SURGICAL INFECTIONS

A Clinical and Surgical Review for Postgraduate Students

Classification & Microbiology • Surgical Site Infections
Antimicrobial Prophylaxis • Soft Tissue & Necrotising Infections
Intra-Abdominal Sepsis & Source Control • MDR Organisms
Asepsis & Sterilisation • Current Guidelines

Prof. Dr. Zahid Mahmood
MBBS, FCPS (Surgery)

A concise, examination-oriented text for postgraduate trainees in general surgery, surgical specialties and critical care

2026

Contents

Learning Objectives

LEARNING OBJECTIVES

Classify surgical infections by source, timing and anatomical site, and apply the CDC surgical wound classification.

Describe the microbiology of surgical infections and the host and operative factors that predispose to them.

Define and stage surgical site infections (SSI) using CDC/NHSN criteria, and outline an evidence-based SSI prevention bundle.

Apply the principles of surgical antimicrobial prophylaxis — timing, agent selection, weight-based dosing, and intraoperative redosing.

Recognise and manage soft tissue infections, including the early diagnosis and emergency management of necrotising soft tissue infections.

Outline the management of intra-abdominal infection, surgical sepsis and septic shock, including the role and timing of source control.

Describe the surgical relevance of multidrug-resistant organisms and the principles of antimicrobial stewardship.

Explain the principles of asepsis, antisepsis and sterilisation used to prevent surgical infection.

1. Introduction and Classification of Surgical Infections

Surgical infections remain among the commonest and most preventable complications of surgical care, ranging from a minor superficial wound infection to fulminant necrotising soft tissue infection and septic shock. They may be classified by their relationship to the operative wound (surgical site infection versus a remote-site infection such as pneumonia or catheter-related bloodstream infection), by their origin (community-acquired versus healthcare-associated), or by the anticipated degree of microbial contamination at operation.

1.1 CDC Surgical Wound Classification

The four-tier CDC wound classification predicts infection risk and guides the need for antimicrobial prophylaxis:

Class	Definition and examples
Class I — Clean	No inflammation, no break in aseptic technique, respiratory/GI/GU tract not entered (e.g., hernia repair, thyroidectomy).
Class II — Clean-contaminated	Respiratory, GI, or GU tract entered under controlled conditions without unusual contamination (e.g., elective cholecystectomy, elective colonic resection).
Class III — Contaminated	Major break in sterile technique, gross spillage from the GI tract, or acute non-purulent inflammation encountered (e.g., open fresh traumatic wound, enterotomy with spillage).
Class IV — Dirty/infected	Established infection, pus encountered, perforated viscus, or old traumatic wound with retained devitalised tissue.

Expected SSI rates rise progressively across this classification — approximately 1–5% for clean, 3–11% for clean-contaminated, 10–17% for contaminated, and over 27% for dirty/infected wounds — though modern prevention bundles have reduced these figures substantially across all classes.

2. Microbiology of Surgical Infections

Most surgical infections are endogenous, arising from the patient's own skin or mucosal flora displaced into a normally sterile space at the time of surgery or trauma, rather than from exogenous contamination. The expected pathogens vary predictably by anatomical site, and this predictability underpins rational empirical antibiotic selection.

Surgical site	Typical pathogens
Skin/soft tissue, clean surgery	*Staphylococcus aureus* (including MRSA), coagulase-negative staphylococci, β-haemolytic streptococci
Gastrointestinal tract surgery	Gram-negative enteric bacilli (*E. coli*, *Klebsiella*), anaerobes (*Bacteroides fragilis*), enterococci
Biliary tract surgery	*E. coli*, *Klebsiella*, enterococci, occasionally anaerobes
Head and neck / oropharyngeal surgery	Oral streptococci, anaerobes (*Peptostreptococcus*, *Fusobacterium*), *S. aureus*
Vaginal/genitourinary surgery	Gram-negative enteric bacilli, enterococci, group B streptococcus, anaerobes

2.1 Biofilm

Bacteria adherent to a prosthetic surface (mesh, joint prosthesis, vascular graft, central line) organise into a biofilm — an extracellular polysaccharide matrix that impairs both host immune clearance and antibiotic penetration. This explains why implant-associated infections are notoriously difficult to eradicate with antibiotics alone and usually require device removal or exchange for cure.

