

3.1 General guidelines for the rational use of antibiotics and surgical prophylaxis

Antimicrobial therapy differs from every therapy so far in this subject because its target evolves, its benefit is shared and its harm is communal: resistance selected in one patient is inherited by the next. The rational use guidelines that open the infectious units are therefore stated as principles from which every later section draws. Confirm that infection is present and bacterial before an antibacterial is started, since viral illness, colonisation and non infective inflammation are its great mimics and none of them is an indication. Identify the likely source and the likely organisms from the site, the patient and local resistance patterns, and take cultures and specimens before the first dose wherever the illness allows, because therapy chosen blind stays blind. Choose the agent on the narrowest spectrum that covers the likely organisms, its tissue penetration to the site, allergy history, organ function, pregnancy and interacting medicines, and local formulary guidance built on local resistance data. Give it by the route, and for the duration, that the severity demands, reviewing intravenous therapy for oral switch as the patient recovers, since prolonged courses select resistance and invite *Clostridioides difficile* and fungal overgrowth without adding cure. Review at a defined point against cultures and response: narrow, stop or change on evidence, the stewardship review that turns an empirical start into a rational course. Document indication, agent, review date and planned duration on the prescription itself. Allergy is recorded precisely, since a false penicillin label condemns patients to broader, costlier and less effective therapy for life, and delabelling assessment is stewardship work.

Rational antibiotic use, the stewardship sequence

Confirm bacterial infection, find the source,
judge severity, record true allergy

|
v

Specimens and cultures first where possible,
then empirical therapy from local guidance,
narrowest spectrum covering likely organisms

|
v

Review against cultures and response:
narrow the spectrum, switch route when
recovering, stop where infection is excluded

|
v

Fixed review date and planned duration
documented, course completed or stopped
on evidence, never drifted

Surgical prophylaxis is rational use in its most disciplined form: a single perioperative antibacterial cover, in most procedures, for operations in which wound infection is likely or its consequences grave, chosen against the organisms expected from the operative site, timed so tissue levels are present at incision, repeated only for long operations or major blood loss under protocol, and stopped at wound closure rather than continued as a course, because continued prophylaxis adds resistance and *Clostridioides* risk without protecting the wound. The principles of the general sections that follow are constant: source control, drainage of pus, removal of infected devices and debridement, is treatment no antibiotic replaces.

Rational antibiotic use: the stewardship review



Source control, drainage and device removal are treatment too

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Figure: The stewardship review. An empirical start becomes a rational course only when it is reviewed on evidence.

3.2 Urinary tract infections

Urinary tract infection is bacterial infection of the urinary tract, classified by site into lower tract infection, cystitis of the bladder, and upper tract infection, pyelonephritis of the kidney, and by host into uncomplicated infection in a healthy non pregnant woman and complicated infection in men, pregnancy, obstruction, stones, catheters, diabetes and immune suppression, a classification that decides investigation, agent and duration together. *Escherichia coli* ascending from the perineum through the urethra causes the large majority, with other coliforms, *Proteus* associated with stones, enterococci and *Staphylococcus saprophyticus* in young women completing the usual set; catheter associated infection adds hospital organisms and biofilm biology. The defences the organism overcomes are urine flow, bladder emptying, mucosal immunity and the acidity and osmolality of urine, which is why obstruction, residual urine and catheterisation dominate the risk factors. Cystitis presents with dysuria, frequency, urgency, suprapubic discomfort and cloudy or blood stained urine without fever; pyelonephritis adds loin pain, fever, rigors and vomiting and may progress to the septicemia of Section 3.8. Asymptomatic bacteriuria, bacteria in the urine of a patient without symptoms, is not infection for treatment purposes except in pregnancy and before defined urological procedures, and treating it is the commonest irrational act in this topic. Diagnosis is clinical in typical cystitis, supported by urine dipstick and culture where the picture is atypical, recurrent, in pregnancy, in men or where treatment fails, with imaging reserved for obstruction and complicated disease.

Pharmacotherapy follows the classification. Uncomplicated cystitis is treated with a short course of a urinary antibacterial chosen from local guidance, nitrofurantoin and fosfomycin and trimethoprim among the standard options by local resistance, with simple analgesia and hydration advice, and no routine post treatment culture where symptoms resolve. Pyelonephritis requires a systemic agent with tissue penetration, broader cover, assessment for sepsis and admission where vomiting, pregnancy, comorbidity or severity demands it, and review against culture. Complicated and catheter associated infection is treated only when symptomatic, with the catheter removed or changed, the obstruction relieved and culture directed therapy. Recurrent infection prompts review of behavioural and anatomical causes, vaginal oestrogen in postmenopausal women where appropriate, and prophylactic strategies, continuous or postcoital antibacterial prophylaxis or self start therapy, under guidance and stewardship review. Cranberry products and hydration are adjuncts of limited evidence and are never substitutes for assessment of complicated disease.

