoma)	3–5% lifetime; 1 in 3 childhood melanomas
Lentigo maligna	Lentigo maligna melanoma	Melanoma in situ; risk of vertical growth phase
Extramammary Paget's disease	Invasive adenocarcinoma	25% have underlying adenocarcinoma; up to 20% invasive at excision

Source: BL 28e pp. 650–655, 657–658; SAB 22e pp. 723–724, 745, 749

1. Precursors of squamous cell carcinoma

- **Actinic (solar) keratosis (AK):**
 - Area of **permanent sun damage**: dyskeratosis, partial-thickness cellular atypia, subepidermal inflammation, **intact basement membrane**.
 - Scaly, uneven, <1 cm, on an erythematous base in chronically sun-damaged skin; “waxes and wanes” between macule and papule; most improve with moisturisation.
 - Diagnosis clinical and histological (shares features with SCC in situ).
 - **Treatment: cryotherapy**; topical 5-fluorouracil, curettage and electrodesiccation, CO₂ laser, dermabrasion, chemical peel; **biopsy** if raised or recurrent after topical therapy.
- **Cutaneous horn**: an AK with a keratinous surface taller than its base diameter — excise, as **10%** hide an SCC.
- **Keratoacanthoma**:
 - Rapidly growing nodule, symmetrical around a **central keratin-filled crater**; face or limbs of sun-damaged 50–70-year-olds; men 2:1; linked to HPV, smoking and chemical carcinogens.
 - Now considered a **self-healing SCC**; **excise** rather than observe — the differential is anaplastic SCC, and the scar is better than after spontaneous resolution.
- **Bowen's disease** (SCC in situ):

- Full-thickness dysplasia, often within a hypertrophic AK; slowly enlarging **erythematous scaly plaque** anywhere on the mucocutaneous surface; on the glans penis = **erythroplasia of Queyrat**.
- **Treatment:** topical 5-FU or imiquimod; excision with a **4-mm margin**, or Mohs' surgery for large or recurrent lesions.
- **Arsenical keratoses:** after inorganic arsenic exposure; predilection for the palm.
- **Chronic wounds and scars** (burns, osteomyelitis sinuses, chronic ulcers): long-standing inflammation and irritation; **pseudo-epitheliomatous hyperplasia** on biopsy is premalignant and demands further biopsies; biopsy any atypical or broken-down healed wound.

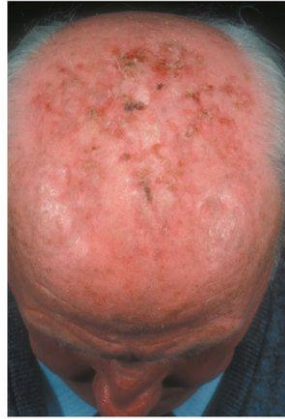


Figure 15.1a.1 — Actinic keratosis of the sun-damaged scalp.

BL 28e Fig. 45.31, p. 653

2. Precursors of melanoma

- Only **10–20%** of melanomas arise in a pre-existing naevus (SAB: up to 40%); most likely are atypical naevi, atypical junctional lentiginous (facial) naevi and giant congenital naevi.
- **Atypical (dysplastic) naevus:**
 - Clinically “atypical” = three of variegated pigmentation, ill-defined borders, irregular surface, size >5 mm; “dysplastic” is a histological term (irregular basal melanocyte proliferation).
 - Sporadic or familial — **FAMMM syndrome** (melanoma in relatives, often >100 naevi, *CDKN2A*): 6-monthly lifelong screening.
 - Mild dysplasia observed; moderate or severe dysplasia excised with negative margins (wide excision unnecessary); a **marker of melanoma risk** elsewhere.
- **Giant congenital pigmented naevus** (>20 cm):
 - Hamartoma of naevus cells in all skin layers into fat and muscle, dermatomal distribution.
 - Investigate for **neurocutaneous melanosis**; consider removal for aesthetic and oncological reasons; regular dermatological review.
- **Lentigo maligna** (melanoma in situ, Hutchinson's melanotic freckle): excise completely, **5-mm margin**, because of the risk of vertical growth.
- Small or medium congenital naevi are low risk; **Spitz naevus** has little or no risk.
- Features of malignant change in a naevus: change in size, shape, colour, thickness (nodularity, ulceration), satellite lesions; itching or discharge are late.



Figure 15.1a.2 — Bowen's disease — squamous cell carcinoma in situ.

BL 28e Fig. 45.34, p. 654

3. Extramammary Paget's disease

- **Intraepidermal adenocarcinoma** of apocrine-rich skin (axilla, perianal region, vulva, scrotum); presents as eczema or intertrigo that fails to respond.
- Diagnose by **biopsy**; survey the GI and GU tracts endoscopically for internal cancer; excise to histologically clear margins; local recurrence common.

4. Predisposing conditions

- **Xeroderma pigmentosum**: defective nucleotide excision repair, extreme photosensitivity, **2000-fold** risk of skin cancer.
- **Albinism**, fair skin (Fitzpatrick I–II) and **immunosuppression** raise the risk of SCC and melanoma.

Pearls

- ✓ BCC has no precursor; SCC precursors: AK, Bowen's, chronic scars; melanoma precursors: dysplastic and giant congenital naevi, lentigo maligna.
- ✓ Bowen's disease = SCC in situ; on the glans = erythroplasia of Queyrat.
- ✓ Cutaneous horn: 10% have an underlying SCC.
- ✓ Keratoacanthoma is now considered a self-healing SCC — excise it.
- ✓ Giant congenital naevus: 3–5% lifetime melanoma risk; screen for neurocutaneous melanosis.
- ✓ Extramammary Paget's: look for an underlying or internal adenocarcinoma.

Sources

- BL 28e, ch. 45 "Skin and subcutaneous tissue", §"Skin tumours: premalignant lesions", pp. 650–651.
- BL 28e, ch. 45 "Skin and subcutaneous tissue", §"Cutaneous squamous cell carcinoma: actinic keratosis, keratoacanthoma, Bowen's disease", pp. 652–654; Fig. 45.31, p. 653; Fig. 45.34, p. 654.
- BL 28e, ch. 45 "Skin and subcutaneous tissue", §"Cutaneous malignant melanoma: macroscopic; local treatment", pp. 656–658.
- SAB 22e, ch. 63 "Melanoma and Cutaneous Malignancies", §"Melanoma: precursor lesions", pp. 723–724.
- SAB 22e, ch. 63 "Melanoma and Cutaneous Malignancies", §"Squamous cell carcinoma: presentation; treatment of actinic keratosis", pp. 745–746.
- SAB 22e, ch. 63 "Melanoma and Cutaneous Malignancies", §"Extramammary Paget disease", p. 749.
- SAB 22e, ch. 45 "Management of Chronic Wounds", §"Chronic wounds: malignant transformation", p. 511.
- SAB 22e, ch. 119 "Hand Surgery", §"Hand surgery: malignant skin tumours", p. 2028.
- SAB 22e, ch. 60 "Tumor Biology and Tumor Markers", §"Tumour biology: DNA repair and xeroderma pigmentosum", p. 721.e57.

Asked in: Paper II · Oct 2023 · Q3 (4+6 marks)

“Enumerate pre-malignant and malignant tumors of the skin. Discuss the etiopathogenesis, investigations and management of any two.”

1. Premalignant and malignant tumours of the skin

Table 15.1b.1. Enumeration of premalignant and malignant skin tumours

Group	Premalignant	Malignant
Keratinocyte	Actinic (solar) keratosis; cutaneous horn; keratoacanthoma (self-healing SCC); Bowen's disease / erythroplasia of Queyrat (SCC in situ); arsenical keratosis; chronic scars and sinuses	Basal cell carcinoma; squamous cell carcinoma (incl. Marjolin's ulcer)
Melanocytic	Atypical (dysplastic) naevus, FAMMM syndrome; giant congenital pigmented naevus; lentigo maligna (melanoma in situ)	Malignant melanoma — superficial spreading, nodular, lentigo maligna, acral lentiginous; amelanotic, desmoplastic
Adnexal / glandular	Extramammary Paget's disease (intraepidermal adenocarcinoma)	Invasive extramammary Paget's disease
Neuroendocrine	—	Merkel cell carcinoma
Dermal / vascular	—	Dermatofibrosarcoma protuberans; cutaneous angiosarcoma; Kaposi sarcoma
Genetic predisposition	Xeroderma pigmentosum; albinism; genetic skin cancer syndromes	—

Source: BL 28e pp. 650–659; SAB 22e pp. 723–724, 744–749

2. Basal cell carcinoma (BCC)

Aetiopathogenesis

- Slow-growing, **locally invasive** malignant tumour of pluripotential epithelial cells of the **basal epidermis and hair follicles** — a tumour of pilosebaceous skin; commonest skin cancer (~80% of non-melanoma skin cancer).
- Risk factors:
 - **Ultraviolet radiation** — strongest; intense, intermittent exposure; 95% between 40 and 80 years; incidence rises towards the equator, yet 33% arise on non-exposed skin.
 - Arsenicals, coal tar, aromatic hydrocarbons, ionising radiation; **genetic syndromes** (e.g. Gorlin — *PTCH*); immunosuppression (risk ×10 after transplantation).
 - White skin almost exclusively; men more than women.
- **No precursor lesion**; development is proportional to the initial carcinogen dose, not its duration.
- **Hedgehog pathway** mutated in up to 90%: inactivating *PTCH* or activating *SMO* mutations → unrestrained growth signalling.
- **Histology**: ovoid cells in nests with a **peripheral palisade**; only the outer layer divides (slow growth; incompletely excised lesions are more aggressive); **morphoeic BCC** makes type 4 collagenase and spreads rapidly.
- Types: localised (nodular, nodulocystic — **90%**, cystic, pigmented) and generalised (superficial, infiltrative/morphoeic); metastasis **extremely rare**.



Figure 15.1b.1 — (a) Nodulocystic BCC — pearly surface with telangiectasia. (b) Ulcerating BCC on the lower eyelid. (c) Recurrent morpheic BCC.

BL 28e Fig. 45.30, p. 652

Investigations

- **Clinical examination:** pearly, waxy nodule with telangiectasia and rolled edge, central ulceration; pigmented BCC mimics melanoma.
- **Biopsy** — excisional (small lesions), incisional/punch (≥ 4 mm, large lesions) or shave; topical therapy only for **biopsy-proven** superficial tumours.
 - Report subtype, margins and perineural invasion; specimen orientated and marked.
- **Risk assessment** — high-risk BCC: **>2 cm**; at sites where invasion reaches the cranium (**near the eye, nose and ear**); **recurrent**; arising with **immunosuppression**; **micronodular or infiltrating** histology; SAB adds poorly defined borders, prior radiotherapy and perineural invasion.
- **No sentinel node biopsy** — nodal metastases exceedingly rare.

Management

- **Surgical excision:** margins marked under loupe magnification, **2–15 mm** by variant (SAB: gross 5 mm usually adequate).
- Ill-defined margins or tissue at a premium (nose, eyelid): **two-stage excision** with delayed reconstruction, or **Mohs' micrographic surgery**.
- **Radiotherapy** for the elderly or infirm — recurrence similar to surgery, but risk of new malignancy after 1–2 decades; adjuvant RT for high-risk lesions.
- Superficial BCC: **topical 5-fluorouracil or imiquimod**, cryosurgery, photodynamic therapy, curettage and electrodesiccation.
- Incomplete excision: recurrence **67%** if grossly involved, **33%** within 2 years if microscopically involved/close → re-excise.
- Locally advanced/metastatic: **hedgehog inhibitors** vismodegib (response 30% locally advanced, 43% metastatic) and sonidegib; checkpoint inhibitors after failure.
- **Follow-up** only for high-risk sites, sun-damaged skin, syndromes or incomplete excision.

3. Cutaneous squamous cell carcinoma (SCC)

Aetiopathogenesis

- Malignant tumour of **keratinising cells** of the epidermis or its appendages, arising from the stratum basale; expresses cytokeratins 1 and 10; one SCC for every four BCCs.
- Risk factors:

- **Cumulative sun exposure** (UVB), fair skin, age, proximity to the equator, men.
- **Chronic inflammation**: burn scars, sinuses, osteomyelitis, chronic ulcers, vaccination scars → **Marjolin's ulcer**.
- **Immunosuppression**: transplant recipients ×65 risk; more aggressive tumours.
- Ionising radiation, arsenicals, tar, hydrocarbons, **HPV 5 and 16**, smoking (doubles risk), xeroderma pigmentosum, albinism, BRAF inhibitors.
- **Mechanism**: UVB damage to dividing basal keratinocytes; **p53 silenced in >90%** → no cell-cycle arrest or apoptosis → dysplasia (actinic keratosis) → SCC in situ (Bowen's) → invasive SCC.
- Nodular, verrucous or ulcerating; ulcer with **everted edge** and indurated surround.



Figure 15.1b.2 — SCC: (a) face; (b) recurrence in a skin-grafted scalp; (c) dorsum of hand in a renal transplant recipient; (d) lip of an outdoor-working smoker.

BL 28e Fig. 45.36, p. 655

Investigations

- **Biopsy** of the lesion (and of any atypical chronic wound) — distinguishes SCC from keratoacanthoma.
- **Histopathology report**: Broders' grade, depth of invasion, **perineural or lymphovascular invasion**, deep and peripheral margins.
- **Regional lymph node examination; SLN biopsy** may have a role in high-risk lesions (occult nodes in 7–20%).
- **TNM staging**: T1 <2 cm, T2 >2 cm, T3 facial bone, T4 muscle, skull base or other bone.
- **Risk stratification** from the work-up (worse prognosis):
 - **Depth**: <2 mm metastasis highly unlikely; >6 mm — 15% have metastasised.
 - **Size** >2 cm; high **Broders' grade**; perineural or lymphovascular invasion.
 - **Site**: lips and ears recur more; extremities fare worse than trunk.
 - **Aetiology**: scar, sinus, ulcer or irradiated-skin SCC; **immunosuppression**; poorly defined borders, recurrence, rapid growth, neurological symptoms (SAB high-risk features).

Management

- **Surgical excision** — the only means of histology and clearance: **4-mm** margin for SCC <2 cm, **1-cm** margin for >2 cm (SAB: 5 mm, 10 mm for high-risk).
- **Mohs' surgery** for cosmetically sensitive, recurrent or high-risk tumours.
- Must address **lymphatic and in-transit spread**; after a positive SLN, completion lymphadenectomy ± radiotherapy (indications less defined than in melanoma).
- **Adjuvant radiotherapy** for extensive perineural or large-nerve involvement.
- Small superficial lesions: cryotherapy (local control >90%), curettage (<2 cm), photodynamic therapy, imiquimod.
- Advanced/metastatic: **cemiplimab, pembrolizumab** (response 30–50%); EGFR agents; platinum chemotherapy.
- **Follow-up 5 years** — 95% of recurrences and nodal metastases occur within 5 years; metastasis 2% (low-risk) to 30% (high-risk).

How to use these notes

- **One answer, many questions:** repeats and near-repeats are merged into one answer; its "Asked in" line lists every paper and year, and the badge (1×, 2×, 3×, 4×+) shows how often it was asked.
- **Red box — NOT COVERED IN SOURCE:** the point is not in the three textbooks; the box names the searches made and the answer gives the closest related book material.
- **Amber box — Current guideline:** the only material not taken from the textbooks; it names the body, document and year, and states that the textbook predates it.
- **Figures** are real figures cropped from the books, with the book figure number and printed page; **schematics** are simplified exam diagrams drawn for these notes and are labelled as such.
- **Sources** at the end of each answer give book, chapter, section and printed pages.