3. Host Defence and Risk Factors for Infection

Whether contamination progresses to established infection depends on the balance between the bacterial inoculum/virulence and host defence, summarised classically as: **Risk of infection ∝ (Dose × Virulence) / Host resistance.**

3.1 Patient-Related Risk Factors

- Extremes of age.
- Diabetes mellitus and perioperative hyperglycaemia.
- Obesity (impaired tissue perfusion and higher relative antibiotic under-dosing).
- Malnutrition and hypoalbuminaemia.
- Immunosuppression — corticosteroids, chemotherapy, HIV, transplant recipients.
- Smoking (impaired tissue oxygenation and microvascular perfusion).
- Remote site infection at the time of surgery.
- ASA physical status ≥ III.

3.2 Operative Risk Factors

- Prolonged operative duration (risk rises with each hour beyond the procedure-specific benchmark time).
- Perioperative hypothermia and poor tissue oxygenation.
- Emergency surgery and higher CDC wound class.
- Poor surgical technique — excessive tissue trauma, dead space, haematoma, prolonged retraction.
- Presence of a foreign body/implant.
- Inadequate skin antisepsis or breach of sterile technique.

4. Surgical Site Infections

Surgical site infection (SSI) is the commonest healthcare-associated infection in surgical patients, complicating an estimated 2–5% of all operations and disproportionately increasing length of stay, readmission, reoperation and cost. The CDC/NHSN definitions, used worldwide for surveillance, stratify SSI by depth:

SSI depth	Defining criteria (CDC/NHSN)
Superficial incisional SSI	Involves only skin and subcutaneous tissue, within 30 days of surgery, with purulent drainage, a positive culture from aseptically obtained fluid, or clinical signs of infection with deliberate opening by the surgeon.
Deep incisional SSI	Involves fascia and/or muscle layers, within 30 or 90 days depending on implant status, with purulent drainage from the deep incision, spontaneous dehiscence or deliberate opening with fever/localised pain, or an abscess on imaging/re-operation.
Organ/space SSI	Involves any anatomical part opened or manipulated during surgery other than the incision itself (e.g., intra-abdominal abscess after bowel surgery), with purulent drainage from a drain placed into that space, a positive culture, or an abscess found on imaging or reoperation.

The surveillance window is conventionally 30 days after operation, extended to 90 days when a prosthetic implant has been placed, reflecting the longer latency of implant-associated infection.

5. Prevention of Surgical Site Infection

Current WHO and CDC guidance on SSI prevention (WHO Global Guidelines, and the CDC's “Strategies to Prevent Surgical Site Infections in Acute-Care Hospitals”, most recently updated 2022–2024) organises prevention into bundles applied before, during and after surgery.

5.1 Pre-operative Measures

- Treat any remote infection before elective surgery, and defer elective surgery in the presence of active infection where possible.
- Pre-operative bathing/showering with plain or antiseptic soap.
- Avoid hair removal unless it will interfere with the operation; if required, use clippers immediately before surgery — never razors, which increase SSI risk via microscopic skin trauma.
- Optimise perioperative glycaemic control (target blood glucose generally <180–200 mg/dL) in diabetic and non-diabetic patients alike.
- Screen and decolonise *S. aureus* nasal carriage (mupirocin ± chlorhexidine wash) before cardiac and orthopaedic implant surgery in selected protocols.
- Smoking cessation counselling where time permits.

5.2 Intra-operative Measures

- Administer antimicrobial prophylaxis within 60 minutes before incision (see Chapter 6).
- Alcohol-based chlorhexidine skin antisepsis in preference to povidone-iodine where not contraindicated.
- Maintain perioperative normothermia (core temperature ≥ 36°C) and adequate tissue oxygenation (supplemental FiO■ intraoperatively and briefly postoperatively for selected procedures).
- Meticulous aseptic technique, minimising tissue trauma, dead space and unnecessary electrocautery of skin edges.
- Use wound protectors/wound edge protection devices for contaminated abdominal surgery, and change gloves and instruments before fascial/skin closure in contaminated cases.
- Irrigate contaminated wounds; routine intraperitoneal antibiotic irrigation is not recommended over antibiotic prophylaxis alone.