3.3 Respiratory tract infections

Respiratory tract infection spans the upper tract, rhinosinusitis, pharyngitis and otitis media, and the lower tract, bronchitis, bronchiolitis and pneumonia, and its first pharmacotherapeutic act is the viral and bacterial separation, since most upper tract infection and acute bronchitis are viral and self limiting and antibacterial therapy for them is irrational use in its commonest community form. Pneumonia is the serious centre of the topic: infection of the lung parenchyma by *Streptococcus pneumoniae* as the leading bacterial cause, *Haemophilus influenzae*, atypical organisms including *Mycoplasma* and *Legionella*, respiratory viruses including influenza of Unit 4, and aspiration and hospital acquired organisms in their settings. Its pathophysiology is alveolar filling by inflammatory exudate as organisms inhaled or aspirated overcome mucociliary and immune defence, producing consolidation, impaired gas exchange and the systemic inflammatory response. Presentation divides by pattern: cough, fever, breathlessness, pleuritic pain and purulent sputum with crackles and consolidation signs in typical pneumonia, a drier, more insidious course in atypical infection, and severity graded by confusion, respiratory rate, blood pressure and age in the standard assessments that decide home care, admission and critical care. Diagnosis combines examination and oxygen saturation with chest imaging confirming consolidation, sputum and blood cultures, viral testing in season, and severity scoring; pharyngitis assessment separates streptococcal disease, the rheumatic fever precursor of Unit 4, from viral sore throat by its clinical pattern.

Pharmacotherapy of pneumonia is empirical antibacterial therapy started promptly in proportion to severity, chosen from local guidance by setting and severity, an oral agent such as amoxicillin for mild community disease, combination or broader therapy including atypical cover as severity rises, hospital acquired pneumonia treated against hospital organisms and local resistance, with oxygen, fluids and sepsis care alongside, review for oral switch, and a defined course lengthened only on evidence. Prevention is pharmacotherapy too: influenza, pneumococcal and other respiratory vaccination in risk groups, and smoking cessation. Sinusitis is managed supportively with analgesia and saline measures, antibacterials reserved for persistent, severe or worsening bacterial patterns; otitis media likewise settles in most children with analgesia and observation, antibacterials for defined age, severity and bilateral patterns under guidance. Acute bronchitis receives no antibacterial in the uncomplicated patient, and its cough is treated by time and honest explanation, the consultation skill this section exists to teach.

3.4 Gastroenteritis

Gastroenteritis is inflammation of the stomach and intestine producing diarrhoea with or without vomiting, abdominal pain and fever, and it links back to the diarrhoea principles of Part I, now read from the infectious side. Its causes are chiefly viral, norovirus and rotavirus prominent, self limiting and highly transmissible, and bacterial, *Salmonella*, *Campylobacter*, *Shigella*, enterotoxigenic and enterohaemorrhagic *Escherichia coli*, *Vibrio cholerae* and *Clostridioides difficile* after antibacterial therapy, with parasites including *Giardia* completing the set. Mechanisms repeat Part I from the organism side: toxin mediated secretion, cholera toxin driving the profuse pure secretory diarrhoea that kills by volume, mucosal invasion producing dysentery with blood, pus and fever, and osmotic and malabsorptive patterns. Presentation is judged first by dehydration, the governing danger in children and older patients, then by the dysentery pattern, severity, outbreak context and host risk. Diagnosis is clinical in most episodes, stool culture and multiplex testing reserved for dysentery, severe or prolonged illness, outbreaks, travel and immune suppression, and public health notification where the organism demands it.

Pharmacotherapy is rehydration first and drugs a distant second, exactly as Part I taught: oral rehydration solution exploiting glucose coupled sodium absorption, intravenous replacement where oral therapy fails or shock threatens. Antibacterials are withheld in most viral and mild bacterial illness, reserved for dysentery patterns, cholera, identified pathogens requiring therapy, severe disease and vulnerable hosts, because

they prolong carriage in some infections and precipitate haemolytic uraemic syndrome risk in enterohaemorrhagic infection; *Clostridioides difficile* infection is treated by stopping the provoking antibacterial and giving its targeted oral therapy under guidance. Antimotility agents have a narrow place in afebrile non bloody diarrhoea in adults and no place in dysentery or in young children. Antiemetics serve fluid tolerance in defined settings. Prevention is hygiene, safe food and water, rotavirus vaccination in childhood, and outbreak infection control, norovirus closing wards by its transmissibility.

