

4.1 Meningitis

Meningitis is inflammation of the meninges, the membranes surrounding brain and spinal cord, most seriously by bacterial infection of the cerebrospinal fluid in the subarachnoid space. Bacterial causes divide by age and host: *Streptococcus pneumoniae* and *Neisseria meningitidis* across older children and adults, group B streptococci, *Escherichia coli* and *Listeria* in the newborn period, *Listeria* again in older and immune suppressed adults, and *Haemophilus influenzae* type b now uncommon where vaccination holds; viral meningitis, enteroviruses and herpes simplex prominent, is commoner, usually benign and self limiting, except herpes encephalitis, which is a brain emergency of its own. Organisms reach the meninges by blood from nasopharyngeal colonisation or by direct extension, multiply in a space poor in antibody and complement, and the inflammatory response to their components, not the organisms alone, produces raised intracranial pressure, cerebral oedema, vascular injury and the neurological damage that survivors carry. Presentation is the classical triad in its complete form in a minority: fever, neck stiffness and altered consciousness, with headache, photophobia, vomiting, and in meningococcal disease the non blanching purpuric rash of accompanying septicemia; infants present non specifically with poor feeding, irritability and a bulging fontanelle. Diagnosis is lumbar puncture where safe, cerebrospinal fluid showing the bacterial pattern of turbid fluid, high neutrophil count, high protein and low glucose against viral and tuberculous contrasts, with Gram stain, culture and molecular testing, and blood cultures and scanning sequenced so that imaging never delays antibacterial therapy in the deteriorating patient.

Pharmacotherapy begins before the diagnosis is complete, because delay costs brain: empirical intravenous antibacterials covering the likely organisms for the age and host, a third generation cephalosporin as the usual backbone with additions for *Listeria* risk and local resistance under guidance, given as soon as bacterial meningitis is seriously suspected. Adjunctive corticosteroid therapy with the first antibacterial doses reduces hearing loss and neurological injury in defined bacterial causes and settings and is protocol guided. Herpes encephalitis cover with aciclovir is added wherever encephalitis is in the picture until excluded. Supportive care manages pressure, seizures of Section 1.2, fluid and electrolyte disturbance including the syndrome of inappropriate antidiuretic hormone secretion, and critical care where consciousness falls. Prevention is vaccination, pneumococcal, meningococcal and *Haemophilus* programmes, and chemoprophylaxis of close contacts in meningococcal disease under public health direction, with the exposed household treated as part of the case.

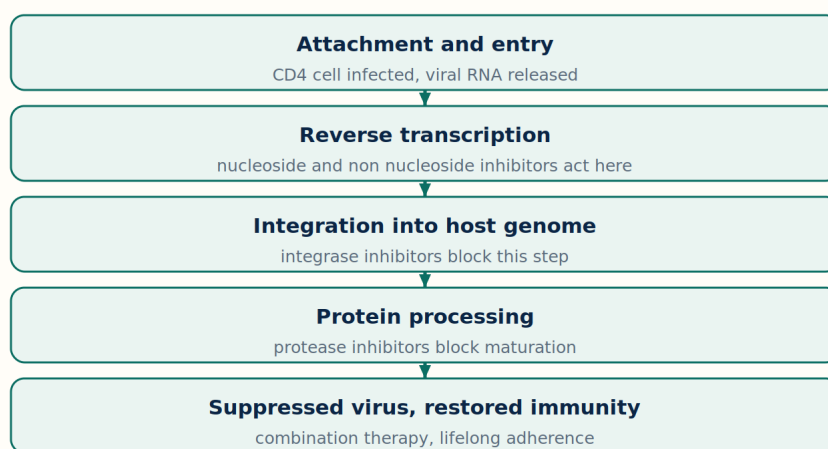
4.2 HIV and opportunistic infections

Human immunodeficiency virus infection is chronic infection by a retrovirus that integrates into the genome of its target cells, the CD4 T lymphocytes whose progressive destruction is the disease: untreated infection passes through acute seroconversion illness, a long clinically latent phase of slow immune decline, and advanced disease, AIDS in the staging language, defined by the opportunistic illnesses that a competent immune system prevents. Transmission is sexual, by blood including shared injecting equipment, and vertical from mother to child in pregnancy, birth and breastfeeding, and not by casual contact, a fact counselling must state without hedging. The opportunistic infections are taught as a pattern matched to immune depth: *Pneumocystis pneumonia* as the emblematic AIDS defining lung infection, toxoplasma encephalitis, cryptococcal meningitis extending Section 4.1, cytomegalovirus retinitis and colitis,