5.3 Post-operative Measures

- Keep a sterile dressing on a primarily closed incision for at least 24–48 hours.
- Do not continue prophylactic antibiotics beyond the closure of the incision for most clean and clean-contaminated procedures — prolonged prophylaxis does not reduce SSI further and promotes resistance.
- Maintain postoperative glycaemic control and normothermia.

6. Antimicrobial Prophylaxis in Surgery

Surgical antimicrobial prophylaxis aims to achieve bactericidal tissue and serum concentrations for the duration of the operation, not to sterilise the wound or treat established infection. The ASHP/IDSA/SHEA/SIS clinical practice guideline for antimicrobial prophylaxis in surgery remains the key reference underpinning the principles below.

6.1 Core Principles

- **Timing:** administer within 60 minutes before skin incision (within 120 minutes for vancomycin or fluoroquinolones, given their longer infusion times), so that adequate tissue levels are present at the time of the first incision.
- **Agent selection:** chosen to cover the expected pathogens for that procedure (see Chapter 2), balancing efficacy against the risk of promoting resistance — typically a first- or second-generation cephalosporin (e.g., cefazolin) for most clean and clean-contaminated procedures.
- **Weight-based dosing:** higher doses are used in obese patients (e.g., cefazolin 3 g instead of 2 g above a defined weight threshold) to achieve adequate tissue concentration.
- **Intraoperative redosing:** repeat the dose if the operation exceeds two half-lives of the agent (e.g., roughly every 4 hours for cefazolin) or if there is major blood loss (>1500 mL).
- **Duration:** a single pre-operative dose is sufficient for most procedures; there is no benefit to continuing prophylaxis after skin closure, even in the presence of a drain.
- **Route:** intravenous administration is standard; oral/mechanical bowel preparation combined with oral non-absorbable antibiotics is recommended before elective colorectal resection in patients who can tolerate it.

7. Soft Tissue Infections: Cellulitis, Erysipelas and Abscess

7.1 Cellulitis and Erysipelas

Cellulitis is a spreading infection of the dermis and subcutaneous tissue, typically caused by β-haemolytic streptococci or *S. aureus*, presenting with erythema, warmth, tenderness and ill-defined margins, usually without a discrete fluctuant collection. **Erysipelas** is a more superficial variant, confined to the upper dermis and lymphatics, classically caused by *Streptococcus pyogenes*, with a sharply demarcated, raised advancing edge. Both are managed with appropriate oral or intravenous antibiotics according to severity, without surgical drainage unless an underlying abscess is identified.

7.2 Cutaneous Abscess

A localised collection of pus requires **incision and drainage** as the primary treatment — antibiotics alone are inadequate once a discrete abscess has formed, since antibiotic penetration into an avascular pus collection is poor. Adjunctive antibiotics are indicated for abscesses with surrounding cellulitis, systemic signs of infection, immunosuppression, or in high-risk anatomical locations (face, hands, perineum). Simple incision and drainage alone is usually sufficient for an uncomplicated, localised abscess in an immunocompetent patient.

8. Necrotising Soft Tissue Infections

Necrotising soft tissue infections (NSTIs) are rapidly progressive, life-threatening infections of the fascia and subcutaneous tissue, with or without muscle involvement, characterised by pain disproportionate to clinical findings, rapid spread, and systemic toxicity. Mortality remains high (approximately 20–30% in most series) and is directly related to delay in diagnosis and surgical debridement.