A • Parts in this volume

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Oesophagus

25.1a

Achalasia cardia — pathophysiology, clinical features, pre-operative work-up and treatment

4x+

Asked in: Paper I · Jun 2020 · Q2(b) (7 marks) ; Paper III · Aug 2021 · Q10(b) (5 marks) ; Paper III · Dec 2021 · Q5(b) (5 marks) ; Paper III · May 2024 · Q10(b) (5 marks)

“Describe pathophysiology, clinical features, diagnosis & treatment of achalasia cardia.”

“Achalasia cardia.”

“Discuss management of Achalasia Cardia.”

“Discuss the preoperative workup of a patient with achalasia cardia.”

- **Achalasia** (“failure to relax”): failure of lower oesophageal sphincter (LOS) relaxation with absent peristalsis — the commonest oesophageal motility disorder; usually presents at 30–60 years.

Pathophysiology

- **Loss of inhibitory ganglion cells in the myenteric (Auerbach’s) plexus**, possibly a virus-induced autoimmune process; histology shows fewer ganglion cells with chronic inflammation.
- Excitatory/inhibitory mismatch → **LOS fails to relax and peristalsis is absent**.
- Oesophagus empties only by the hydrostatic pressure of its contents → stasis, progressive dilatation and tortuosity (**megaoesophagus**, “sigmoid” oesophagus).
- Retention oesophagitis from fermenting food → **increased risk of oesophageal carcinoma**.
- Look-alikes: **Chagas’ disease** (*Trypanosoma cruzi*); Allgrove syndrome (achalasia, alacrimia, adrenal insufficiency).

Clinical features

- **Dysphagia to both solids and liquids**, regurgitation (often of food eaten earlier), heartburn — frequently mistaken for GORD.
- Chest pain/odynophagia early; weight loss variable; halitosis from fermenting residue.
- Aspiration — nocturnal cough, pneumonia — with a dilated, food-filled oesophagus.
- Severity and response to treatment graded by the **Eckardt score** (0–3 each for dysphagia, regurgitation, retrosternal pain, weight loss).

Diagnosis and pre-operative work-up

Table 25.1a.1. Investigations in achalasia

Test	Finding	Purpose
Chest X-ray	Dilated oesophagus, air–fluid level; absent gastric air bubble	Suggests advanced disease
Barium swallow / timed barium oesophagram	Hold-up, tapering distal narrowing — “bird’s beak / rat’s tail” ; sigmoid oesophagus late	Anatomy, severity (column height at set times)
Upper GI endoscopy (mandatory)	Frothy saliva, food residue, tight OGJ that admits the scope with gentle pressure; normal in 30–40%	Exclude pseudo-achalasia (cardia cancer), oesophagitis
High-resolution manometry (gold standard)	Raised integrated relaxation pressure + 100% failed peristalsis	Confirms diagnosis; Chicago type I/II/III guides treatment
24-h pH monitoring	Distinguishes true reflux from stasis/fermentation	When heartburn is the complaint
EUS / CT chest-abdomen	Mass at OGJ	If pseudo-achalasia is suspected
FLIP (impedance planimetry)	Distensibility of the OGJ	Corroborates HRM; assesses myotomy

Source: BL 28e pp. 1108–1109, 1121–1122; SAB 22e pp. 1115, 1117

- **Chicago classification (HRM):**

- **Type I** — 100% failed peristalsis without pan-oesophageal pressurisation.
- **Type II** — 100% failed peristalsis with pan-oesophageal pressurisation in $\geq 20\%$ of swallows (best response to pneumatic dilatation).
- **Type III** — $\geq 20\%$ premature (spastic) contractions.
- Work-up before surgery also includes fitness assessment and the baseline Eckardt score.

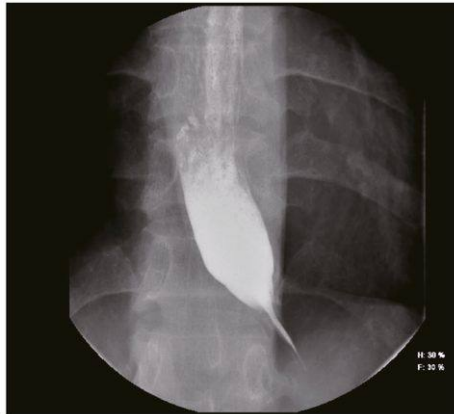


Figure 25.1a.1 — Barium swallow: dilated oesophagus tapering to the typical “rat’s tail” at the oesophagogastric junction.
 BL 28e Fig. 66.25, p. 1123

Treatment (palliative — aims to reduce LOS resistance)

- **Medical:** calcium-channel blockers, nitrates, phosphodiesterase-5 inhibitors lower LOS pressure.
 - Limited, short-lived benefit; headache, oedema, hypotension — only for patients unfit for definitive treatment.
- **Endoscopic botulinum toxin:** blocks acetylcholine release; 100 units in four quadrants about 1 cm above the GEJ.
 - Relief about 70% at 3 months, ~40% at 1 year; repeated injections scar the planes and make later myotomy harder — reserve for the elderly/unfit or when the diagnosis is in doubt.
- **Pneumatic dilatation (PD):** non-compliant balloon tears the LOS fibres; graded **30 → 35 → 40 mm**.
 - Best response: age >45 years, female, undilated oesophagus, type II.
 - Perforation averages ~1.9% (<0.5% with a 30-mm balloon); about one-quarter need repeat dilatation; surgical back-up essential.
- **Laparoscopic Heller myotomy (LHM) + partial fundoplication** — standard operation.
 - Myotomy ≥ 6 cm on the oesophagus and 2–3 cm onto the gastric cardia (SAB: ~8.5 cm total); edges separated over $\sim 140^\circ$.
 - Add **Dor (anterior)** or **Toupet (posterior)** wrap: reflux falls from up to 40–48% to ~9%.
 - **Nissen (360°) is contraindicated** — outflow resistance against an aperistaltic oesophagus causes dysphagia.
 - More effective and durable than a single PD; 5-year success 84–92%.
- **POEM (peroral endoscopic myotomy):** mucosal entry → submucosal tunnel → myotomy of circular fibres ≥ 6 cm in the oesophagus and 2–3 cm into the cardia → clip closure.
 - Can be extended proximally — **preferred for type III**.
 - No antireflux wrap: pathological reflux ~50–57%.
- **Oesophagectomy:** last resort for end-stage, sigmoid megaesophagus not helped by other methods (aspiration risk).
- **Choice:** types I and II — laparoscopic Heller myotomy with partial fundoplication or POEM; **type III — POEM** (longer myotomy).
- Heller myotomy is superior to a single pneumatic dilatation in efficacy and durability.
- **Recurrent dysphagia:** re-investigate (barium, endoscopy, manometry, pH); PD after Heller, POEM after failed Heller, oesophagectomy last.
- **Follow-up:** Eckardt score; ideally HRM, barium, endoscopy and pH study; lifelong diet advice (body peristalsis remains abnormal).

Pearls

- ✓ Dysphagia to liquids as well as solids from the start → think achalasia.
- ✓ HRM is the gold standard; endoscopy is mandatory to exclude pseudo-achalasia.
- ✓ Barium: bird's beak / rat's tail.
- ✓ Operation of choice: laparoscopic Heller myotomy + Dor/Toupet; never a Nissen.
- ✓ Type III → POEM.
- ✓ Long-standing achalasia carries a raised risk of oesophageal carcinoma.

Sources

- BL 28e, ch. 66 "The oesophagus", §"Investigation: plain radiography", pp. 1108–1109; Fig. 66.4, p. 1109.
- BL 28e, ch. 66 "The oesophagus", §"Motility disorders: achalasia — pathology, clinical features, diagnosis", pp. 1121–1122; Fig. 66.23, Table 66.1, p. 1122.
- BL 28e, ch. 66 "The oesophagus", §"Achalasia: treatment; summary box 66.5", pp. 1123–1125; Fig. 66.25, p. 1123.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", §"Achalasia: diagnosis, treatment, recurrent symptoms", pp. 1115–1117.

25.1b

Physiology of oesophageal motility

1×

Asked in: Paper I · Jun 2020 · Q2(a) (3 marks)

"Outline the physiology of oesophageal motility."

Functions

- Propels food from pharynx to stomach **even against gravity and whatever the bolus consistency**, while protecting the **airway from aspiration** and itself from **gastro-oesophageal reflux**.
- No digestive, absorptive, endocrine or exocrine function.

Muscle and nerve control

- **Muscularis propria**: inner circular and outer longitudinal layers; **upper third striated, middle third mixed, lower third smooth** muscle.
- **Dual innervation**: extrinsic sympathetic and parasympathetic (vagus) nerves centrally; **enteric nervous system** peripherally.
- Excitatory motor neurones contain **acetylcholine**; inhibitory neurones of the myenteric plexus contain **nitric oxide and VIP**, needed for smooth-muscle peristalsis and LOS relaxation.

Sequence of a swallow

- **UOS** (cricopharyngeus + inferior pharyngeal constrictor, striated) relaxes in coordination with pharyngeal contraction — the start of a swallow.
- **Primary peristalsis**: triggered by the **swallow centre in the brain**; the bolus activates excitatory motor neurones sequentially along an aboral progression of muscle segments.
- **Deglutitive LOS relaxation** occurs reflexly with each swallow, allowing oesophageal emptying.
- Delayed refluxate clearance from defective motility increases oesophageal acid exposure.

Lower oesophageal sphincter and antireflux barrier

- **LOS**: 3–4 cm of smooth muscle forming a **high-pressure zone** — defined physiologically, not anatomically.
- Tone from intrinsic muscle plus the **crural diaphragm**, against negative intrathoracic and positive intra-abdominal pressure.
- Other components: **intra-abdominal oesophagus**, acute **angle of His**, Gubaroff mucosal valve.
- **Transient LOS relaxations (TLOSrs)**: physiological relaxation on fundal stretch after a meal, venting swallowed air; excess TLOSrs → GORD.

Pearls

- ✓ Upper third striated, lower third smooth muscle — scleroderma hits the lower oesophagus.
- ✓ Primary peristalsis is swallow-centre driven; LOS relaxes reflexly with every swallow.
- ✓ Loss of NO/VIP inhibitory neurones = achalasia (failed LOS relaxation, aperistalsis).
- ✓ HRM is the test that measures all of these functions.

Sources

— BL 28e, ch. 66 “The oesophagus”, § “Applied surgical anatomy and physiology”, pp. 1106–1107.

— BL 28e, ch. 66 “The oesophagus”, § “Oesophageal manometry”, p. 1110.

— BL 28e, ch. 66 “The oesophagus”, § “Gastro-oesophageal reflux disease: aetiology”, pp. 1111–1112.

— BL 28e, ch. 66 “The oesophagus”, § “Achalasia: pathology and aetiology”, p. 1121.

— SAB 22e, ch. 83 “Benign Esophageal Disorders”, § “Esophageal physiology: swallowing; antireflux mechanism”, pp. 1108–1109.

— SAB 22e, ch. 83 “Benign Esophageal Disorders”, § “Esophageal manometry”, p. 1110.

— SAB 22e, ch. 83 “Benign Esophageal Disorders”, § “Achalasia: pathophysiology”, p. 1114.

25.1c

High-resolution oesophageal manometry (HRM)

1x

Asked in: Paper I · May 2024 · Q9(a) (5 marks)

“High resolution oesophageal manometry.”

Definition and principle

- **HRM:** transnasal **solid-state catheter with 36 circumferential pressure sensors spaced 1 cm apart**, recording pressure from pharynx to stomach simultaneously.
- Data shown as **Clouse plots** (colour isobaric contour plots) — replaced conventional water-perfused catheters (8 channels, station pull-through).
- With the **Chicago classification (v4.0)** it is the standard worldwide and the **gold standard for diagnosing oesophageal motility disorders**.

Technique

- Fasting; **no sedation** (patient cooperation needed; drugs alter motility); motility-altering drugs stopped beforehand.
- Catheter tip in the stomach.
- **Static phase:** basal pressures and length of the sphincters.
- **Dynamic phase: 10 wet (water) swallows** in the primary position → sphincter relaxation and peristalsis.
- Step 2 (if needed): swallows in a secondary position, **multiple rapid swallows (MRS)**, **rapid drink challenge (RDC)**; optional solid swallows, meal challenge or pharmacological provocation.

Metrics measured

Table 25.1c.1. HRM metrics

Metric	Definition	Meaning
LOS basal pressure, length	Resting pressure; total and intra-abdominal length	Hypotensive or normal LOS
Integrated relaxation pressure (IRP)	Lowest 4 s of OGJ pressure in a 10-s window after a swallow (not necessarily consecutive)	Raised = OGJ outflow disorder (non-relaxing LOS)
Distal contractile integral (DCI)	Pressure (mmHg) × time (s) × length (cm) of the distal oesophageal contraction	Vigour: failed, weak, normal, hypercontractile
Distal latency (DL)	Swallow onset (UOS relaxation) to the contractile deceleration point (CDP)	Coordination: normal vs premature
LOS–crus separation	Disjunction of LOS and diaphragm pressure (OGJ types I–III)	Manometric hiatus hernia

Source: BL 28e p. 1111; SAB 22e pp. 1110–1111

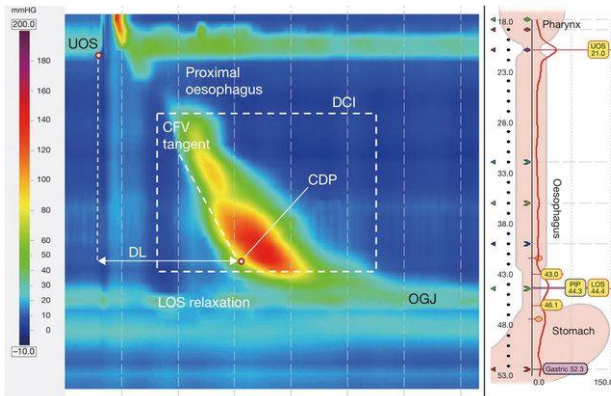


Figure 25.1c.1 — HRM of a typical swallow: UOS break marks the swallow; DCI box, distal latency (DL) to the contractile deceleration point (CDP); reflex LOS relaxation.

BL 28e Fig. 66.9, p. 1111

Chicago classification 4.0 (hierarchical)

- **Disorders of OGJ outflow** (raised median IRP):
 - **Achalasia type I** — 100% failed peristalsis without pressurisation.
 - **Type II** — 100% failed peristalsis with pan-oesophageal pressurisation in $\geq 20\%$ of swallows.
 - **Type III** — $\geq 20\%$ premature (spastic) contractions.
 - **OGJ outflow obstruction (OGJOO)** — raised IRP with peristalsis; needs timed barium oesophagram or FLIP support.
- **Disorders of peristalsis** (normal IRP):
 - **Absent contractility** — 100% failed peristalsis.
 - **Distal oesophageal spasm** — $\geq 20\%$ premature contractions.
 - **Hypercontractile oesophagus** (jackhammer) — $\geq 20\%$ hypercontractile swallows.
 - **Ineffective oesophageal motility** — $>70\%$ ineffective or $\geq 50\%$ failed swallows.
- A manometric diagnosis must be associated with symptoms or other supporting tests to be clinically relevant.