3.5 Tuberculosis

Tuberculosis is chronic infection by *Mycobacterium tuberculosis*, an acid fast bacillus whose waxy cell wall underlies its staining, its environmental toughness and much of its drug behaviour. Infection spreads by inhaled droplets from pulmonary disease, establishes a primary focus with hilar nodes, and is then contained by cell mediated immunity in the granuloma, whose caseous centre shelters dormant bacilli as latent infection, or progresses, at once in the young and immune suppressed or years later by reactivation, to active disease. Pulmonary tuberculosis, the infectious and emblematic form, destroys lung by caseous necrosis and cavitation, cavities discharging bacilli into airways and sputum; extrapulmonary disease reaches lymph nodes, pleura, bone, genitourinary tract, meninges of Section 4.1 and, in miliary spread, every organ. Presentation of pulmonary disease is a chronic cough, sputum and haemoptysis, with fever, night sweats and weight loss, the constitutional triad examinations expect, evolving over weeks in a patient with exposure, origin, contact, immune suppression including HIV of Unit 4, diabetes or previous incomplete therapy among the risk factors. Diagnosis is microbiological and urgent for infection control: sputum microscopy for acid fast bacilli, culture as the reference standard, rapid molecular testing that also flags rifampicin resistance, chest imaging showing upper lobe infiltrates and cavitation, and HIV testing offered to every patient; latent infection is identified by tuberculin or interferon gamma testing in contacts and before immunosuppressive therapy.

Pharmacotherapy is combination therapy for months, and its structure is the topic. Active drug susceptible disease is treated with a standard multi drug regimen in two phases in its classical form: an intensive phase combining isoniazid, rifampicin, pyrazinamide and ethambutol, followed by a continuation phase of isoniazid and rifampicin, the combination serving three purposes at once, rapid killing of multiplying bacilli, sterilisation of dormant persisters, pyrazinamide and rifampicin carrying that role, and prevention of resistance, since a bacillus resistant to one drug is killed by the others. Each first line agent carries a signature caution that monitoring is built on: isoniazid hepatitis and peripheral neuropathy prevented by pyridoxine in at risk patients, rifampicin hepatitis, orange discolouration of urine and tears, and enzyme induction reaching contraceptives, anticoagulants, antiretrovirals and many other medicines, ethambutol ocular toxicity with visual monitoring, pyrazinamide hepatitis and arthralgia. Drug resistant disease, multidrug resistant and extensively resistant tuberculosis, requires specialist regimens built from second line agents and newer drugs, longer and harder, and is prevented by the adherence architecture of the standard programme: fixed dose combinations, directly observed or video supported therapy in defined settings, public health supervision, contact tracing and treatment of latent infection in contacts and high risk groups. HIV coinfection is managed jointly, antiretroviral timing and rifampicin interactions planned together, and cure is verified microbiologically, not assumed.

3.6 Malaria

Malaria is infection by *Plasmodium* parasites transmitted by the bite of female *Anopheles* mosquitoes, with five species infecting humans: *Plasmodium falciparum* causing the great majority of deaths, *Plasmodium vivax* and *Plasmodium ovale* forming dormant liver hypnozoites that relapse months or years later, *Plasmodium malariae* running a chronic low grade course, and *Plasmodium knowlesi* as a zoonotic fifth. The life cycle explains both disease and therapy: sporozoites injected with the bite invade hepatocytes and

multiply silently in the liver phase, merozoites then invade red cells and multiply in cycles whose synchronous rupture releases parasite material and produces the periodic fever, while falciparum infected cells adhere to endothelium and sequester in brain, placenta and other organs, producing the severe disease of cerebral malaria, severe anaemia, acidosis, hypoglycaemia and kidney failure. Presentation in the non immune traveller is fever in or after the endemic area, with chills, rigors, sweating, headache and myalgia in cycles that cannot be relied on for diagnosis, and any fever in a returned traveller is malaria until excluded the same day. Diagnosis is parasitological and urgent: microscopic examination of thick and thin blood films speciating the parasite and estimating its density, supported by rapid antigen tests where microscopy is unavailable, with repeat films where suspicion survives a negative first test.

Pharmacotherapy is species and severity specific, and is specialist guided in non endemic settings. Uncomplicated falciparum malaria is treated with an artemisinin based combination therapy, pairing a rapidly acting artemisinin derivative that clears parasites faster than any older class with a longer acting partner drug that removes the remainder and protects against resistance. Severe falciparum malaria is a medical emergency treated with parenteral artesunate, supportive critical care, and exchange of the failing organs of Section 3.8, followed by a complete oral course when the patient can swallow. Non falciparum malaria is treated with chloroquine where the species and region remain sensitive, or an artemisinin combination, and vivax and ovale infection adds radical cure of the liver hypnozoites by primaquine or tafenoquine after glucose 6 phosphate dehydrogenase testing, because those agents haemolyse deficient red cells, the Part I haemolysis mechanism returning as a prescribing gate. Quinine retains defined places with its hypoglycaemia and cinchonism cautions. Prevention completes the topic: mosquito avoidance, chemoprophylaxis chosen by destination resistance for the traveller and continued after return by its schedule, intermittent preventive treatment in pregnancy and childhood in endemic programmes, and the malaria vaccines now entering endemic childhood programmes, presented as partial protection alongside, never instead of, the older measures.