8.1 Classification

Type	Microbiology and typical setting
Type I	Polymicrobial — mixed aerobic and anaerobic organisms; the commonest type, often in diabetic, elderly or immunocompromised patients.
Type II	Monomicrobial — classically group A streptococcus (*S. pyogenes*), often with *S. aureus* co-infection; can affect healthy young patients and progresses extremely rapidly.
Type III	Gram-negative monomicrobial, including marine organisms (e.g., *Vibrio vulnificus*) associated with seawater or seafood exposure.
Type IV	Fungal (e.g., *Candida*, Zygomycetes), typically in severely immunocompromised or burn patients.

8.2 Special Anatomical Forms

- **Fournier's gangrene** — necrotising fasciitis of the perineal, genital and perianal region, often arising from an anorectal, urogenital or cutaneous source, disproportionately affecting diabetic and immunosuppressed men.
- **Clostridial myonecrosis (gas gangrene)** — a fulminant, toxin-mediated infection of muscle caused by *Clostridium perfringens* and related species, producing gas in tissue, profound systemic toxicity and rapid muscle necrosis; classically follows deep, devitalised, contaminated wounds.

8.3 Diagnosis

Diagnosis is primarily clinical; imaging or scoring systems should never delay surgical exploration when clinical suspicion is high. Hallmark features include pain out of proportion to examination findings, rapidly spreading erythema with indistinct margins, skin bullae or necrosis, crepitus (gas in tissue), and systemic toxicity disproportionate to the apparent extent of skin involvement. The **LRINEC score** (Laboratory Risk Indicator for Necrotising Fasciitis — based on CRP, total white cell count, haemoglobin, sodium, creatinine and glucose) can support risk stratification, but a low score does not reliably exclude NSTI and must never override clinical judgement.

8.4 Management

Management follows three simultaneous priorities: **early aggressive surgical debridement** of all necrotic tissue down to viable, bleeding margins — with a low threshold for re-exploration at 24–48 hours and repeated debridement until the wound is clean; **broad-spectrum empirical antibiotics** covering Gram-positive, Gram-negative and anaerobic organisms (with clindamycin added for its antitoxin effect against toxin-producing streptococci and clostridia); and **haemodynamic resuscitation and critical care support**, since these patients are frequently septic or in septic shock at presentation. Delay in surgical debridement is the single strongest modifiable predictor of mortality.

9. Intra-Abdominal and Deep Space Infections

9.1 Peritonitis

Primary (spontaneous) peritonitis occurs without a breach in the gastrointestinal tract, typically in patients with ascites (e.g., cirrhosis) or on peritoneal dialysis, and is usually monomicrobial — managed medically with antibiotics rather than surgery. **Secondary peritonitis** follows perforation, anastomotic leak or gangrene of an intra-abdominal viscus and is typically polymicrobial, requiring both source control (surgical or radiological) and antibiotics. **Tertiary peritonitis** describes persistent or recurrent intra-abdominal infection after apparently adequate treatment of secondary peritonitis, often with more resistant or opportunistic organisms, reflecting a dysregulated host response.

9.2 Intra-Abdominal Abscess

A localised collection walled off by omentum, bowel loops or adjacent viscera, most often following anastomotic leak, perforated appendicitis or diverticulitis, or postoperative contamination. Diagnosis is by CT (the most sensitive modality) or ultrasound, and treatment is source control — most often percutaneous image-guided drainage where anatomically feasible, reserving open surgical drainage for multiloculated collections, inaccessible location, or failure of percutaneous drainage — combined with targeted antibiotics.

9.3 Principles of Source Control

Source control — the physical measures taken to eliminate a focus of infection, control ongoing contamination, and restore anatomical and functional integrity — is the single most important determinant of outcome in intra-abdominal and other surgical infections, and antibiotics alone cannot substitute for it. Source control may include drainage of an abscess, resection of infected or necrotic tissue, debridement, or diversion (e.g., stoma formation) when primary repair or anastomosis is unsafe in a heavily contaminated field.