Indications

- **Dysphagia** with normal endoscopy; diagnosis of primary motility disorders and **achalasia subtype** (guides POEM vs Heller myotomy).
- **Locating the LOS** to position the pH catheter (5 cm above its upper border).
- **Before antireflux surgery** (baseline body motility) and oesophageal operations at the OGJ; oesophageal involvement in **systemic disease** (scleroderma).
- Symptomatic patients **after oesophageal surgery** (completeness of myotomy).

Limitations

- **Cannot diagnose GORD** — a normal LOS does not exclude it; an abnormal LOS is not synonymous with it (pH monitoring needed).
- Invasive; gives no information on bolus retention or reflux.
- UOS assessment is limited and excluded from the current classification.
- Manometric findings alone are not a diagnosis — symptoms or supporting tests are required.

Adjuncts

- **FLIP** (functional lumen imaging planimetry): impedance balloon measuring OGJ **distensibility index** (cross-sectional area $\div$ intra-balloon pressure); corroborates HRM, guides completeness of myotomy, screens for need of formal HRM.
- **Timed barium oesophagram** and FLIP support an OGJOO diagnosis; **24-h pH-impedance** monitoring is placed by HRM-defined LOS position.

Pearls

- ✓ 36 sensors, 1 cm apart; 10 wet swallows.
- ✓ IRP = outflow; DCI = vigour; DL = coordination.
- ✓ Raised IRP + 100% failed peristalsis = achalasia; subtype by pressurisation/spasm.
- ✓ HRM is the gold standard for motility disorders but cannot diagnose GORD.
- ✓ Type III achalasia $\rightarrow$ POEM (longer myotomy).

Sources

- BL 28e, ch. 66 “The oesophagus”, §“Oesophageal manometry; ambulatory reflux monitoring”, pp. 1110–1112; Fig. 66.9, p. 1111.
- BL 28e, ch. 66 “The oesophagus”, §“Motility disorders: manometric classification”, pp. 1121–1122; Fig. 66.23, p. 1122.
- SAB 22e, ch. 83 “Benign Esophageal Disorders”, §“Esophageal function tests: esophageal manometry”, pp. 1109–1112; Table 83.1, p. 1110.
- SAB 22e, ch. 83 “Benign Esophageal Disorders”, §“Primary esophageal motility disorders”, pp. 1113–1114.
- SAB 22e, ch. 83 “Benign Esophageal Disorders”, §“Achalasia: treatment by subtype”, p. 1117.

Asked in: Paper IV · Dec 2025 · Q3(b) (5 marks)

“Third space endoscopy.”

NOT COVERED IN SOURCE

Searched BL 28e and SAB 22e full text and the BL index for 'third space endoscopy', 'third-space', 'submucosal tunnel', 'tunnelling'; chapters BL 9 (Gastrointestinal endoscopy), BL 66 (Oesophagus), SAB 12 (Advances in endoscopy), SAB 83 (Benign oesophageal disorders). The term 'third space endoscopy' is not used; the books describe these procedures as submucosal tunnelling techniques, given below.

Principle

- Mucosa opened a **short distance away from the target**; a **submucosal tunnel** is developed between mucosa and muscle layer to reach it.
- Myotomy or resection is done inside the tunnel with **ESD instruments**; the intact overlying mucosa is closed with **endoclips** (or endoscopic sutures).
- Incisionless, natural-orifice surgery — performed by advanced surgical endoscopists.

Procedures

Table 25.1d.1. Submucosal tunnel procedures

Procedure	Indication	Key step	Result
POEM (peroral endoscopic myotomy)	Achalasia (all types; preferred for type III); DES, hypercontractile oesophagus, hypertensive LOS	Circular-muscle myotomy ≥ 6 cm oesophageal + 2–3 cm onto cardia	Relief >90%; ~80% clinical success at 5 years
Z-POEM	Zenker's diverticulum (best with septum 2.5–5 cm)	Tunnel on both sides of the septum; cricopharyngeus divided	Good results ~90%; complications ~6%
G-POEM / POP (per-oral pyloromyotomy)	Refractory gastroparesis; post-oesophagectomy without pyloroplasty	Tunnel from 2 cm proximal to pylorus; all circular pyloric fibres divided	Success 85.1% vs 84% for surgical pyloroplasty
D-POEM	Epiphrenic diverticulum	Myotomy without resecting the diverticulum	High success; reflux 40–50%
STER (submucosal tunnelling endoscopic resection)	Oesophageal leiomyoma / subepithelial tumour of muscularis propria (<5 cm)	Entry 3–5 cm proximal; ESD enucleation; specimen out through mouth	Lowers threshold for removing small leiomyomas

Source: BL 28e pp. 1124, 1134; SAB 22e pp. e157–e159, e162, 1117, 1120–1121

Technique of POEM (prototype)

- **Preparation:** clear liquids 48 h, nil by mouth from midnight; some give **fluconazole 200 mg daily for 3 days** (oesophageal candidiasis).
- General anaesthesia, supine; gastroscope with dissecting cap and overtube; **CO₂ insufflation** (absorbs faster).
- Lavage of residue; inspect for neoplasia/**pseudo-achalasia**; tattoo 2 cm distal to the GEJ on the lesser curve.
- 1. **Saline lift + vertical mucosotomy** (10–12 cm above the GEJ).
- 2. **Submucosal tunnel** past the GEJ for 2–3 cm onto the stomach.
- 3. **Myotomy of circular fibres**, longitudinal fibres preserved; scope passes easily, Hill grade II valve.
- 4. **Clip closure** of the mucosotomy, distal to proximal.
- **Post-op:** chest X-ray (capnotherax), contrast study on day 1, liquids then purée for 1 week.

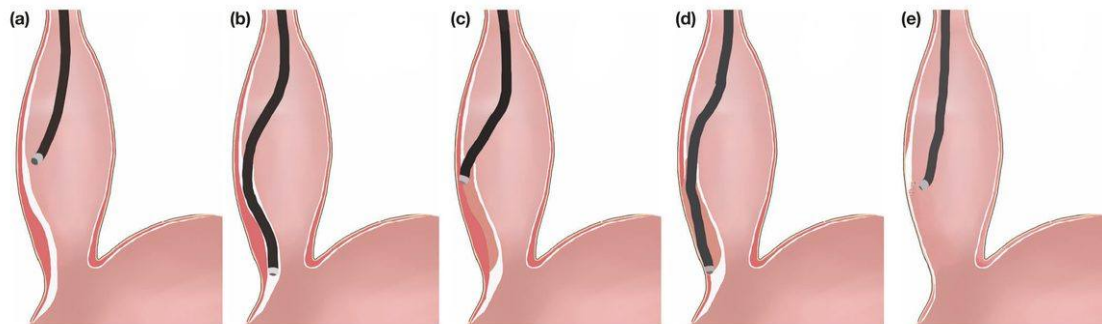


Figure 25.1d.1 — POEM: (a) mucosal opening; (b) submucosal tunnel to the stomach; (c) myotomy begins below the opening; (d) myotomy into proximal stomach; (e) closure with endoclips.

BL 28e Fig. 66.27, p. 1125

Advantages

- No skin incision; **myotomy length tailored** to HRM and barium findings — can extend proximally for spastic disorders.
- POEM **non-inferior to laparoscopic Heller myotomy** (RCT, 2 years: 83% vs 81.7%); similar efficacy to pneumatic dilatation.
- G-POEM: shorter stay, operative time and blood loss than pyloroplasty.

Follow-up after POEM

- **Eckardt score** at first visit — >3 means persistent or recurrent symptoms.
- **Timed barium swallow**: column >5 cm at 5 minutes predicts failure.
- **pH monitoring at 6–12 months** (symptoms correlate poorly with reflux); **EGD** for oesophagitis, Barrett's and the raised squamous carcinoma risk; FLIP distensibility; repeat HRM only for persistent dysphagia.

Limitations and complications

- **Reflux** (no fundoplication): pathological reflux ~50% (57% vs 20% after LHM); oesophagitis up to 57% at 3 months — lifelong PPI or later antireflux operation.
- **Capnothorax, capnomediastinum, capnoperitoneum** — decompress with Veress needle or pigtail drain if large.
- Mucosal injury, **leak**, bleeding, perforation; sigmoid oesophagus doubles complication risk.
- Learning curve about **20 cases**; long-term data limited for newer variants.

Pearls

- ✓ Third-space = submucosal tunnel: enter mucosa away from target, work in the tunnel, clip the entry.
- ✓ POEM: circular myotomy ≥ 6 cm + 2–3 cm onto the cardia; longitudinal fibres spared.
- ✓ Type III achalasia → POEM.
- ✓ Achilles heel of POEM = reflux (~50%).
- ✓ STER for leiomyoma <5 cm; G-POEM for gastroparesis; Z-POEM for Zenker's.

Sources

- BL 28e, ch. 9 "Gastrointestinal endoscopy", §"Gastrointestinal endoscopy: achalasia", p. 151.
- BL 28e, ch. 66 "The oesophagus", §"Achalasia: peroral endoscopic myotomy", pp. 1124–1125; Fig. 66.27, p. 1125.
- BL 28e, ch. 66 "The oesophagus", §"Benign tumours: leiomyoma, STER", pp. 1134–1135; Fig. 66.43, p. 1135.
- SAB 22e, ch. 12 "Advances and Training Considerations in Endoscopy and En", §"Per oral endoscopic myotomy; Zenker diverticulotomy; per oral pyloromyotomy", pp. e155–e159.
- SAB 22e, ch. 12 "Advances and Training Considerations in Endoscopy and En", §"Submucosal tunneled endoscopic resection", p. e162.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", §"Achalasia: peroral endoscopic myotomy", p. 1117.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", §"Z-POEM; epiphrenic diverticulum: D-POEM", pp. 1119–1121.

Asked in: Paper III · Dec 2020 · Q4 (3+2+5 marks) ; Paper III · Apr 2023 · Q1 (3+3+4 marks)

“Clinical features, investigations and treatment of gastro-oesophageal reflux disease in a 40-year-old male.”
“Briefly discuss the clinical features, investigations and treatment of gastroesophageal reflux Disease (GERD).”

Definition and basis

- **Montreal definition:** reflux of stomach contents causing **troublesome symptoms and/or complications**; mild symptoms ≥ 2 days a week or moderate/severe symptoms >1 day a week are troublesome.
- Imbalance between the **reflux barrier** (LOS, crural diaphragm, angle of His, intra-abdominal oesophagus) and the **thoraco-abdominal pressure gradient**.
- Contributors: excess **TLOS**s, hypotensive LOS, **sliding hiatus hernia**, defective oesophageal clearance, post-meal **acid pocket**, **central obesity** (raised intra-abdominal pressure).

Clinical features

- **Typical (oesophageal) symptoms:**
 - **Heartburn** — retrosternal burning; **regurgitation** of gastric content into mouth or hypopharynx (indicates more severe disease); acid or bitter taste.
 - **Waterbrash** (reflex salivation), epigastric pain (erosive oesophagitis).
 - Provoked by **large, fatty or spicy meals**, stooping, exercise; **nocturnal/supine** symptoms disturb sleep.
- **Extra-oesophageal symptoms:**
 - Chronic **cough**, **laryngitis**, throat clearing, globus, **asthma**, **dental erosions** (lingual and palatal surfaces); association with sinusitis, pharyngitis, pulmonary fibrosis.
- **Symptoms of complications** (alarm features):
 - **Dysphagia** — stricture, large hiatus hernia or carcinoma.
 - **Bleeding** — erosive oesophagitis, **Cameron ulcers** in a large hernia → haematemesis, anaemia.
 - Barrett's oesophagus is usually silent.
- **Examination** usually normal; record **BMI** (central obesity); dental changes, cough.
- Chest pain with cardiac risk factors → exclude a **cardiac cause** first.
- Symptoms correlate poorly with the severity of mucosal injury.

Investigations

- Uncomplicated typical symptoms: diagnosis is often **presumed** and an **empirical PPI trial** given — a 2-week response occurs in 69% with GORD but also in 51% without it (placebo effect).
- Investigate if symptoms are atypical, **refractory to PPI**, alarm features are present, or **surgery is planned** (four-test work-up).

Table 25.2a.1. Investigations in GORD

Test	Finding	Meaning / use
Upper GI endoscopy (essential before surgery)	Oesophagitis (LA grade A–D), stricture, hiatus hernia on retroflexion, Barrett's	LA C/D oesophagitis, peptic stricture and Barrett's are diagnostic ; excludes cancer, eosinophilic oesophagitis
24-h ambulatory pH / pH-impedance (gold standard)	Sensor 5 cm above LOS; AET $>6\%$ diagnostic ($>4\%$ abnormal); DeMeester score >14.7 ; symptom index $>50\%$	Proves pathological reflux; impedance detects non-acid reflux and belching; off PPI for 2 weeks
Wireless capsule pH	Records up to 96 h	Higher yield; better tolerated
High-resolution manometry	LOS pressure/length, hiatus hernia, body peristalsis	Excludes achalasia; places pH probe; tailors fundoplication ; cannot diagnose GORD
Barium swallow	Hiatus hernia anatomy, stricture, reflux	Large/paraoesophageal hernia; post-operative wrap problems
Biopsy	Intestinal metaplasia, dysplasia	Barrett's; eosinophilic oesophagitis

Source: BL 28e pp. 1111–1115; SAB 22e pp. 1113, 1121–1122

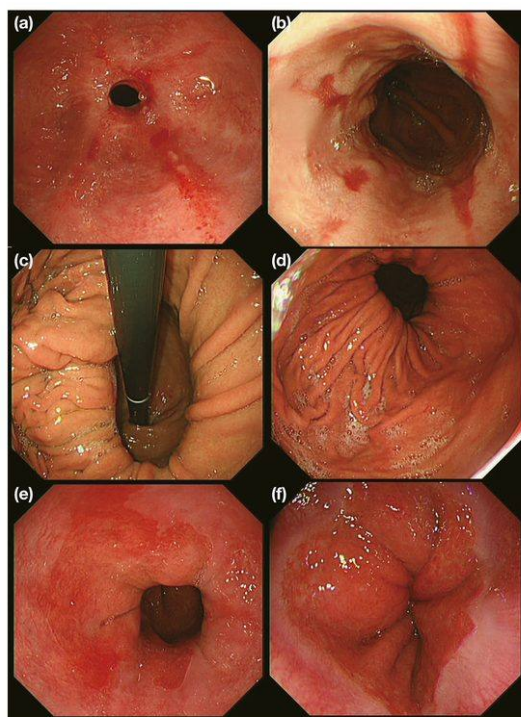


Figure 25.2a.1 — Endoscopy in GORD: (a) stricture with oesophagitis; (b) LA grade C oesophagitis; (c, d) hiatus hernia (retrograde and end view); (e) Barrett's oesophagus; (f) normal OGJ.

BL 28e Fig. 66.14, p. 1114

Treatment

1. Lifestyle modification

- **Weight loss** if overweight; **stop smoking** and alcohol; avoid overeating and trigger foods (carbonated drinks, tea, coffee).
- Early evening meal, avoid lying down after meals, **elevate the head of the bed**.