3.7 Bacterial endocarditis

Bacterial endocarditis, infective endocarditis in current usage, is infection of the endocardial surface of the heart, usually of a valve, native or prosthetic, by microorganisms circulating in the blood, forming vegetations of platelets, fibrin, organisms and inflammatory cells that destroy valve tissue and embolise to distant organs. Its substrate is a susceptible surface and a bacteraemia to seed it: damaged or rheumatic valves of Section 4.3, congenital lesions, prosthetic valves and implanted devices, degenerative valve disease of older patients, and the right sided valves of people who inject drugs; its organisms divide by tempo, Staphylococcus aureus acute and destructive on normal valves, viridans streptococci subacute on damaged valves after dental bacteraemia, enterococci from genitourinary and gut sources, coagulase negative staphylococci on prosthetic material, and culture negative disease from fastidious organisms and prior antibacterials. Presentation follows that tempo: subacute disease as weeks of fever, malaise, weight loss and a new or changed murmur with the peripheral stigmata of immune and embolic phenomena, splinter haemorrhages, Roth spots, Osler nodes and Janeway lesions in the classical teaching set, and acute disease as fulminant sepsis with valve destruction and heart failure. Diagnosis rests on the Duke criteria in their modern form, combining repeated blood cultures taken before antibacterials, echocardiography seeking vegetations, abscess and new regurgitation, transthoracic first and transoesophageal where suspicion survives, and the clinical phenomena, because therapy is long and surgery is major and the diagnosis must be secure.

Pharmacotherapy is prolonged, bactericidal, intravenous antibacterial therapy in hospital, begun empirically after cultures in the acutely ill against staphylococci, streptococci and enterococci by local guidance, then narrowed to the cultured organism and its sensitivities for a course measured in weeks from the first

negative culture, with combination synergy, an aminoglycoside partner in defined streptococcal and enterococcal regimens, used selectively against its kidney and ear toxicities of Section 2.9. Prosthetic valve and staphylococcal regimens add their specific agents under specialist direction. Surgery is co therapy, not failure: valve replacement or repair for heart failure from valve destruction, uncontrolled infection and abscess, and prevention of embolism in defined large vegetations. Prophylaxis has narrowed in modern guidance to antibacterial cover for the highest risk patients at defined dental and other procedures, a deliberate stewardship retreat from the broad prophylaxis of older teaching, and dental hygiene in at risk patients is prevention of equal standing.

3.8 Septicemia

Septicemia, taught in current usage within the sepsis family, is the presence and effect of microorganisms and their products in the bloodstream producing a dysregulated host response with organ dysfunction: sepsis as infection with that dysfunction, septic shock as its circulatory failure state requiring vasopressor support with persisting cellular and metabolic abnormality. It is a syndrome, not a single infection, and its sources are the infections of this and the next unit: pneumonia first, then urinary, abdominal, skin and soft tissue, device and line infection, meningitis and endocarditis, with Gram negative, Gram positive and fungal organisms all represented and the immune suppressed and very young and old at greatest risk. The pathophysiology is the host response itself: microbial patterns trigger cytokine release, endothelial injury and capillary leak, vasodilation and maldistributed flow, activation of coagulation toward disseminated intravascular coagulation of Part I, and mitochondrial and organ dysfunction in brain, kidney, lung, liver and heart together. Presentation is fever or hypothermia with tachycardia, tachypnoea, hypotension and altered mentation, warm vasodilated shock early and cold hypodynamic shock late, with lactate rising as the marker of tissue hypoperfusion and severity. Diagnosis is recognition under time pressure: cultures of blood and source before antibacterials where this does not delay them, lactate and organ function measurement, severity scoring, and an active hunt for the source and for any focus needing drainage or removal.

Pharmacotherapy is the sepsis bundle delivered as one act: broad spectrum intravenous antibacterials covering the likely source and local resistance, given promptly after cultures in the shocked patient, fluid resuscitation against the leak and vasodilation, vasopressor therapy, noradrenaline first, where fluids do not restore perfusion, oxygen and ventilatory support, source control by surgery, drainage or device removal as urgently as any drug, and organ support including the dialysis of Section 2.8. Therapy is then de escalated on cultures and response with the discipline of Section 3.1, stress ulcer and thrombosis prophylaxis and glucose control support the critical care, corticosteroids hold a defined place in refractory shock under protocol, and every hour of the early phase is taught as carrying outcome weight, which is why sepsis recognition by ward pharmacists reviewing fever, new confusion and rising early warning scores is part of this subject.

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