disseminated *Mycobacterium avium* disease, oesophageal candidiasis of Section 4.7, and tuberculosis of Section 3.5 at any immune level, together with the malignancies of immune failure, Kaposi sarcoma and lymphomas bridging to Unit 5. Diagnosis is by HIV antibody and antigen testing with consent and counselling, confirmed and staged by viral load and CD4 count and screened for coinfections including hepatitis and tuberculosis; every positive test is also the start of partner, prevention and retention care.

Pharmacotherapy is antiretroviral therapy, combination treatment that suppresses viral replication to undetectable levels, restores immune function and, so suppressed, prevents sexual transmission, the treatment as prevention principle. Regimens combine agents from classes attacking successive steps of the viral life cycle, nucleoside reverse transcriptase inhibitors as the backbone pair, with an integrase strand transfer inhibitor as the usual modern anchor class, or a non nucleoside reverse transcriptase inhibitor or a boosted protease inhibitor in defined roles; the Unit 5 chemotherapy logic of combination against resistance applies here exactly. Therapy is lifelong, adherence is its whole effectiveness, and its teaching burdens are interactions, the boosted regimens above all inhibiting drug metabolism widely, immune reconstitution inflammatory syndrome as recovering immunity unmasks occult infection, and metabolic and bone and kidney monitoring by agent. Opportunistic infections are prevented by prophylaxis at defined immune depths under guidance, treated specifically when established, *Pneumocystis* therapy and its corticosteroid adjunct in hypoxic disease the standard example, and prevention extends to pre exposure and post exposure prophylaxis for the uninfected at risk and to antiretroviral cover preventing vertical transmission. Vaccination of the immune suppressed follows its own careful rules, live vaccines avoided at low immunity.

HIV life cycle and antiretroviral classes



Undetectable virus does not transmit sexually: treatment is prevention

© 2026 PharmacoLearn, pharmacolearn.fyi

Figure: The HIV life cycle as a class map. Combination therapy blocks successive steps so resistance cannot escape.

4.3 Rheumatic fever

Rheumatic fever is an immune mediated multisystem illness following group A streptococcal pharyngitis, in which antibodies and T cells raised against streptococcal antigens cross react with host tissues, molecular mimicry, injuring heart, joints, skin and brain after a latent interval; it is therefore a post infective immune disease, not an infection of those organs, and no streptococci are found in the inflamed joint or valve. Its burden falls on children and young adults in crowded, low income settings, and its importance is its cardiac sequel: acute carditis involving endocardium, myocardium and pericardium heals by scarring and deforming the valves, mitral first, producing rheumatic heart disease, the chronic stenosis and regurgitation that fills cardiology and endocarditis risk lists for life, female patients carrying much of its burden including in pregnancy. Presentation is organised by the Jones criteria in their modern form: major manifestations of

carditis, a migratory polyarthritis of large joints that answers strikingly to anti inflammatory therapy, Sydenham chorea, erythema marginatum and subcutaneous nodules, with minor manifestations of fever, raised inflammatory markers, arthralgia and conduction prolongation, all requiring evidence of preceding streptococcal infection. Diagnosis is that criteria synthesis with echocardiography detecting subclinical carditis, and its mimics, viral arthritis, reactive arthritis and infective endocarditis among them, excluded.

Pharmacotherapy has three distinct acts. Eradicate the streptococcus: a full course of penicillin class therapy on diagnosis, even though the fever itself is immune, to remove carriage and antigen drive, with macrolide alternatives in true allergy. Suppress the inflammation: aspirin and the non steroidal class for arthritis and mild carditis, corticosteroids for severe carditis with heart failure under specialist direction, carbamazepine or other agents for disabling chorea in defined cases, and heart failure therapy of Part I where valves fail acutely. Prevent recurrence, the act that changes the lifetime outcome: long term secondary prophylaxis with regular penicillin, intramuscular benzathine penicillin as the most reliable form, oral therapy where injections are refused against its adherence weakness, continued for years scaled to cardiac involvement under guidance, because every recurrence adds valve damage. Primary prevention closes the loop back to Section 3.3: adequate antibacterial treatment of streptococcal pharyngitis in at risk populations, and the social determinants, crowding and access, named honestly as the disease true causes.