10. Surgical Sepsis and Septic Shock

Sepsis is defined (Sepsis-3 consensus definitions) as life-threatening organ dysfunction caused by a dysregulated host response to infection, identified clinically by an acute increase of ≥2 points in the Sequential Organ Failure Assessment (SOFA) score. **Septic shock** is a subset of sepsis with profound circulatory, cellular and metabolic abnormalities, identified by the need for vasopressors to maintain a mean arterial pressure ≥65 mmHg together with a serum lactate >2 mmol/L despite adequate fluid resuscitation. The bedside **qSOFA** score (respiratory rate ≥22/min, altered mentation, systolic blood pressure ≤100 mmHg) helps flag patients outside critical care who warrant closer assessment, though it is a prompt for further evaluation rather than a diagnostic or screening tool in its own right.

10.1 Management Principles

The most recent Surviving Sepsis Campaign international guidelines (2026 update, building on the 2021 guideline framework) continue to anchor management around early recognition, prompt and appropriate empirical antibiotics, timely source control, and physiologically guided resuscitation:

- Measure serum lactate; remeasure to guide resuscitation if the initial value is elevated.
- Obtain blood cultures before starting antibiotics, provided this does not significantly delay antibiotic administration.
- Administer broad-spectrum empirical antibiotics as soon as possible after recognition of sepsis or septic shock — every hour of delay is associated with incrementally worse survival.
- Begin resuscitation with balanced crystalloid for hypotension or lactate ≥4 mmol/L.
- Start norepinephrine as the first-line vasopressor if blood pressure is not restored with fluid resuscitation, targeting a mean arterial pressure of 65 mmHg.
- Identify and achieve source control as soon as anatomically and physiologically feasible — for a surgically controllable source, this should not be delayed beyond what is required to adequately resuscitate the patient for theatre.

10.2 The Surgeon's Role

For the surgical patient, timely source control is often the single intervention with the greatest impact on survival, and its urgency must be balanced against — but should rarely be delayed indefinitely by — ongoing resuscitation; damage-control principles (rapid, abbreviated surgery focused on controlling contamination, followed by staged definitive repair once physiology has been restored) are frequently appropriate in the physiologically unstable septic surgical patient.

11. Multidrug-Resistant Organisms in Surgical Patients

Organism	Surgical relevance
MRSA (Methicillin-resistant *S. aureus*)	Surgical site and soft tissue infection; screening and decolonisation before elective implant surgery; vancomycin or linezolid for empirical/directed therapy.
VRE (Vancomycin-resistant enterococcus)	Intra-abdominal and line-related infection in critically ill/prolonged-stay surgical patients; linezolid or daptomycin for treatment.
ESBL-producing Enterobacterales	Intra-abdominal and urinary infection; typically resistant to penicillins and cephalosporins, requiring carbapenem therapy.
CRE (Carbapenem-resistant Enterobacterales)	Increasingly reported in healthcare-associated intra-abdominal infection; management requires specialist microbiology input and agents such as ceftazidime-avibactam or other novel β-lactam/β-lactamase-inhibitor combinations.

Risk factors for MDR organism involvement include recent hospitalisation, prior antibiotic exposure, healthcare-associated (versus community-acquired) infection, and residence in a long-term care facility — these should inform empirical antibiotic choice pending culture results, in line with local antibiogram data.

12. Antimicrobial Stewardship in Surgery

Antimicrobial stewardship — the coordinated set of interventions to ensure antibiotics are used appropriately — is now a core surgical responsibility, not solely an infectious diseases function. Key surgical stewardship principles include:

- Confining prophylaxis to the peri-incisional period only, as detailed in Chapter 6.
- Selecting the narrowest effective empirical agent for an established infection, guided by local antibiogram data, then **de-escalating** promptly once culture and sensitivity results are available.
- Limiting the duration of therapy for intra-abdominal infection to a fixed, evidence-based course (commonly 4 days) once adequate source control has been achieved, rather than continuing until clinical parameters fully normalise.
- Distinguishing colonisation/contamination from true infection before starting antibiotics — not every positive culture requires treatment.
- Regular multidisciplinary review of antibiotic use on surgical wards ("antibiotic ward rounds") with input from clinical microbiology/infectious diseases.

13. Asepsis, Antisepsis and Sterilisation

Asepsis refers to practices that prevent contamination by pathogenic organisms; **antisepsis** refers to the application of an agent to living tissue to reduce the number of microorganisms; **disinfection** reduces the microbial load on inanimate objects to a safe level; and **sterilisation** is the complete elimination or destruction of all forms of microbial life, including bacterial spores.