2. Medical treatment

- **Antacids / antacid-alginate** (raft over the acid pocket) — temporary relief, do not heal oesophagitis.
- **H₂-receptor antagonists** (famotidine) — breakthrough symptoms; tachyphylaxis.
- **PPI — first-line**: full dose for **8 weeks** → symptom relief within days and **>90% healing** of oesophagitis; then **step down** to the lowest effective dose (or stop).
- Inadequate response: switch PPI, increase dose or give **twice daily**, add an H₂ antagonist; persistent symptoms after 4–8 weeks → test before further escalation.
- **P-CABs** (potassium-competitive acid blockers): faster, reversible proton-pump inhibition.
- Prokinetics (metoclopramide, domperidone) and other TLOSR inhibitors — not useful.
- Hypersensitivity/functional heartburn: **perception modulators** (tricyclics, SSRIs).
- Long-term PPI: possible CKD worsening, *C. difficile*, pneumonia, fundic gland polyps — use the lowest effective dose.
- **Peptic stricture**: dilatation + long-term PPI (prolongs the interval between dilatations); antireflux surgery is an alternative.
- **Barrett's oesophagus**: treat the underlying reflux; surveillance or endoscopic eradication by grade of dysplasia.
- **Symptomatic paraoesophageal hernia** with reflux: repair with crural closure and a partial fundoplication.

3. Surgery — laparoscopic fundoplication

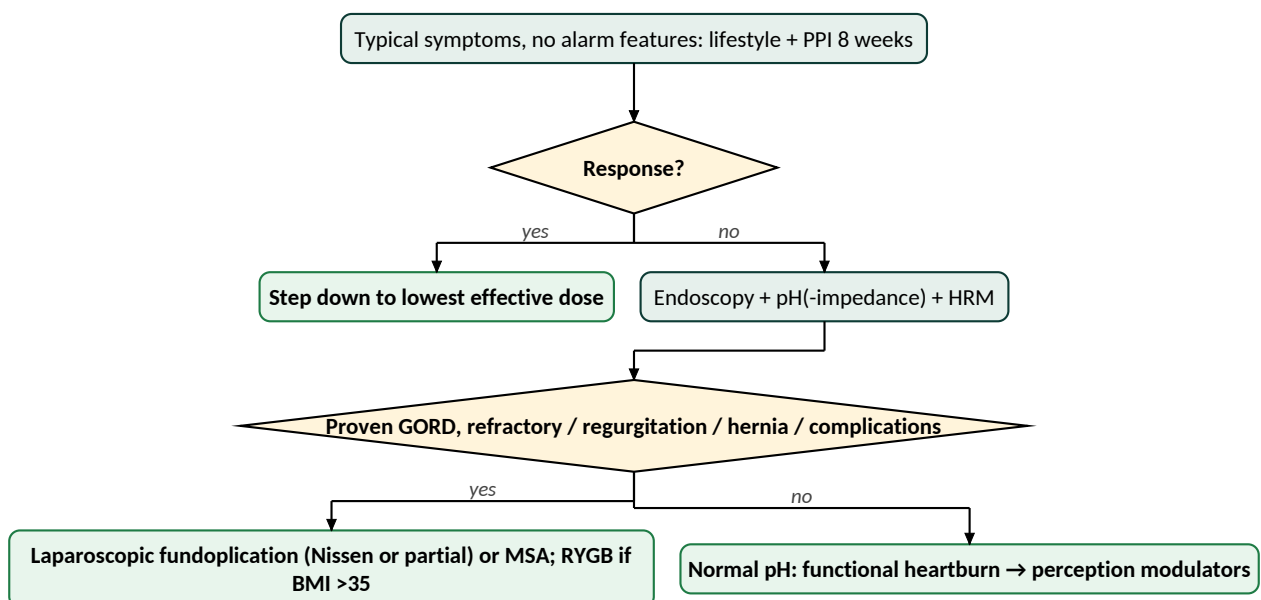
- **Indications**:
 - Incomplete control or **refractory** to maximal medical therapy; intolerance of or unwillingness to take long-term PPI (a young patient).
 - **Regurgitation** despite PPI; **large hiatus hernia**; **complications** (oesophagitis ≥ grade B, stricture, non-dysplastic Barrett's); extra-oesophageal symptoms.

- **Good outcome predicted by:** typical symptoms, PPI response, hiatus hernia, objective reflux; **poor** with normal pH study, functional heartburn, eosinophilic oesophagitis, connective tissue disease, extreme obesity.
- **Pre-requisites:** endoscopy, pH monitoring and HRM; tailor wrap to motility.
- **Principles:** restore ≥ 2 –3 cm intra-abdominal oesophagus, **crural repair**, and LOS reinforcement:
 - **Nissen 360°** (short floppy wrap over a bougie) — most durable, more early dysphagia and gas bloat.
 - **Partial** — posterior **Toupet** (240–270°) or anterior **Dor/Watson**: fewer side effects, slightly higher late failure; preferred with poor motility.
- **Magnetic sphincter augmentation (LINX):** titanium bead ring around the OGJ; similar efficacy, less gas bloat; for LA A/B, hernia ≤ 3 cm, BMI < 35 , adequate motility.
- **BMI > 35 :** Roux-en-Y gastric bypass is an antireflux option (only option if BMI > 50).
- **Operative steps (laparoscopic):**
 - Supine or French position, steep reverse Trendelenburg, liver retractor, ≥ 4 ports; antibiotics and DVT prophylaxis.
 - Open pars flaccida $\rightarrow$ right crus; divide short gastric vessels $\rightarrow$ left crus and angle of His; posterior window, Penrose sling; 360° hiatal clearance.
 - Mobilise oesophagus in the mediastinum for **≥ 3 cm intra-abdominal length**; close crura posteriorly with interrupted permanent sutures without narrowing the oesophagus; construct the wrap.
- **Post-operative:** full liquid diet ≥ 1 week, then purée and soft diet; early dysphagia (up to 50%) is usually self-limiting.
- **Counselling:** small mortality, failure, side effects (dysphagia, gas bloat — failure to belch or vomit, flatulence) before an irreversible operation.
- **Results:** 80–90% satisfied; mortality 0.1–0.5%; failure 5–10%; dysphagia/gas bloat/discomfort $\sim 10\%$; 20–40% back on PPI at 15–20 years.
- **MSA is contraindicated** with paraoesophageal hernia, LA C/D oesophagitis, Barrett's, dysphagia, poor motility or need for MRI > 1.5 T.

4. Endoscopic options (selected patients)

- **Transoral incisionless fundoplication (TIF 2.0)** — 270°, 3-cm valve with H-fasteners; better than PPI but fundoplication preferred.
- Radiofrequency (Stretta), MUSE stapling, antireflux mucosectomy / argon plasma coagulation — only for small or no hiatus hernia, no LA C/D oesophagitis, no obesity.

Flowchart. Management of GORD



Pearls

- ✓ GORD = troublesome symptoms and/or complications of reflux (Montreal).
- ✓ Heartburn + regurgitation; regurgitation responds poorly to PPI.
- ✓ Gold standard test: ambulatory pH monitoring (AET >6%, DeMeester >14.7).
- ✓ Endoscopy before surgery: LA C/D, stricture or Barrett's confirm GORD.
- ✓ PPI 8 weeks then step down; HRM before surgery to exclude achalasia.
- ✓ Operation of choice: laparoscopic fundoplication with crural repair.
- ✓ Exam trap: a normal pre-operative pH study predicts a poor surgical result.

Sources

- BL 28e, ch. 66 "The oesophagus", § "Ambulatory reflux monitoring; GORD aetiology", pp. 1111–1112.
- BL 28e, ch. 66 "The oesophagus", § "GORD: clinical features, diagnosis, management of uncomplicated GORD", pp. 1114–1115; Fig. 66.14, p. 1114.
- BL 28e, ch. 66 "The oesophagus", § "Surgical management; endoscopic treatment; peptic strictures", pp. 1116–1117.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", § "Antireflux mechanism: pathophysiology of GERD", pp. 1108–1109.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", § "Ambulatory pH monitoring", p. 1113.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", § "GERD: evaluation, diagnostic workup, medical and surgical management, MSA", pp. 1121–1126.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", § "Endoscopic management; GERD and obesity", pp. 1127–1128.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", § "Paraesophageal hernia repair: fundoplication", p. 1132.

25.2b

Management of Barrett's oesophagus

1x

Asked in: Paper III · Oct 2023 · Q6(a) (5 marks)

"Management of Barrett's esophagus."

Definition

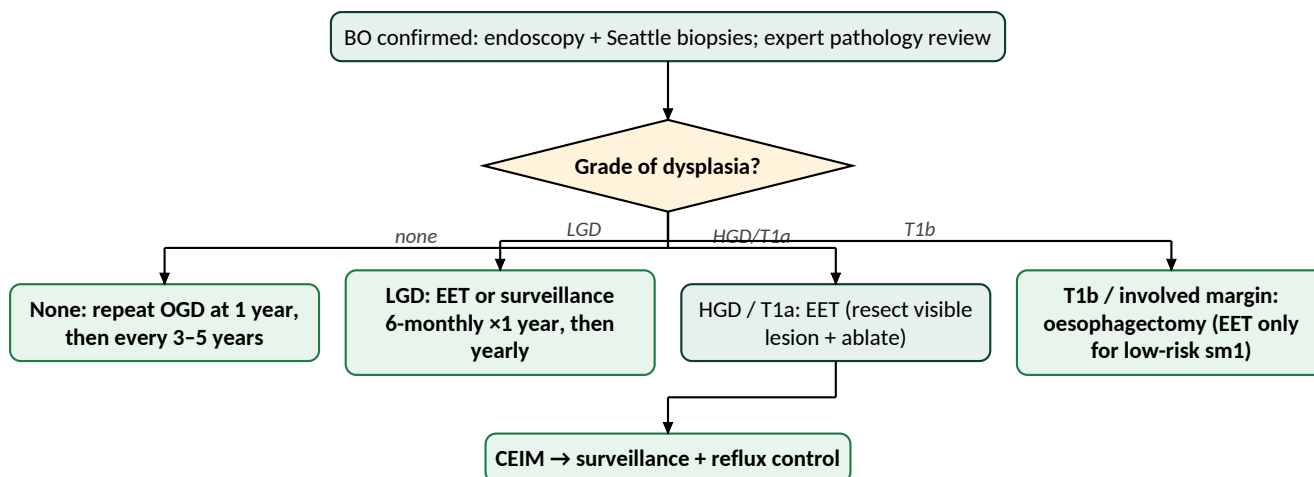
- **Barrett's oesophagus (BO)**: proximal migration of **columnar (salmon-coloured) epithelium** in the lower oesophagus extending **>1 cm above the OGJ** (top of gastric folds); the need for biopsy-proven **intestinal metaplasia (goblet cells)** is controversial.
- Precursor of **adenocarcinoma**; annual cancer risk ~**0.2–0.5%** (non-dysplastic), ~**0.7%** (LGD), up to **7%** (HGD).

Assessment

- **High-definition white-light endoscopy + NBI**; chromoendoscopy (methylene blue, acetic acid, indigo carmine).
- Extent by **Prague C&M** (circumferential and maximal length) — longer segments carry more risk.
- **Seattle protocol**: four-quadrant biopsies every **2 cm** (every 1 cm if previous dysplasia) + targeted biopsies of visible lesions.
- **Dysplasia must be confirmed by a second expert pathologist**; grade of dysplasia is the best predictor of progression.

Management by grade

Flowchart. Management of Barrett's oesophagus (by histology)



1. Treat the reflux

- **At least daily PPI; twice daily during eradication** to favour neosquamous regrowth; aspirin with high-dose PPI may improve outcome (chemoprevention trial).
- **Antireflux surgery** for GORD symptoms or complications not controlled medically, large hiatus hernia, or difficulty with eradication — **not as an antineoplastic measure**.

2. Surveillance

- **Non-dysplastic**: repeat OGD within 1 year, then every **3 years (>3 cm)** to **5 years (<3 cm)**.
- Mass screening of the population is not cost-effective.

3. Endoscopic eradication therapy (EET)

- **Endoscopic resection** of every visible (nodular, ulcerated, irregular) lesion first — **EMR** (cap or multiband; en bloc <2 cm) or **ESD** (en bloc, >15 mm, poor lift) — to stage depth.
- **Ablation** of the remaining flat Barrett's: **radiofrequency ablation (RFA)** — most popular, depth to muscularis mucosae, low stricture rate; cryotherapy; photodynamic therapy.
- Circumferential EMR causes strictures — stage it (half the circumference at a time).
- **Goal: complete eradication of intestinal metaplasia (CEIM)** — two endoscopies ≥ 3 months apart without visible or histological BO.
- Recurrence after EMR $\pm$ RFA $\sim 2.6\text{--}3.3\%$ $\rightarrow$ endoscopy within 1 year, then lifelong surveillance.

4. Surgery

- **Oesophagectomy**: T1b disease or involved lateral/deep margins after endoscopic resection; EET is preferred over oesophagectomy even for HGD.

Pearls

- ✓ BO = columnar lining >1 cm above the OGJ; describe with Prague C&M.
- ✓ Seattle protocol: 4-quadrant biopsies every 2 cm.
- ✓ Dysplasia needs two expert pathologists.
- ✓ HGD/T1a $\rightarrow$ resect visible lesions, then RFA; goal CEIM.
- ✓ Fundoplication controls reflux but is not anticancer therapy.

Sources

- BL 28e, ch. 66 "The oesophagus", §"Barrett's oesophagus: diagnosis, surveillance, treatment", pp. 1119–1120; Fig. 66.21, p. 1119.
- SAB 22e, ch. 84 "Esophageal Cancer", §"Endoscopic therapies: Barrett esophagus, EET, EMR/ESD", pp. 1145–1147; Fig. 84.8, p. 1146.
- SAB 22e, ch. 12 "Advances and Training Considerations in Endoscopy and En", §"Endoscopic mucosal resection: Barrett esophagus", p. e160.

Asked in: Paper III • May 2024 • Q10(a) (5 marks)

“Different types of funduplications.”

Principle

- Three components of every antireflux operation: (i) **restore intra-abdominal oesophagus** (≥ 3 cm), (ii) **crural repair**, (iii) **reinforce the LOS** by the gastric fundus (fundoplication) or a prosthesis.
- Creates a **floppy valve** at the OGJ and restores the **angle of His** → raises LOS basal pressure, reduces TLOSRS, reduces fundal capacity (faster gastric emptying).
- Standard approach: **laparoscopic** (or robotic) with hiatal reconstruction.

Types

Table 25.2c.1. Types of fundoplication

Feature	Nissen (complete)	Toupet (posterior partial)	Dor / Watson (anterior partial)
Wrap	360° total wrap of fundus behind and around the oesophagus	240–270° posterior wrap	Anterior wrap (~180°)
Technique	Short gastrics divided; fundus passed behind (shoeshine); 56 Fr bougie ; fundus-to-fundus sutures over 2–3 cm	Shoeshine as for Nissen; fundus sutured to the oesophagus on each side; top stitches include crura	No posterior dissection or shoeshine; fundus brought over the front and sutured to hiatus and oesophagus
Reflux control	Most durable	Equivalent to Nissen at 3 years (RCT)	Slightly less than Nissen at 15–20 years
Side effects	More early dysphagia , gas-bloat , inability to belch	Slightly less short-term dysphagia	Less dysphagia and belching
Main use	Normal motility GORD	GORD with dysmotility (IEM); paraoesophageal hernia; after Heller myotomy	After Heller myotomy (covers mucosa); paraoesophageal hernia

Source: BL 28e pp. 1116, 1124; SAB 22e pp. 1122–1125

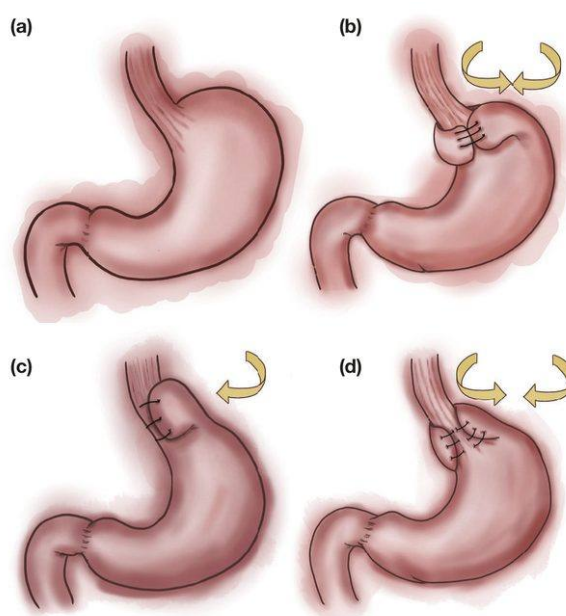


Figure 25.2c.1 — Types of fundoplication: (a) normal anatomy; (b) Nissen 360°; (c) Dor anterior; (d) Toupet posterior.