4.4 Dengue fever

Dengue fever is acute infection by dengue viruses, four related serotypes transmitted by Aedes mosquitoes, ranging from undifferentiated fever through classical dengue to severe dengue with plasma leakage, haemorrhage and organ impairment. Its pathophysiology turns on two mechanisms: direct viral and immune injury, and antibody dependent enhancement, in which antibodies from a previous infection by one serotype bind a second serotype without neutralising it and escort it into cells, worsening the second illness, which is why severe dengue concentrates in second infections and why its vaccine use is restricted by prior infection status. Capillary leak is the dangerous physiology: in the critical phase around defervescence, plasma leaves the circulation into pleural and peritoneal spaces, haematocrit rises as volume falls, platelets drop and bleeding ranges from skin and mucosal oozing to major haemorrhage, and shock follows leak unreplaced. Presentation divides into phases for examination: the febrile phase of high fever, severe headache, retro orbital pain, muscle and joint pains earning its backbone name, flushing and rash; the critical phase of warning signs, abdominal pain, persistent vomiting, mucosal bleeding, lethargy and rising haematocrit with falling platelets; and recovery with reabsorption and a convalescent rash. Diagnosis is clinical and epidemiological in season and endemic settings, supported by viral antigen and molecular testing early and antibody testing later, full blood counts tracking the leak, and ultrasound showing its fluid collections; malaria of Section 3.6 and leptospirosis and typhoid are its standing differentials.

Pharmacotherapy is supportive, and its restraint is the teaching point: there is no antiviral of established benefit, and management is fluid therapy matched to phase and leak, oral hydration in uncomplicated fever, intravenous crystalloid titrated carefully in warning sign and shock states, and blood products for significant haemorrhage under guidance, with monitoring of haematocrit, platelets, perfusion and urine output as the treatment chart. Paracetamol is the antipyretic and analgesic of choice; non steroidal anti inflammatory drugs and aspirin are avoided for their platelet and bleeding effects, and intramuscular injections and unnecessary invasive procedures are avoided in the thrombocytopenic phase. Corticosteroids and prophylactic platelet transfusion without bleeding are not standard care. Prevention is vector control, personal protection in Aedes daytime biting hours, and the vaccines now licensed with serostatus restrictions following the enhancement lesson above.

4.5 H1N1

H1N1 in the syllabus names influenza A H1N1, and its teaching is the influenza teaching of this subject: acute infection by influenza A viruses whose surface haemagglutinin and neuraminidase antigens define subtypes and whose antigenic drift drives seasonal epidemics and shift drives pandemics, the 2009 pandemic virus being an H1N1 lineage now circulating seasonally. The virus infects respiratory epithelium, destroys mucociliary defence and provokes the abrupt systemic illness that distinguishes influenza from the common cold: sudden fever, headache, myalgia, prostration and dry cough with sore throat, over days, with recovery in most and danger concentrated in pregnancy, chronic heart, lung and metabolic disease, immune suppression and the extremes of age. Its complications are primary viral pneumonia progressing to respiratory failure, secondary bacterial pneumonia, staphylococcal superinfection classically, exacerbations of asthma and COPD of Part I, myocarditis and the decompensation of every chronic disease it visits. Diagnosis in season is clinical, confirmed by molecular testing from respiratory samples where the result changes management, isolation or outbreak control, and severity is judged by oxygen saturation, respiratory rate and the sepsis assessments of Section 3.8 where pneumonia supervenes.

Pharmacotherapy centres on the neuraminidase inhibitors, oseltamivir orally and zanamivir and peramivir in defined settings, which block viral release from infected cells: their benefit is greatest started early in illness and in hospitalised, severe and high risk patients treatment is not withheld for delay, under current guidance, while the healthy mild case is managed supportively with antipyretics, fluids and rest and honest advice on course. The older adamantane class is obsolete against circulating resistance and survives as a stewardship history lesson. Antibacterials treat only the bacterial complications, by Section 3.3 principles, and are not influenza therapy. Prevention is annual vaccination matched to circulating strains, offered first to the risk groups and health workers, infection control and isolation in outbreaks, and antiviral prophylaxis in defined exposed high risk and outbreak settings under public health direction.