13.1 The Spaulding Classification

Instrument reprocessing requirements are guided by intended use: **critical items** (entering sterile tissue or the vascular system, e.g., surgical instruments) require sterilisation; **semi-critical items** (contacting mucous membranes or non-intact skin, e.g., endoscopes) require at minimum high-level disinfection; and **non-critical items** (contacting only intact skin, e.g., blood pressure cuffs) require only low-level disinfection.

13.2 Methods of Sterilisation

Method	Notes
Steam (autoclave)	Moist heat under pressure (typically 121°C/15 psi/15 min, or 134°C/30 psi/3 min); cheap, reliable, first-line for heat-stable instruments.
Ethylene oxide gas	For heat- and moisture-sensitive equipment; effective but requires prolonged aeration to eliminate toxic residue.
Hydrogen peroxide gas plasma	Low-temperature method for heat-sensitive items; rapid cycle, no toxic residue.
Dry heat	For items damaged by moisture (e.g., oils, powders); requires higher temperature and longer exposure than steam.
Ionising (gamma) radiation	Industrial-scale sterilisation of single-use disposable instruments and consumables.

13.3 Hand Hygiene and Skin Antisepsis

Surgical hand antisepsis (traditional scrub or alcohol-based hand-rub technique) and patient skin antisepsis before incision are foundational, low-cost, high-impact SSI prevention measures. Alcohol-based chlorhexidine preparations achieve faster and more sustained antimicrobial action than aqueous povidone-iodine and are generally preferred where not contraindicated (e.g., near the eyes or on neonatal skin, where povidone-iodine may be preferred).

14. Prosthetic and Implant-Related Infections

Infection involving a prosthetic implant is disproportionately difficult to treat because of biofilm formation on the device surface (Section 2.1), and cure generally requires removal or exchange of the infected hardware in addition to antibiotics and debridement.

- **Mesh infection** (hernia repair) — superficial infection may be managed with drainage and antibiotics with mesh preservation attempted where feasible, but deep infection involving a non-absorbable synthetic mesh usually requires mesh excision.
- **Vascular graft infection** — a serious, high-mortality complication requiring multidisciplinary management; options include graft excision with extra-anatomical bypass, in-situ graft replacement with antibiotic-impregnated or biological conduit, and prolonged targeted antibiotic therapy.
- **Periprosthetic joint infection** — classified as early (typically due to intraoperative contamination), delayed, or late/haematogenous; management ranges from debridement with implant retention (for early infection with a stable, well-fixed implant) to staged revision arthroplasty.
- **Cardiac implantable device and mesh graft infections** follow similar principles — biofilm-forming organisms (commonly coagulase-negative staphylococci and *S. aureus*) necessitate device removal for reliable cure in most established infections.

15. Special Clinical Situations

The Immunocompromised Surgical Patient

Patients on corticosteroids, chemotherapy, biologic agents, or with diabetes, chronic kidney disease, cirrhosis or HIV mount a blunted inflammatory response — clinical signs of infection (fever, leucocytosis, localised findings) may be subtle or absent despite serious underlying sepsis, requiring a lower threshold for imaging and source control, and often broader empirical antimicrobial cover pending culture results.

Tetanus Prophylaxis in Wound Management

Every traumatic wound should prompt review of tetanus immunisation status. Clean, minor wounds in a fully immunised patient (booster within 10 years) need no further prophylaxis; tetanus-prone wounds (contaminated, devitalised, puncture, or delayed presentation >6 hours) in an inadequately immunised patient require both tetanus toxoid-containing vaccine and human tetanus immunoglobulin, in addition to thorough surgical debridement — since debridement of devitalised tissue is itself a critical part of tetanus prevention.

Diabetic Foot Infection

A distinct and common category of surgical infection, ranging from superficial cellulitis to deep abscess, necrotising infection and osteomyelitis; management follows the same core principles of source control (debridement, drainage, or amputation when required), targeted antibiotics guided by deep tissue/bone culture, and addressing the underlying vascular and glycaemic contributors to poor healing.