BL 28e Fig. 66.16, p. 1116

Steps common to all types (laparoscopic)

- Reverse Trendelenburg, liver retractor, ≥ 4 ports.
- Divide the **pars flaccida** → right crus (watch for an aberrant hepatic artery); open the phreno-oesophageal ligament, leaving fascia on the crus.

- Divide the **short gastric vessels** (needed for Nissen/Toupet) → left crus and angle of His; posterior window with a Penrose sling around the oesophagus.
- Mediastinal mobilisation until **≥3 cm of intra-abdominal oesophagus**; **posterior crural closure** with interrupted permanent sutures.
- Nissen: keep the **greater curve (cut short-gastric edge) cephalad**; the shoeshine manoeuvre avoids a twisted, tight or too-loose wrap; optional wrap-to-oesophagus and gastropexy sutures prevent slippage and herniation.
- Partial wraps need **no bougie** (not circumferential); Toupet adds a posterior wrap-to-hiatus stitch.

Choice

- No type has proven superior in trials; **tailor to anatomy and HRM motility**.
- **Partial** wraps: fewer short-term side effects at the cost of slightly higher long-term failure; 2023 multisociety consensus conditionally **favours partial** (less hernia recurrence, dysphagia, inability to belch).
- **Achalasia**: Heller myotomy + **Dor or Toupet**; **Nissen contraindicated** (outflow resistance against an aperistaltic oesophagus).
- FLIP distensibility index (Toupet 2.6–3.7; Nissen >2.2) may calibrate tightness — not yet mainstream.

Non-fundoplication alternatives

- **Magnetic sphincter augmentation (LINX)**: titanium-coated magnetic bead ring, “Roman arch”; similar efficacy, less gas-bloat; erosion 0.3–1%.
- **Transoral incisionless fundoplication (TIF 2.0)**: endoscopic 270°, 3-cm valve with polypropylene H-fasteners.
- **Roux-en-Y gastric bypass** — antireflux operation for BMI >35.

Pearls

- ✓ Nissen 360°: most durable, most dysphagia/gas bloat; short, floppy, over a bougie.
- ✓ Toupet 240–270° posterior; Dor anterior.
- ✓ Partial wrap for poor motility and with Heller myotomy.
- ✓ Never a Nissen after myotomy for achalasia.
- ✓ Satisfaction ~90% after Nissen or Toupet.

Sources

- BL 28e, ch. 66 “The oesophagus”, §“Surgical management: which operation?”, pp. 1116–1117; Fig. 66.16, p. 1116.
- BL 28e, ch. 66 “The oesophagus”, §“Achalasia: laparoscopic myotomy with partial fundoplication”, p. 1124.
- SAB 22e, ch. 83 “Benign Esophageal Disorders”, §“GERD: operative technique; Nissen and partial fundoplication; MSA”, pp. 1122–1126; Fig. 83.17, p. 1125.

Asked in: Paper III · Dec 2025 · Q6(a) (5 marks)

“Scoring systems for GERD.”

Purpose

- Objective grading of **symptoms, mucosal injury, reflux burden, complications and anatomy** — to prove the diagnosis, select patients for surgery and compare outcomes.

1. Symptom definition — Montreal

- GORD = reflux causing **troublesome symptoms and/or complications**; troublesome = mild symptoms ≥ 2 days/week or moderate–severe >1 day/week.

2. Endoscopic — Los Angeles (LA) classification of oesophagitis

Table 25.2d.1. Los Angeles classification

Grade	Mucosal break
A	One or more breaks ≤ 5 mm, not extending between the tops of two mucosal folds
B	One or more breaks >5 mm, not extending between the tops of two folds
C	Breaks continuous between the tops of ≥ 2 folds , involving $<75\%$ of the circumference
D	Breaks involving $\geq 75\%$ of the circumference

Source: BL 28e Fig. 66.15, p. 1115

- LA C and D**, peptic stricture and Barrett’s are **diagnostic of GORD**; grade $\geq B$ predicts a good surgical result; MSA and endoscopic therapy are for LA A/B only.

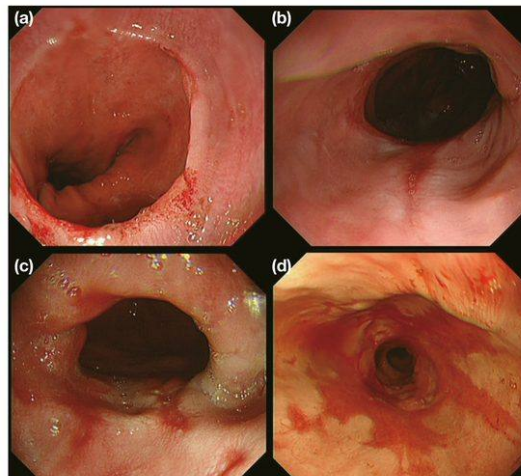


Figure 25.2d.1 — Los Angeles grades A–D of reflux oesophagitis.

BL 28e Fig. 66.15, p. 1115

3. Ambulatory pH monitoring scores

- Acid exposure time (AET)** — % time pH <4 ; **$>4\%$ abnormal, $>6\%$ diagnostic.**
- Johnson–DeMeester composite score** (six parameters), **abnormal >14.7** :
 - Total number of reflux episodes; % total time pH <4 ; % upright time pH <4 ; % supine time pH <4 ; number of episodes ≥ 5 minutes; longest episode (minutes).
 - Reflects reflux pattern (upright, supine, bipositional) and clearance; correlates with severity.
- Symptom correlation**:
 - Symptom index (SI)** — % of symptoms related to reflux; positive **$>50\%$** .
 - Symptom sensitivity index (SSI)** and **symptom association probability (SAP)** (SAP 100% = every reflux episode symptomatic).
- Discordant reflux and symptom association (hypersensitivity, functional heartburn) predicts failure of antireflux therapy.

- **How applied:** pH study **off PPI for 2 weeks** to diagnose; **on-PPI** study to test breakthrough reflux in refractory cases; wireless capsule extends recording to 96 h.
- **pH-impedance:** drop in impedance = liquid, rise = air; distinguishes acid, non-acid reflux and belching.
- **Limitations:** no reference value or standard technique for the height of proximal reflux; meal periods excluded; manometry cannot diagnose GORD.

4. Barrett's — Prague C&M criteria

- **C** = circumferential extent, **M** = maximal extent (cm above the top of the gastric folds); e.g. C3 M8. Length is a risk factor for neoplasia.

5. Anatomical classifications

- **Hiatus hernia types I–IV:** I sliding (85–95%); II true paraoesophageal; III mixed; IV other viscera in the sac.
- **HRM OGJ morphology types I–III:** progressive separation of LOS and crural pressure (manometric hiatus hernia).
- **Horgan classification of fundoplication failure:** IA (OGJ and wrap above diaphragm), IB (OGJ above), II (paraoesophageal herniation of the wrap), III (malformed wrap from gastric body).

Pearls

- ✓ DeMeester score >14.7 = abnormal; AET >6% = diagnostic.
- ✓ LA grade by length (5 mm) and circumference (75%) of mucosal breaks.
- ✓ LA C/D, stricture or Barrett's confirm GORD without pH testing.
- ✓ Symptom index positive >50%.
- ✓ Prague C&M describes Barrett's length.

Sources

- BL 28e, ch. 66 "The oesophagus", § "Ambulatory reflux and pH-impedance monitoring; GORD aetiology", pp. 1111–1113; Fig. 66.12, p. 1113.
- BL 28e, ch. 66 "The oesophagus", § "GORD: clinical features, diagnosis", pp. 1114–1115; Fig. 66.15, p. 1115.
- BL 28e, ch. 66 "The oesophagus", § "Barrett's oesophagus: Prague C&M", p. 1119; Fig. 66.21, p. 1119.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", § "Esophageal manometry: LES and EGJ", p. 1110.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", § "Ambulatory pH monitoring", p. 1113.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", § "GERD: upper endoscopy", p. 1121.
- SAB 22e, ch. 83 "Benign Esophageal Disorders", § "Outcomes of fundoplication: Horgan classification", p. 1126; Fig. 83.18, p. 1126.

25.2e

Complications of laparoscopic antireflux surgery

1×

Asked in: Paper III · Dec 2025 · Q6(b) (5 marks)

"Complications of laparoscopic anti-reflux surgery."

Overview

- Perioperative **mortality ~0.1%** (0.1–0.5%); **bleeding, infection, gastric or oesophageal perforation ~1% each**.
- Failure 5–10%; side effects (dysphagia, gas bloat, discomfort) ~10%; **reoperation 5–7%** in the long term.

Table 25.2e.1. Complications of laparoscopic antireflux surgery

Complication	Timing / cause	Recognition	Management
Bleeding	Intra-op: short gastrics, spleen, aberrant hepatic artery in pars flaccida	Intra-op; falling Hb	Careful energy use away from stomach and spleen; control
Gastric / oesophageal perforation	~1%; thermal energy applied near the stomach during dissection	Intra- or post-operative	Prevention: keep energy device off the stomach; repair
Pleural breach / capnothorax	Mediastinal dissection, recurrent repairs	Respiratory change, CXR	Usually inconsequential (positive-pressure ventilation, CO ₂ absorbs); if respiratory problems: enlarge the tear, lower insufflation pressure, raise ventilation rate
Vagal nerve injury	Hiatal/mediastinal dissection	Gas bloat, gastroparesis	Identify and protect vagi; gastric emptying study
Early dysphagia	Up to 50% in first months — oedema, inflammation	Usually self-limiting by 3 months	Liquid diet ≥ 1 week, then purée/soft; severe (cannot swallow saliva) → immediate revision
Persistent dysphagia (>3 months)	Tight/long wrap, tight hiatal closure, herniated or malformed wrap	Barium swallow , endoscopy	Endoscopic dilatation; revision (Nissen → partial)
Gas-bloat syndrome	Loss of belching (more after Nissen)	Distension, failure to belch/vomit, flatulence	Avoid carbonated drinks, eat slowly, simethicone; exclude gastroparesis
Recurrent / persistent GORD	Loose, disrupted, slipped or herniated wrap	Symptoms + pH study	PPI (20–40% at 15–20 years); revision if anatomical
Wrap herniation / failure	Commonest anatomical failure; obesity raises risk	Barium, endoscopy (Horgan types)	Revision surgery after full work-up

Source: BL 28e p. 1117; SAB 22e pp. 1122, 1124, 1131–1132

Prevention (technique)

- Stay close to the crus and leave its fascia intact — avoids injury to nearby structures and allows closure.
- Keep the energy device off the stomach and spleen when dividing the short gastrics.
- Identify and **protect both vagi**; obtain ≥ 3 cm intra-abdominal oesophagus off tension.
- Calibrate the Nissen over a **bougie**, keep it **short (2–3 cm) and floppy**; avoid an over-tight hiatal closure (test by retracting the oesophagus).
- Use a **partial wrap** for dysmotility; anchor sutures reduce slippage; avoid permanent mesh at the hiatus (erosion).
- **Obesity** (BMI >35) increases wrap slippage, hernia recurrence and failure — consider RYGB.

Anatomical failures — Horgan classification

- **Type IA**: OGJ and wrap above the diaphragm — mostly heartburn ± dysphagia.
- **Type IB**: OGJ above the diaphragm; **Type II**: paraoesophageal herniation of the top of the wrap; **Type III**: malformed wrap made from the gastric body — mostly **dysphagia**.

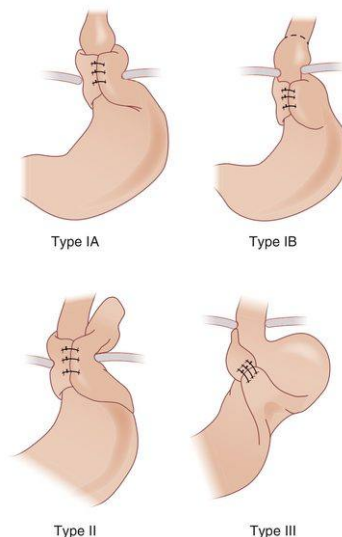


Figure 25.2e.1 — Horgan classification of fundoplication failures (types IA, IB, II, III).

SAB 22e Fig. 83.18, p. 1126

Evaluation of symptoms after surgery

- **Dysphagia >3 months** → barium swallow: hiatal herniation, over-tight hiatal closure or malformed wrap.
- **Endoscopy, contrast radiography and functional tests** (HRM, pH study) define whether the wrap is too tight, too loose, disrupted, slipped or herniated.
- **Bloating** not settling → gastric emptying study for post-vagotomy gastroparesis.
- Management ladder: **PPI** for recurrent acid reflux → **endoscopic dilatation** for stenosis → **surgical revision** as a last resort (tight complete wrap → convert to partial).

Complications of magnetic sphincter augmentation

- Prolonged **dysphagia** needing dilatation; device removal 2.7–3.3%.
- **Erosion** into the oesophagus 0.15–1% (1–4 years; undersizing a risk); migration.

Revisional surgery

- Full work-up: **HRM, pH monitoring, endoscopy, barium swallow**; exclude dysmotility or hypersensitivity.
- Higher threshold than primary surgery; satisfaction falls from **91% → 76% → 49%** with each operation; adhesions may make revision impossible.

Pearls

- ✓ Commonest side effect: transient dysphagia — resolves by 3 months.
- ✓ Inability to swallow saliva after fundoplication = immediate re-operation.
- ✓ Gas bloat is commoner after Nissen.
- ✓ Commonest cause of anatomical failure: wrap herniation (Horgan IA).
- ✓ Investigate persistent dysphagia with barium swallow.

Sources

- BL 28e, ch. 66 “The oesophagus”, §“Surgical management; complications of antireflux surgery and revisional surgery”, pp. 1115–1117.
- SAB 22e, ch. 83 “Benign Esophageal Disorders”, §“GERD: operative technique; outcomes of fundoplication”, pp. 1122–1124.
- SAB 22e, ch. 83 “Benign Esophageal Disorders”, §“Magnetic sphincter augmentation”, pp. 1125–1126; Fig. 83.18, p. 1126.
- SAB 22e, ch. 83 “Benign Esophageal Disorders”, §“Obesity and antireflux surgery”, p. 1128.
- SAB 22e, ch. 83 “Benign Esophageal Disorders”, §“Paraesophageal hernia repair: operative technique”, pp. 1131–1132.

Asked in: Paper III · Dec 2022 · Q9 (3+7 marks) ; Paper III · Jun 2020 · Q4(a) (5 marks) ; Paper III · Jun 2020 · Q4(b) (5 marks)

“Briefly discuss the diagnosis and treatment of carcinoma esophagus.”