4.6 Helminthiasis

Helminthiasis is infection by parasitic worms, divided for therapy into the nematodes or roundworms, *Ascaris*, hookworms, *Enterobius*, *Strongyloides* and the filarial worms; the cestodes or tapeworms, *Taenia* species, the larval cyst diseases of hydatid and neurocysticercosis; and the trematodes or flukes, schistosomes and liver and lung flukes. Their burdens are chronic and nutritional as much as acute: intestinal worms competing for nutrition and, in hookworm, taking blood directly to produce iron deficiency anaemia of Part I, larval migration through lung and tissue, obstruction by heavy *Ascaris* loads, and the tissue diseases, lymphatic filariasis with elephantiasis, onchocerciasis with river blindness, schistosomiasis with bladder and liver fibrosis and bladder cancer risk bridging to Unit 5. Presentation follows the worm: perianal itch at night in enterobiasis, abdominal symptoms and passage of worms in ascariasis, ground itch and anaemia in hookworm, and often silence found on stool examination or eosinophilia on a blood count, eosinophilia being the laboratory signature of tissue helminths. Diagnosis is by stool microscopy for ova and parasites, concentration methods and repeat samples, serology and imaging for tissue disease, and species identification because therapy is species shaped.

Pharmacotherapy uses the anthelmintic classes by worm group. The benzimidazoles, albendazole and mebendazole, bind parasite tubulin and starve the worm of glucose uptake, and cover the common intestinal nematodes and, in prolonged specialist courses, the larval cyst diseases, where killing cysts in brain or eye is preceded by corticosteroid cover against the inflammatory response to dying parasites and by specialist assessment, a caution examinations prize. Praziquantel, increasing calcium permeability of the worm tegument, is the therapy of schistosomiasis and most fluke and tapeworm infections. Ivermectin, paralysing invertebrate muscle through its chloride channels, serves strongyloidiasis, onchocerciasis and filarial programmes, and diethylcarbamazine serves lymphatic filariasis where onchocerciasis coinfection

does not forbid it. Public health pharmacotherapy is half the topic: mass drug administration programmes delivering single encounter therapy to whole at risk communities for filariasis, schistosomiasis and soil transmitted helminths, with sanitation, footwear, safe water and meat inspection as the non drug prevention that ends reinfection.

4.7 Fungal infections

Fungal infections divide by depth: superficial infections of skin, hair, nails and mucosa; subcutaneous infections after traumatic implantation; and invasive systemic infections of the immune suppressed, which carry the mortality and the pharmacology of the topic. The superficial set repeats Part I skin teaching from the organism side: dermatophyte tinea of body, groin, feet and scalp, nail onychomycosis, and mucosal candidiasis, oral and vaginal, and pityriasis versicolor. The invasive set is a roll of the opportunist world shared with Section 4.2: invasive candidiasis and candidaemia in critical care and line infection, invasive aspergillosis of the neutropenic lung, mucormycosis invading sinuses and brain in diabetic and immune suppressed patients with surgical urgency, cryptococcal meningitis of Section 4.1, and the endemic mycoses of geography. Pathophysiology follows host defence: intact skin and flora exclude the superficial organisms to their niches, while neutropenia, corticosteroids, transplantation, HIV and broad antibacterial therapy open the body to moulds and yeasts that a competent immune system destroys unnoticed. Presentation of invasive disease is fever unresponsive to antibacterials in the at risk host, pulmonary infiltrates and sinus pain and eschar in mucormycosis demanding immediate action, and diagnosis is culture and microscopy from sterile sites, fungal biomarkers and antigen tests, imaging and tissue histology, pursued aggressively because therapy delayed is therapy denied in mould disease.

Pharmacotherapy is built from four systemic classes whose targets are the fungal cell structures human cells lack. The polyene amphotericin B binds ergosterol in the fungal membrane and remains the broadest fungicidal agent, in lipid formulations that soften its signature kidney and infusion toxicities of Section 2.9. The azoles inhibit ergosterol synthesis at its demethylase step and divide into the older agents, fluconazole serving candidiasis and cryptococcal maintenance with a clean interaction free reputation it does not deserve, since azoles inhibit human metabolic enzymes and interact widely, and itraconazole, voriconazole serving invasive aspergillosis as a standard first therapy, posaconazole and isavuconazole extending mould cover and prophylaxis. The echinocandins block glucan synthesis in the fungal wall and serve invasive candidiasis with excellent tolerability. Flucytosine serves cryptococcal induction in combination, never alone against resistance. Superficial disease is treated topically where it is limited, allylamines and azoles, and systemically for scalp, nail and widespread disease, terbinafine the standard for dermatophyte nails. Prophylaxis in defined transplant and haematology settings, resistance surveillance in *Candida* species, and therapeutic drug monitoring for the mould azoles complete the specialist picture.