16. Summary of Current Guidelines and Evidence

- **CDC “Strategies to Prevent Surgical Site Infections in Acute-Care Hospitals” (updated 2022–2024)** and the **WHO Global Guidelines for the Prevention of Surgical Site Infection** remain the primary evidence-based references for SSI prevention bundles.
- **ASHP/IDSA/SHEA/SIS Clinical Practice Guidelines for Antimicrobial Prophylaxis in Surgery** remains the key reference for prophylactic antibiotic selection, timing and dosing.
- **Sepsis-3 consensus definitions** (2016) remain the standard definitions for sepsis and septic shock, operationalised via SOFA/qSOFA.
- **Surviving Sepsis Campaign International Guidelines 2026** — the current update of the Surviving Sepsis Campaign framework — continues to emphasise early recognition, prompt antibiotics, physiologically guided resuscitation, and timely source control.
- **World Society of Emergency Surgery (WSES)** guidance on necrotising soft tissue infection and complicated intra-abdominal infection reinforces early aggressive debridement and time-limited antibiotic courses after adequate source control.

Key Points for Revision

HIGH-YIELD SUMMARY

SSI risk rises with CDC wound class: clean < clean-contaminated < contaminated < dirty/infected.

Prophylactic antibiotics: give within 60 minutes of incision, redose intraoperatively for long cases or major blood loss, and stop within 24 hours — prolonged prophylaxis adds no benefit.

A discrete abscess needs drainage, not antibiotics alone — pus under pressure will not be sterilised by systemic therapy.

Pain out of proportion to signs + rapidly spreading erythema + systemic toxicity = necrotising soft tissue infection until proven otherwise — operate early; do not wait for imaging or a low LRINEC score to reassure you.

Source control is the decisive intervention in intra-abdominal sepsis and surgical sepsis generally — antibiotics support, but do not replace, drainage/debridement/resection.

Sepsis = life-threatening organ dysfunction from infection (SOFA rise ≥2); septic shock adds vasopressor-dependent hypotension with lactate >2 mmol/L despite fluids.

Biofilm on prosthetic material explains why implant infections usually need device removal for cure, not antibiotics alone.

Antimicrobial stewardship is a surgical responsibility: narrow the spectrum once cultures return, and stop therapy once source control is adequate and a fixed course is complete.

This review is intended as a structured revision resource for postgraduate trainees preparing for surgical postgraduate examinations (e.g., FCPS, MS, MRCS and equivalent), and as a bedside/ward reference. It should be read alongside primary guideline documents and institutional/local antibiogram-based protocols, which take precedence in individual clinical decision-making.

Further Reading and Key Sources

The following primary sources informed this review and are recommended for deeper study:

1. Berríos-Torres SI, Umscheid CA, Bratzler DW, et al. Centers for Disease Control and Prevention Guideline for the Prevention of Surgical Site Infection. *JAMA Surg.* (with subsequent CDC updates through 2024).

2. World Health Organization. Global Guidelines for the Prevention of Surgical Site Infection, 2nd edition.

3. Bratzler DW, Dellinger EP, Olsen KM, et al. Clinical Practice Guidelines for Antimicrobial Prophylaxis in Surgery. *Am J Health Syst Pharm* (ASHP/IDSA/SHEA/SIS).

4. Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). *JAMA.* 2016;315(8):801-810.

5. Evans L, Rhodes A, Alhazzani W, et al. Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2026. *Crit Care Med / Intensive Care Med.*

6. Sartelli M, Coccolini F, et al. World Society of Emergency Surgery (WSES) guidelines for necrotising soft tissue infections and complicated intra-abdominal infections.

7. Wong CH, Khin LW, Heng KS, et al. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score. *Crit Care Med.* 2004;32(7):1535-1541.

8. Williams N, O'Connell PR, McCaskie AW, eds. *Bailey & Love's Short Practice of Surgery*, 28th ed. Boca Raton: CRC Press.