“Outline the investigations and staging of carcinoma oesophagus.”

“Management options for a 45 year old patient with carcinoma of lower third of oesophagus.”

Pathology and spread (relevant to work-up)

- **Squamous cell carcinoma (SCC)**: middle (50%) and upper oesophagus; smoking, alcohol, achalasia, lye stricture, Plummer–Vinson.
- **Adenocarcinoma**: lower oesophagus and OGJ (80%), arises in **Barrett’s oesophagus**; GORD, obesity.
- Rich submucosal lymphatics → **longitudinal submucosal spread**; nodes from neck to mediastinum to **coeliac axis** (30–70%); blood spread to liver, lung, bone, brain; **peritoneal spread** in OGJ adenocarcinoma.

Diagnosis — clinical features

- **Progressive dysphagia** — solids → soft → liquids; usually means $\geq T3$ disease; with **weight loss**, anorexia, odynophagia, regurgitation.
- **Hoarseness** — recurrent laryngeal nerve involvement; **cough or choking on drinking water** — oesophago-airway fistula; haemoptysis.
- Iron-deficiency **anaemia** (commoner in adenocarcinoma); sentinel bleed of aorto-oesophageal fistula.
- Examination: cachexia, dehydration, **supraclavicular/cervical nodes**, hepatomegaly; early cancers are asymptomatic (found at Barrett’s surveillance).

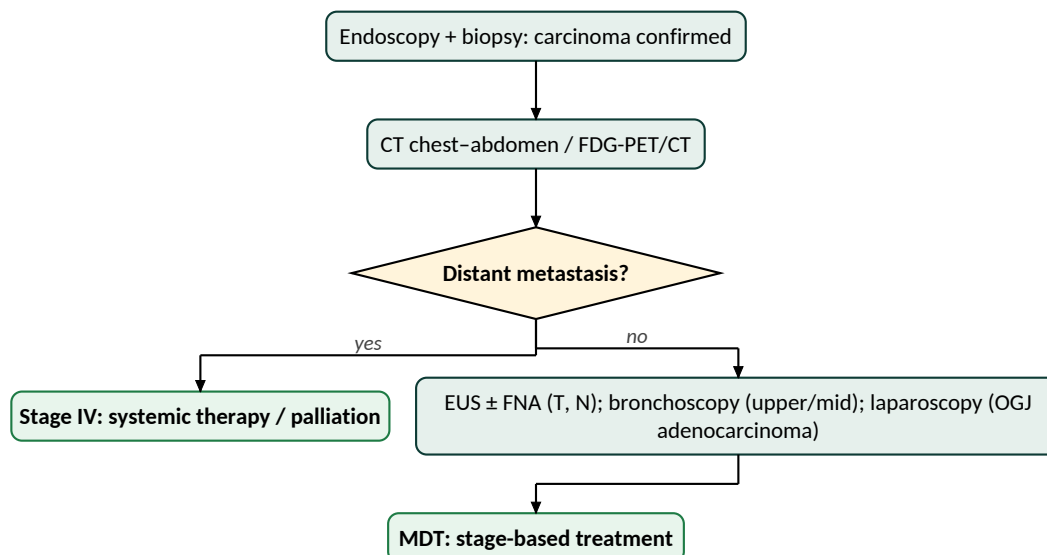
Investigations

Table 25.3.1. Investigations and staging work-up

Test	Finding	Purpose
Upper GI endoscopy + multiple biopsies (diagnostic)	Ulcerated/stenotic mass; distance from incisors and OGJ, length, obstruction, Barrett’s (Prague)	Tissue diagnosis mandatory ; assess stomach as conduit, synchronous tumours, vocal cords
Lugol’s iodine / NBI chromoendoscopy	Unstained areas; abnormal IPCL loops	Early SCC and dysplasia
Barium swallow	Ulcerated or stenotic lesion with proximal dilatation	Length; fistula
CECT chest + abdomen	Loss of fat planes (T4), distant nodes, liver/lung metastases	M staging ; resectability
FDG-PET/CT	Avid primary, regional/non-regional nodes, distant metastases	Best for M1 (sensitivity 69%, accuracy 84%); response after neoadjuvant therapy; no value for T
EUS ± FNA	Five-layer wall; tumour depth; para-oesophageal and coeliac nodes	Best for T and N ($\approx 85\%$ T, 75% N); FNA confirms nodes
EMR / ESD	Depth of invasion (T1a vs T1b), LVI	Most accurate staging of superficial (T1) lesions
Bronchoscopy	Airway infiltration	Upper/mid-third tumours — airway invasion contraindicates resection
Staging laparoscopy	Peritoneal/liver deposits	Lower-third/OGJ adenocarcinoma (not SCC)
Neck ultrasound ± FNA	Cervical nodes	Nodal M/N staging
Bloods; cardiorespiratory assessment; nutrition	Anaemia, hypoproteinaemia, raised calcium/abnormal LFT (metastases)	Fitness for multimodal therapy
Biomarkers	HER2, MMR/MSI, PD-L1	Targeted therapy, immunotherapy

Source: BL 28e pp. 1136–1139; SAB 22e pp. 1139–1143

Flowchart. Staging pathway after biopsy



Staging — AJCC/UICC 8th edition TNM

Table 25.3.2. TNM classification of oesophageal cancer

Category	Definition
Tis	High-grade dysplasia — malignant cells confined to the epithelium by the basement membrane
T1a / T1b	Invades lamina propria or muscularis mucosae / invades submucosa
T2	Invades muscularis propria
T3	Invades adventitia
T4a	Invades pleura, pericardium, azygos vein, diaphragm or peritoneum (resectable)
T4b	Invades aorta, vertebral body or airway (unresectable)
N1 / N2 / N3	1–2 / 3–6 / ≥7 regional nodes (paratracheal in neck to coeliac nodes)
M1	Distant metastasis

Source: BL 28e Table 66.4, p. 1138; SAB 22e Table 84.2, p. 1142

- **Separate stage groups** for SCC and adenocarcinoma; separate **cTNM**, **pTNM** and **ypTNM** (post-neoadjuvant).
- Grade enters pathological staging; **location** (upper/middle/lower by azygos vein and inferior pulmonary vein) only for SCC.
- **OGJ tumour** with epicentre ≤2 cm into the cardia is staged as oesophageal; >2 cm as gastric.

Treatment by stage

Table 25.3.3. Stage-based treatment

Stage	Adenocarcinoma	Squamous cell carcinoma
Tis, T1a	Endoscopic resection (EMR/ESD) ± ablation or oesophagectomy	Endoscopic resection ± ablation or oesophagectomy
cT1b–T2 N0, low risk (well differentiated, no LVI, <3 cm)	Oesophagectomy	Oesophagectomy
Locally advanced resectable (cT2 high-risk, cT1b–T2 N1, cT3–T4a any N)	Neoadjuvant CRT + oesophagectomy, or perioperative chemotherapy (FLOT) + oesophagectomy, or definitive CRT	Neoadjuvant CRT + oesophagectomy, or definitive CRT (cervical oesophagus)
Unresectable (cT4b) or unfit	Definitive CRT; systemic therapy	Definitive CRT; systemic therapy
Stage IV	Systemic therapy, palliation, best supportive care	Systemic therapy, palliation

Source: SAB 22e Table 84.4, p. 1144; BL 28e pp. 1139–1141

1. Endoscopic therapy (early cancer)

- T1a rarely metastasises → **EMR** (small lesions) or **ESD** (en bloc regardless of size); curative if T1a, clear margins, no LVI.
- T1b SCC, or T1b adenocarcinoma with high-risk features (SM2/3 — nodes in ~40%) → oesophagectomy.
- Resection of >2/3 circumference risks stricture — steroids reduce it.

2. Neoadjuvant / perioperative therapy ($\geq$ T3 or N+)

- **CROSS**: carboplatin + paclitaxel with **41.4 Gy in 23 fractions**, surgery at 4–6 weeks — R0 92% vs 69%; pCR 49% SCC, 29% adenocarcinoma; median survival 49.4 vs 24 months; 5-year OS 47% vs 34%.
- **FLOT** (fluorouracil, leucovorin, oxaliplatin, docetaxel; 4 cycles before and after) — standard perioperative regimen for adenocarcinoma (FLOT4: median OS 50 vs 35 months vs ECF); ESOPEC: FLOT better than CROSS (3-year OS 57.4% vs 50.7%).
- Restage with **FDG-PET/CT 5–8 weeks** after neoadjuvant therapy.
- **Adjuvant nivolumab** for residual disease after CRT and R0 resection (CheckMate 577: DFS 22.4 vs 11.0 months).

3. Surgery — oesophagectomy

- **Preparation**: cardiorespiratory assessment; stop smoking and alcohol; chest physiotherapy, incentive spirometry, prehabilitation; nutrition by fine-bore NG tube or **feeding jejunostomy** (preserves the stomach); ERAS pathway.
- **Goals**: **R0 resection**, adequate two-field lymphadenectomy ($\geq$ 15 nodes), well-perfused tension-free conduit — **stomach on the right gastroepiploic artery**; colon if stomach unavailable.
- **Approaches**:
 - **Ivor Lewis (Lewis–Tanner)**: laparotomy/laparoscopy + right thoracotomy/thoracoscopy, intrathoracic anastomosis — for distal tumours.
 - **McKeown (three-stage)**: chest, abdomen, neck — any level; cervical anastomosis.
 - **Transhiatal**: abdomen + neck, blunt mediastinal mobilisation — least invasive, fewer pulmonary complications, limited nodal clearance.
 - **Left thoracoabdominal / Sweet**: bulky distal or OGJ tumours.
 - **Minimally invasive (MIE)**: fewer pulmonary complications, shorter stay, equal survival.
- **Lymphadenectomy fields**:
 - **Two-field** = mediastinum + upper abdomen around the coeliac trifurcation; mediastinal field standard (infracarinal), extended (+ right recurrent laryngeal nerve nodes) or total (+ left recurrent laryngeal chain).
 - **Three-field** adds bilateral cervical nodes — selected SCC, especially upper thoracic; adenocarcinoma usually infracarinal two-field.
- **Reconstruction routes** for a neck anastomosis: posterior mediastinal, retrosternal or subcutaneous; pyloric drainage optional after the inevitable vagotomy.
- **Resectability**: tumour >5 cm from the cricopharyngeus in a fit patient; cervical cancers need pharyngo-laryngo-oesophagectomy or definitive CRT.
- High-volume centres: mortality 2–4% (<5% in dedicated centres); major morbidity 30–40% — AF, pneumonia, anastomotic leak, conduit ischaemia, chyle leak, recurrent laryngeal nerve palsy.

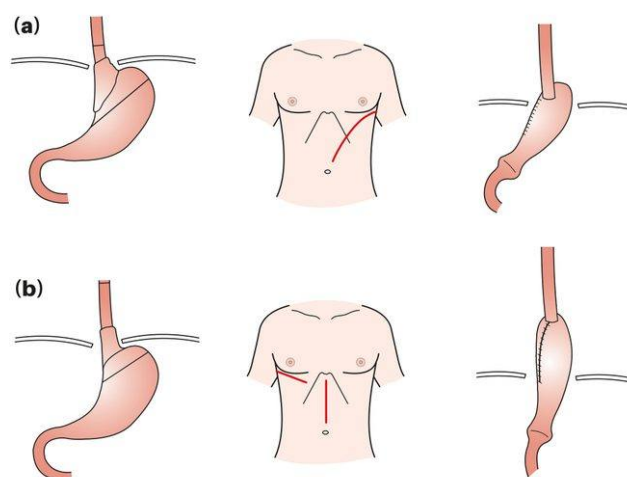


Figure 25.3.1 — Common open approaches to oesophagectomy: (a) left thoracoabdominal; (b) two-stage Lewis-Tanner (Ivor Lewis).
BL 28e Fig. 66.52, p. 1143

Management options — lower-third carcinoma in a 45-year-old

- Usually **adenocarcinoma** (Barrett's); define the epicentre relative to the OGJ (≤ 2 cm into cardia = oesophageal); staging laparoscopy for peritoneal disease.
- Young and fit → aim for **cure** with multimodal therapy after MDT discussion.
- **Early (T1a)**: EMR/ESD + ablation of residual Barrett's; **T1b–T2 N0 low risk**: primary oesophagectomy.
- **Locally advanced (T3/N+)**: **perioperative FLOT** or **CROSS CRT**, then **Ivor Lewis oesophagectomy** (open or minimally invasive) with two-field (infracarinal) lymphadenectomy and gastric pull-up; McKeown if the proximal margin is in doubt.
- OGJ-centred tumour: oesophagectomy or **extended total gastrectomy** with lower oesophageal resection and Roux-en-Y.
- Unfit or declines surgery: **definitive CRT** (≥ 50 Gy with cisplatin + taxane).
- **Metastatic**: systemic chemotherapy $\pm$ trastuzumab (HER2+) or immunotherapy; palliate dysphagia.

4. Definitive chemoradiotherapy

- For unfit patients, those declining surgery and **cT4b**; ≥ 50 Gy (no benefit of dose escalation); RTOG 85-01: 5-year OS 27% vs 0% for RT alone.
- Standard alternative for **cervical oesophagus** (larynx preservation); salvage oesophagectomy for residual or recurrent disease.

5. Palliation

- **Self-expanding metal stent** — immediate relief of dysphagia; risks migration, tumour ingrowth, airway compression, tracheal erosion.
- Palliative RT **30–35 Gy** or brachytherapy; dilatation, laser, photodynamic therapy; RT is haemostatic for bleeding OGJ tumours.

Follow-up and prognosis

- History and examination every 3–6 months for 2 years, then 6–12 months to 5 years; after neoadjuvant therapy + surgery, cross-sectional imaging 6-monthly for 2 years then yearly; endoscopy after definitive CRT or endoscopic therapy.
- ~90% of recurrences within 2 years, mostly distant; 5-year survival 40–50% with multimodality treatment; stage and R0 resection are the strongest predictors.

Pearls

- ✓ Endoscopy with biopsy is diagnostic; EUS is best for T and N; PET/CT for M.
- ✓ Dysphagia usually means at least T3 disease.
- ✓ OGJ epicentre ≤ 2 cm into the cardia = oesophageal staging.
- ✓ T1a → endoscopic resection; T3/N+ → neoadjuvant therapy then oesophagectomy.
- ✓ CROSS = carboplatin/paclitaxel + 41.4 Gy; FLOT for adenocarcinoma.
- ✓ Lower third: Ivor Lewis oesophagectomy with gastric conduit on the right gastroepiploic artery.
- ✓ Palliation of dysphagia: self-expanding metal stent.

Sources

- BL 28e, ch. 66 “The oesophagus”, § “Carcinoma of the oesophagus: pathology, clinical features, diagnosis, staging”, pp. 1136–1139; Table 66.4, p. 1138.
- BL 28e, ch. 66 “The oesophagus”, § “Treatment: endoscopic; surgery; surgical techniques; lymphadenectomy; reconstruction”, pp. 1140–1144; Fig. 66.52, p. 1143.
- BL 28e, ch. 66 “The oesophagus”, § “Perioperative care; complications; multimodality treatment; palliation”, pp. 1145–1146.
- SAB 22e, ch. 84 “Esophageal Cancer”, § “Clinical presentation; diagnosis and evaluation; staging; treatment overview”, pp. 1139–1144; Tables 84.2, 84.4, pp. 1142, 1144.
- SAB 22e, ch. 84 “Esophageal Cancer”, § “Surgery; radiation; chemotherapy; multimodal therapy; definitive chemoradiation; follow-up”, pp. 1147–1153.

25.4 Management of corrosive (caustic) oesophageal injury

1×

Asked in: Paper III · Dec 2021 · Q5(a) (5 marks)

“Describe management of a patient with corrosive esophageal injury.”

Basis

- Ingestion is **accidental in children, suicidal in adults**; severity depends on **type, pH, quantity and duration** of exposure.
- **Acid** → **coagulative necrosis** with eschar that limits deep penetration; **alkali** → **liquefactive necrosis**, penetrating deeper — more severe injury.
- May cause **transmural necrosis and perforation** of the oesophagus; the stomach can also be injured.

1. Immediate assessment and resuscitation

- Identify the agent, **test its pH**; consider a suicidal attempt.
- Symptoms: pain in neck, throat, chest or abdomen; **drooling**, dysphagia, odynophagia.
- **Hoarseness = laryngeal injury** and threatened airway → careful assessment and **fibreoptic intubation** or a **surgical airway**.
- **No gastric lavage and no attempt to neutralise** the acid or alkali.
- Supportive care: **IV fluids, oxygen, cardiorespiratory monitoring**.

2. Evaluation once stable

- **Careful endoscopy** — extent and depth of injury graded by **Zargar**; the longer and more circumferential the injury, the greater the risk of **stricture**; the stomach is assessed too.
- Place a **nasogastric/nasoduodenal tube under endoscopic guidance** for enteral feeding — it also preserves a route to the stomach if a stricture forms.
- **CT with IV contrast** — oesophageal oedema and surrounding soft-tissue infiltration (transmural injury).

Table 25.4.1. Zargar endoscopic classification of caustic oesophageal injury

Grade	Endoscopic description
0	Normal appearance
1	Oedema and hyperaemia
2a	Superficial ulceration and friability
2b	Deep ulceration or circumferential ulceration
3a	Multiple deep ulceration and scattered necrosis
3b	Extensive necrosis
4	Perforation

Source: BL 28e Table 66.2, p. 1130

3. Acute treatment

- **Most injuries are managed conservatively** with supportive measures and tube feeding.
- **Clinical deterioration** (necrosis, perforation) → **emergency oesophagectomy**:
 - Transhiatal or thoracoscopic mobilisation.
 - **No immediate reconstruction** — **cervical oesophagostomy + gastrostomy**; feeding **jejunostomy** if the stomach also needs resection.
 - Planned delayed reconstruction.

4. Late complications — stricture and malignancy

- **Stricture** may form early and resist dilatation; routine systemic or intralesional steroids or topical mitomycin C are **not supported** by evidence.
- **Gradual endoscopic dilatation** — perforation rate is higher than for other strictures; long strictures often resist dilatation.
- **Refractory stricture** → **oesophagectomy or bypass**:
 - Oesophagectomy removes the oesophagus and its long-term **cancer risk**, but scarring and mediastinal adhesions make it difficult.
 - **Bypass** often preferable: gastric conduit by the **retrosternal route** to the neck; **colonic interposition** if the stomach is damaged; the native oesophagus may be left (mucocoele risk low).
- **Lye corrosive stricture** predisposes to **squamous cell carcinoma** — long-term follow-up.

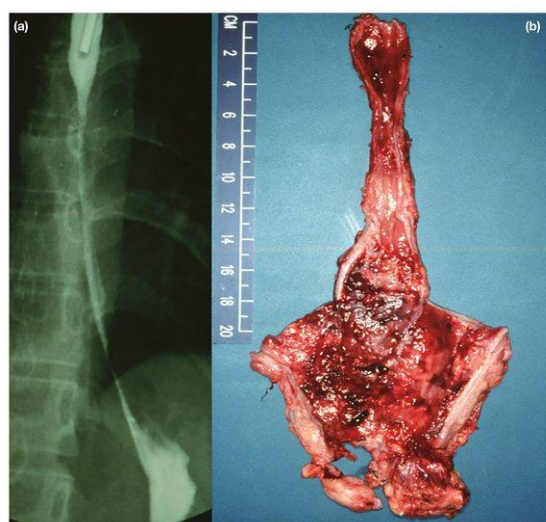
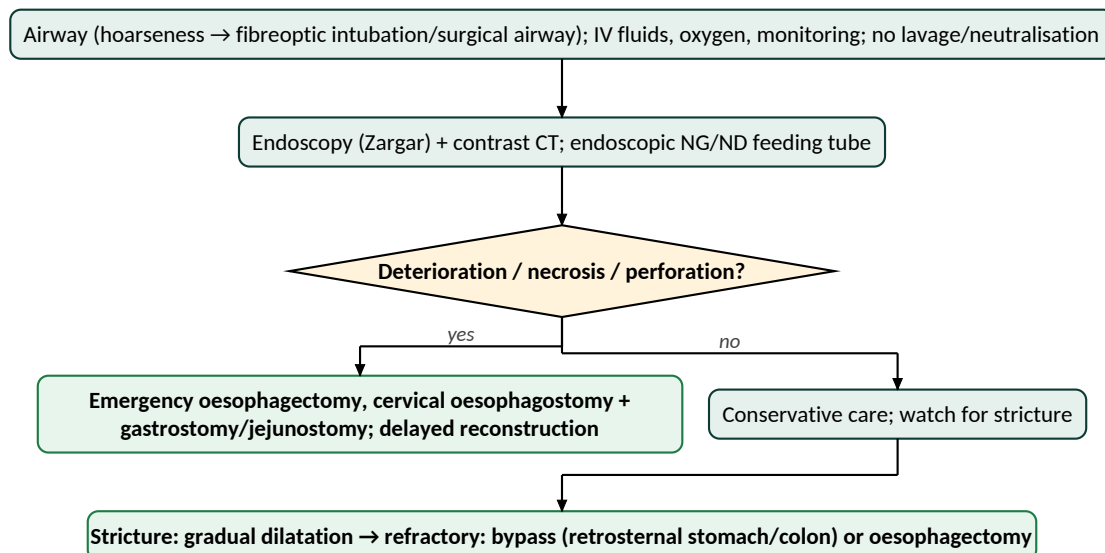


Figure 25.4.1 — (a) Contrast study: long undilatable caustic stricture of the oesophagus; (b) oesophagogastrrectomy specimen (colonic interposition done) — scarred oesophagus and stomach.

BL 28e Fig. 66.35, p. 1131

Flowchart. Management of caustic ingestion



Pearls

- ✓ Alkali = liquefactive necrosis, deeper injury; acid = coagulative necrosis with eschar.
- ✓ Hoarseness signals laryngeal injury — secure the airway first.
- ✓ Never lavage or neutralise.
- ✓ Endoscopy with Zargar grading decides prognosis; long circumferential burns stricture.
- ✓ Emergency oesophagectomy: no immediate reconstruction.
- ✓ Caustic stricture carries a long-term risk of squamous carcinoma.

Sources

- BL 28e, ch. 66 “The oesophagus”, § “Caustic injury”, pp. 1129–1131; Table 66.2, p. 1130; Fig. 66.35, p. 1131.
- BL 28e, ch. 66 “The oesophagus”, § “Oesophageal perforation: aetiology”, p. 1127.
- BL 28e, ch. 66 “The oesophagus”, § “Carcinoma of the oesophagus: aetiology”, p. 1136; Table 66.3, p. 1136.
- SAB 22e, ch. 84 “Esophageal Cancer”, § “Esophageal cancer: risk factors”, p. 1136.

Stomach & Duodenum

26.1a

Gastric ulcer versus duodenal ulcer — differences

1x

Asked in: Paper III · Aug 2021 · Q10(a) (5 marks)

“Differentiate between a gastric and duodenal ulcer.”

Common ground

- **Peptic ulcer:** erosion of the GI mucosa that extends **through the muscularis mucosae**; in the absence of acid, peptic ulcers do not occur.
- Both are caused mainly by *H. pylori* and **NSAIDs**; smoking predisposes and increases relapse.
- Ulcers occur at a **junction between different types of epithelia**, in the epithelium least resistant to acid.
- Both perforate, bleed and cause stenosis; **symptoms are indistinguishable** — history and examination are of limited value in telling them apart.

Differences

Table 26.1a.1. Gastric ulcer versus duodenal ulcer

Feature	Gastric ulcer (GU)	Duodenal ulcer (DU)
Frequency	Much less common	More common than GU
Age and sex	Older patients (peak 55–65 years; rare before 40); equal sex incidence	Peak now in an older age group; still commoner in men
Social pattern	More prevalent in low socioeconomic groups and resource-poor countries	Rising in resource-poor nations
<i>H. pylori</i> association	70–90%	90%
NSAID ulcers	NSAID-induced ulcers more often in the stomach (Johnson type V)	Less often
Acid secretion	Relatively normal or reduced (types I, IV); raised only in types II and III	Higher than normal (overlap considerable)
Commonest site	Lesser curve, especially the incisura angularis (type I, 50–60%)	First part of the duodenum ; anterior and posterior "kissing ulcers"
Size and deformity	Tend to be larger; fibrosis → hourglass stomach	Fibrosis → pyloric stenosis ; giant ulcer may denude the cap
Penetration / bleeding	Posteriorly into pancreas, splenic artery , rarely transverse colon (gastrocolic fistula)	Posterior DU bleeds from the gastroduodenal artery ; anterior DU perforates
Pain	Recurrent epigastric pain	Well-localised mid-epigastric pain, frequently relieved by food ; back pain = penetration into pancreas
Weight	Often underweight	Weight loss or gain
Malignancy	May be malignant ; an ulcerated cancer can mimic a benign ulcer	Malignancy extremely rare, even in a giant DU
Biopsy	Mandatory — multiple (up to 10) biopsies, repeated while healing and when healed	Not routinely needed for malignancy
Follow-up endoscopy	Mandatory to confirm healing	Not routinely required
Commonest complication	Bleeding; perforation is the commonest one needing surgery (anterior lesser curve)	Bleeding (posterior) and perforation (anterior)
Classic operation (historical)	Excision of the ulcer — Billroth I gastrectomy	Acid reduction — vagotomy ± drainage, Pólya (Billroth II) gastrectomy

Source: BL 28e pp. 1157–1161; SAB 22e pp. 1174–1178

Gastric ulcer types (modified Johnson)

Table 26.1a.2. Modified Johnson classification of gastric ulcer

Type	Location	Acid level
I	Lesser curve at incisura (50–60%)	Low to normal
II	Gastric body with a duodenal ulcer (15–20%)	Increased
III	Prepyloric (≈20%) — behaves like a DU	Increased
IV	High on lesser curve near the GE junction (<10%)	Normal
V	Anywhere — long-term NSAID use	Normal, NSAID-induced

Source: SAB 22e Table 86.3, p. 1176

Key practical differences

- **Malignancy:** all gastric ulcers are regarded as malignant until proven otherwise, however classical their benign features; four biopsies give a 95% and seven a **98%** cancer yield.
- **Larger ulcers with irregular or heaped-up edges** are more likely to harbour cancer.
- **Pathogenesis:** DU follows **antral gastritis → raised gastrin and acid → gastric metaplasia in the duodenum** colonised by *H. pylori*; GU results more from the inflammatory response to infection, with normal or low acid.
- **Pyloric channel and prepyloric ulcers** behave like DU but may be malignant, so biopsy is essential.
- **At operation for perforated GU**, the ulcer is excised and sent for histology even if it looks benign; perforated DU is simply patched.
- **Acid-reducing vagotomy** is not needed for type I and IV GU (normal acid) but is added for types II and III.
- **Nonhealing GU** in the *H. pylori* era raises serious concern for malignancy.

Diagnosis — same tools, different emphasis

- **Flexible upper endoscopy** is the most reliable test for both; it allows biopsy for malignancy and *H. pylori* testing.
- At endoscopy, a “**U**” **manoeuvre** inspects the incisura and lesser curve; the whole of the first part of the duodenum is viewed and the scope passed into D2.
- **Barium meal** shows the crater (double contrast detects 80–90%); a deformed pylorus is common with chronic DU.
- **Serum gastrin** for refractory ulcers or before surgery (Zollinger–Ellison syndrome).

Pearls

- ✓ DU: commoner, younger, men, high acid, first part of duodenum, almost never malignant.
- ✓ GU: older, equal sex, normal/low acid, lesser curve at the incisura, may be malignant.
- ✓ Every GU — multiple biopsies + endoscopy until healed.
- ✓ Posterior DU bleeds (gastroduodenal artery); anterior DU perforates.
- ✓ Johnson types II and III are acid-hypersecretory → vagotomy with resection.

Sources

- BL 28e, ch. 67 “The stomach and duodenum”, §“Peptic ulcer: duodenal and gastric ulceration, clinical features, operations”, pp. 1157–1161.
- SAB 22e, ch. 86 “Stomach”, §“Peptic ulcer disease: pathogenesis, gastric ulcer types, clinical manifestations, diagnosis”, pp. 1174–1178.

Asked in: Paper III · Dec 2021 · Q7 (2+2+2+4 marks)

“Describe the surgical anatomy of the stomach and discuss the clinical features, evaluation and management of a case of bleeding duodenal ulcer.”

Surgical anatomy of the stomach

• Parts:

- **Cardia** — opening of the oesophagus, just distal to the lower oesophageal sphincter; **fundus** — to the left of the cardia, the superior-most part, floppy and distensible.
- **Angle of His** between fundus and left margin of oesophagus — an anatomic antireflux sphincter.
- **Body (corpus)** — largest part, between the lesser and greater curvatures; ends at the **incisura angularis**, where the **antrum** begins; the **pylorus** joins antrum to duodenum.

- **Fixed** at the GE junction and pylorus; GE junction lies 2–3 cm below the hiatus at the level of the seventh chondrosternal joint.

• Relations:

- Anterior — left lateral section of the liver.
- Posterior (**stomach bed**) — diaphragm, left kidney, **pancreas, aorta, coeliac trunk**.
- **Gastrohepatic ligament** (right and left gastric arteries), **gastrocolic ligament** (gastroepiploic vessels), **gastrosplenic ligament** (short gastric arteries).

• Arteries — arcades on both curves, mainly from the **coeliac axis**:

- Lesser curve: **left gastric** (coeliac; the largest) + **right gastric** (hepatic).
- Greater curve: **right gastroepiploic** (gastrooduodenal) + **left gastroepiploic** (splenic).
- Fundus: **short gastric arteries (vasa brevia)** from the end of the splenic artery.
- The **gastrooduodenal artery** passes **behind the first part of the duodenum** — the vessel of a bleeding posterior DU; it divides into superior pancreaticoduodenal and right gastroepiploic arteries.
- The stomach survives ligation of three of the four main arteries if the arcades are intact.

- **Veins** follow the arteries — lesser curve to the **portal vein (coronary vein)**, dilates in portal hypertension), greater curve to the splenic vein; right gastroepiploic to **Henle’s trunk** → SMV.

- **Lymphatics** follow the vessels in four zones (superior gastric, suprapyloric, pancreaticolienal, inferior gastric/subpyloric) → **coeliac nodes** → thoracic duct; a rich submucosal plexus explains microscopic spread several cm from a cancer.

• Nerves:

- **Vagus** — left (anterior) gives the hepatic branch and the **anterior nerve of Latarjet**; right (posterior) gives the coeliac branch and the **“criminal” nerve of Grassi** (missed → recurrent ulcer); terminal **crow’s foot** to the antrum.
- Sympathetic T5–T10 via the coeliac ganglion; intrinsic Auerbach and Meissner plexuses.

- **Wall**: serosa, muscularis propria (three layers: middle **circular** — thickened as the pyloric sphincter — outer longitudinal), **submucosa (strongest layer)**, mucosa.

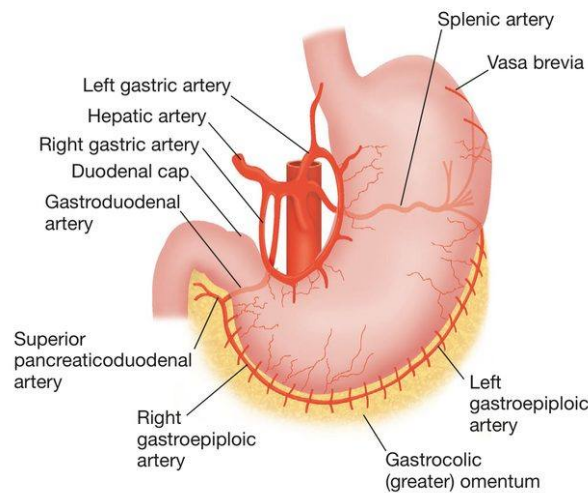


Figure 26.1b.1 — The arterial blood supply of the stomach.

BL 28e Fig. 67.1, p. 1148

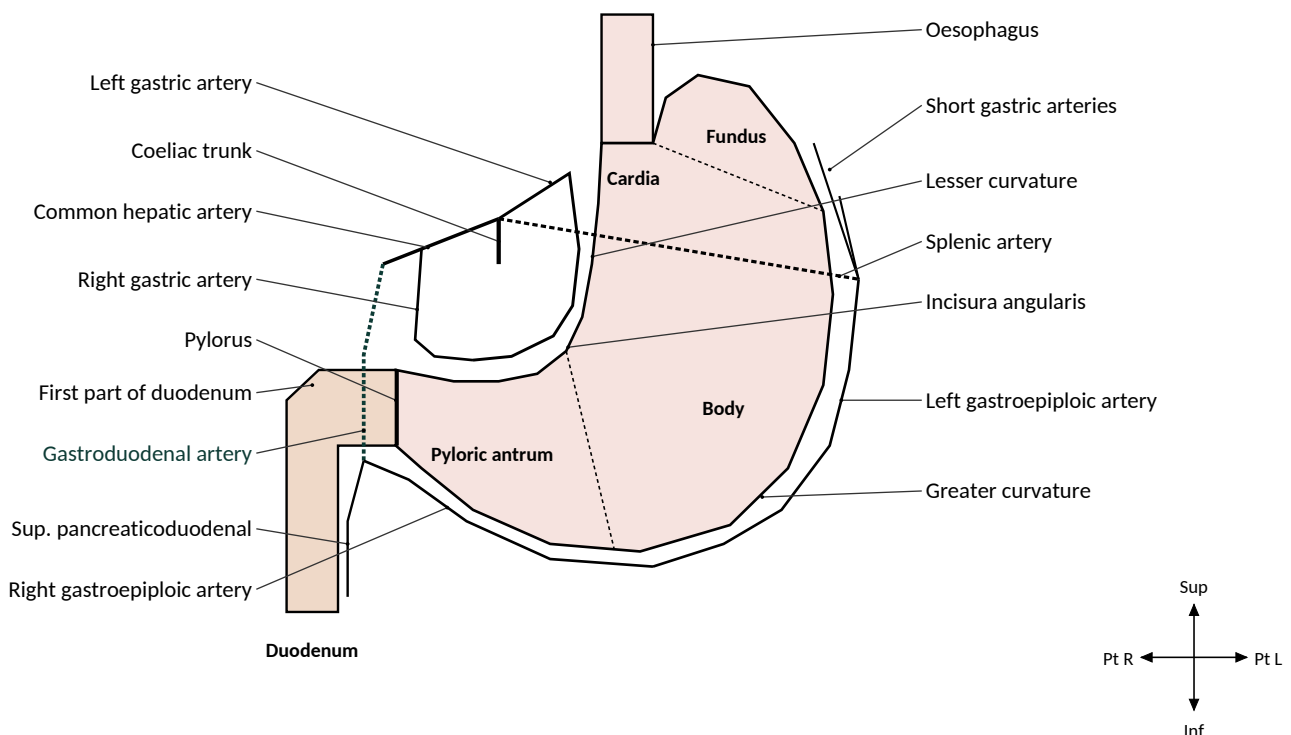


Figure 26.1b.2 — Schematic (exam diagram): parts of the stomach and its arterial supply (dashed = behind the stomach / duodenum; accent = gastroduodenal artery, the vessel in a bleeding posterior duodenal ulcer). Based on BL 28e, Fig. 67.1, p. 1148; text p. 1148; SAB 22e pp. 1166–1167.

Bleeding duodenal ulcer — clinical features

- Peptic ulcer is the **commonest cause** of upper GI bleeding (35–50%; DU 33% of all bleeds); patients are now older and commonly on **NSAIDs**; other risks: *H. pylori*, comorbidity, **anticoagulants**.
- **Posterior DU bleeds** by eroding the **gastroduodenal artery**; anterior DU perforates.
- **Haematemesis** (red or coffee-ground) and/or **melaena** — melaena may follow as little as 50 mL; massive UGIB may present as **haematochezia** with orthostatic hypotension.
- History: previous ulcer or bleed (up to 60% rebleed from the same lesion), NSAIDs, aspirin, P2Y12 inhibitors, warfarin/DOACs, SSRIs; liver disease (varices).
- Abdominal pain with **rebound/guarding** → suspect associated perforation.
- **Haemodynamic signs grade the loss:**
 - <15% (≤ 750 mL): vitals may be normal.
 - 15–30%: tachycardia >100 /min, widened pulse pressure, urine <30 mL/h.
 - 30–40%: hypotension, pulse ≈ 120 /min, confusion, urine <20 mL/h.

- >40%: pulse ≈140/min, tachypnoea, lethargy, negligible urine.

Evaluation

- **Bloods:** CBC (initial Hb may be near baseline; repeat 2–8-hourly), group and cross-match, coagulation screen (correct coagulopathy), LFTs, urea/creatinine — **BUN:creatinine >30:1** (urea:creatinine >100:1) favours an upper source; ECG and cardiac enzymes if at risk.
- **Rockall score** — pre-endoscopy for safe early discharge; post-endoscopy predicts rebleeding and death.
- **Upper GI endoscopy** — investigation of choice; **within 24 h, immediately** in severe bleeding, after resuscitation; erythromycin 250 mg IV 20–90 min before improves the view; routine NG lavage not recommended.
- **Forrest stigmata** grade rebleeding risk (table); test for *H. pylori* at endoscopy.
- **CT angiography / angiography** if no source is seen or bleeding recurs; **barium studies are contraindicated**.

Table 26.1b.1. Forrest classification of bleeding peptic ulcer

Stigmata	Forrest	Prevalence	Rebleeding risk
Active spurting	IA	10%	90%
Active oozing	IB	10%	10–20%
Non-bleeding visible vessel	IIA	25%	50%
Adherent clot	IIB	10%	25–30%
Flat pigmented spot	IIC	10%	5–10%
Clean base	III	35%	3–5%

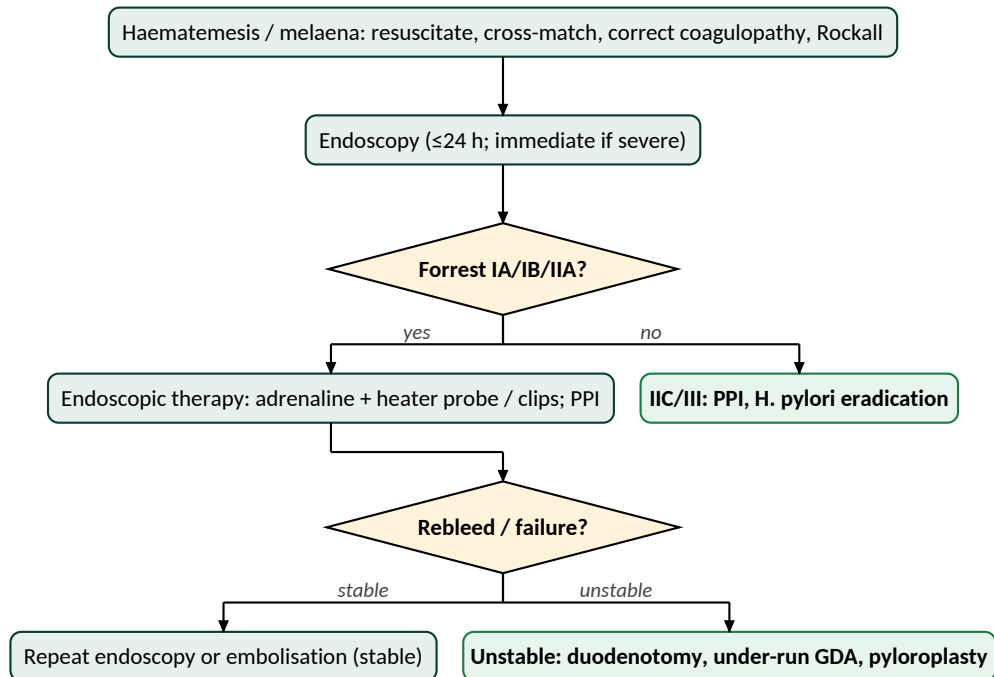
Source: SAB 22e Table 86.4, p. 1180

Management

- **Resuscitation:**
 - Two large-bore (≥18 G) cannulae; ICU for instability or active bleeding; CVP and bladder catheter in severe bleeding; intubation for ongoing haematemesis or altered consciousness.
 - Transfuse as clinically indicated (usually >30% volume lost); restrictive threshold **Hb <7 g/dL** in stable patients, **<8 g/dL** in high-risk (elderly, coronary disease); massive bleeding — RBC:plasma:platelets **1:1:1**.
 - Correct coagulopathy (liver disease, anticoagulants) with FFP or clotting-factor concentrates.
- **Endoscopic haemostasis** (≈70% success) for active bleeding or a visible vessel:
 - **Adrenaline injection + heater probe and/or clips**; electrocoagulation, argon plasma coagulation, ethanol; adrenaline never as monotherapy.
 - Clean base (III): no endoscopic therapy. Rebleed after success → **repeat endoscopy**.
- **Medical:**
 - No evidence for IV PPI **before** endoscopy; **PPI after endoscopy** prevents rebleeding — IV for 3 days, then oral for at least 2 weeks.
 - **Tranexamic acid** may reduce mortality.
 - ***H. pylori* eradication** (triple/quadruple therapy); stop NSAIDs or co-prescribe a PPI.
- **Angiographic embolisation** of the gastroduodenal artery — for rebleeding or unidentified source in stable patients, in expert centres; low ischaemia risk (rich collaterals), but rescue surgery after failed embolisation does poorly.
- **Surgery — indications:**
 - Haemodynamic instability despite resuscitation; failed endoscopic treatment; recurrent bleeding after repeated endoscopic/IR treatment.
 - Spurting vessel, visible vessel or clot in the base; **>6 units** of blood; **frail elderly** — early surgery.
- **Operation** (aim: stop the bleeding):
 - Upper midline laparotomy; **Kocher manoeuvre** — hand behind the gastroduodenal artery.
 - **Longitudinal duodenotomy** across the pylorus; direct pressure, then **under-run the vessel — three-point (U) stitch**: superior and inferior sutures ligate the GDA, a medial U-stitch controls the **transverse pancreatic branch**; avoid the **common bile duct**.
 - Close transversely (**Heineke–Mikulicz pyloroplasty**); truncal vagotomy classically added in the stable patient, but definitive acid-lowering surgery is no longer required.

- Giant ulcer destroying D1 → **distal gastrectomy + Roux-en-Y**, duodenal stump closed over a T-tube or drain (controlled fistula).
- Resection: less rebleeding but more duodenal leaks — **minimum surgery that stops the bleeding.**

Flowchart. Management of a bleeding duodenal ulcer



Pearls

- ✓ Gastroduodenal artery lies behind D1 — posterior DU bleeds, anterior DU perforates.
- ✓ Stomach arcades: LGA + RGA (lesser curve), RGEA + LGEA (greater curve), short gastrics (fundus).
- ✓ Endoscopy is diagnostic and therapeutic; Forrest IA/IB/IIA need treatment.
- ✓ Dual endoscopic therapy — adrenaline is never used alone.
- ✓ Surgery: Kocher, duodenotomy, three-point under-running of GDA (spare the CBD), pyloroplasty.
- ✓ >6 units transfused or frail elderly → operate early.

Sources

- BL 28e, ch. 67 “The stomach and duodenum”, §“Anatomy of the stomach and duodenum: blood supply, nerves”, pp. 1148–1149; Fig. 67.1, p. 1148.
- BL 28e, ch. 67 “The stomach and duodenum”, §“Duodenal ulceration: pathology”, pp. 1157–1158.
- BL 28e, ch. 67 “The stomach and duodenum”, §“Haematemesis and melaena; bleeding peptic ulcers; surgical treatment”, pp. 1165–1167.
- SAB 22e, ch. 86 “Stomach”, §“Stomach: gross anatomy, blood supply, lymphatics, innervation”, pp. 1166–1168.
- SAB 22e, ch. 86 “Stomach”, §“Complicated peptic ulcer disease: hemorrhage”, pp. 1180–1181; Table 86.4, p. 1180.
- SAB 22e, ch. 98 “Acute Gastrointestinal Hemorrhage”, §“Acute GI hemorrhage: initial evaluation, nonsurgical management of UGIB”, pp. 1544–1547.
- SAB 22e, ch. 98 “Acute Gastrointestinal Hemorrhage”, §“Surgical management of UGIB: bleeding duodenal ulcer”, p. 1549.

Every theory question set in the DNB General Surgery exit examination from June 2020 to December 2025, answered the way an examiner wants to read it.

Answers are written from Bailey & Love 28e, Sabiston 22e and Schwartz 11e, arranged part by part across three volumes, and updated till December 2025.

One answer, many questions

Repeats and near-repeats are merged into one answer. Its "Asked in" line lists every paper and year, and a badge shows how often it was set.

Honest about gaps

A red box marks a point the three textbooks do not cover and gives the closest book material. An amber box flags a newer guideline and names it.

Real textbook figures

Figures are cropped from the books with their figure number and printed page. Exam schematics are drawn for these notes and labelled as such.

Every answer sourced

Each answer ends with book, chapter, section and printed pages, so any line can be checked against the original.

The complete set

Volume 1	Volume 2	Volume 3
Principles of Surgery, Trauma & Burns	Skin & Soft Tissue, Musculoskeletal, Neurosurgery, Head & Neck, Endocrine, Breast, Vascular & Thorax	Gastrointestinal, Hepatobiliary-Pancreatic, Abdominal Wall & Hernia, Paediatric Surgery, Urology & Transplantation
Parts 1-13 · 172 answers · 255 questions	Parts 14-24 · 173 answers · 244 questions	Parts 25-39 · 205 answers · 269 questions

Most asked across the set

5.6	Surgical diathermy and energised dissection devices · Vol 1	5*
23.1b	Varicose veins and the CEAP classification · Vol 2	5*
25.1a	Achalasia cardia · Vol 3	4*
7.1a	WHO Surgical Safety Checklist · Vol 1	4*
15.4b	Malignant melanoma · Vol 2	4*

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