4.8 Dysmenorrhea

Dysmenorrhea is painful menstruation, classified as primary, pain with normal pelvic anatomy beginning in adolescence after ovulatory cycles establish, and secondary, pain caused by pelvic pathology, endometriosis first, with adenomyosis, fibroids, pelvic inflammatory disease and intrauterine devices in the set. Primary dysmenorrhea has a clean prostaglandin pathophysiology that Part I prepared: in the late luteal phase and menstruation, endometrial prostaglandin production, driven by progesterone withdrawal, produces intense myometrial contraction, vasoconstriction and ischaemia of the uterus, with prostaglandin effects spilling systemically as nausea, diarrhoea, headache and faintness; pain is therefore worst as flow begins and eases as the endometrium sheds. Presentation of the primary form is cramping lower abdominal pain radiating to back and thighs with the systemic accompaniments, in a young patient with normal examination and a history from soon after menarche; secondary disease declares itself by later onset, progressive course, dyspareunia, infertility, abnormal bleeding or a pelvic mass, any of which

converts the consultation to investigation by examination, testing for infection and imaging, laparoscopy settling endometriosis. Diagnosis of the primary form is clinical after that red flag separation.

Pharmacotherapy of primary dysmenorrhea is a direct application of prostaglandin biology: non steroidal anti inflammatory drugs inhibiting prostaglandin synthesis are first line, started at the onset of pain or flow and taken regularly through the painful days rather than as single rescue doses, with the gastrointestinal and renal cautions of Part I and choice among the class by response and tolerability. Combined hormonal contraception suppressing ovulation and thinning the endometrium is the second pillar, serving patients who also need contraception or in whom non steroidal therapy fails, with progestogen only and intrauterine hormonal options in defined roles. Simple analgesia, heat, exercise and transcutaneous nerve stimulation support the drug core honestly as adjuncts. Secondary dysmenorrhea is treated by treating its cause: endometriosis by the hormonal suppression ladder of combined therapy, progestogens, gonadotropin releasing hormone analogues and antagonists with add back protection, and surgery, infection by its antibacterials, fibroids by their own pathway, with analgesia bridging and never substituting for the diagnosis.

4.9 Hormone replacement therapy

Hormone replacement therapy, taught in current usage also as menopausal hormone therapy, is the replacement of oestrogen, with progestogen protection where the uterus is present, for the symptoms and consequences of the menopause, the permanent cessation of ovarian function whose oestrogen withdrawal produces vasomotor symptoms, hot flushes and night sweats, genitourinary syndrome of dryness and urinary symptoms, sleep and mood disturbance, and accelerated bone loss toward the osteoporosis of Part I. Its pharmacotherapeutics is a balance taught honestly in both directions. Benefits are strongest for vasomotor symptom relief, where it is the most effective therapy, for genitourinary symptoms, where low dose local vaginal oestrogen serves with minimal systemic exposure, and for bone protection and fracture reduction while taken, with favourable timing evidence when started in younger postmenopausal patients close to the menopause. Risks are class and route shaped: combined therapy carries a small increase in breast cancer risk with duration in the trial and observational record, oral oestrogen increases venous thromboembolism and stroke risk through first pass hepatic effects on clotting, transdermal routes avoiding much of that thrombotic signal, and unopposed oestrogen in a woman with a uterus causes endometrial hyperplasia and cancer, which is the absolute rule that progestogen accompanies systemic oestrogen wherever a uterus remains.

Diagnosis and assessment precede therapy: confirm the menopausal pattern clinically in the usual age range, reserving hormone testing for early or atypical cases, exclude other causes of the symptoms, and assess breast, cardiovascular, thrombotic and endometrial risk and patient preference, since therapy is elective and preference sensitive. Pharmacotherapy is then individualised: systemic therapy for vasomotor symptoms at the lowest exposure controlling them, reviewed regularly and revisited as symptoms naturally remit; local therapy alone where symptoms are genitourinary only; progestogen by oral, transdermal combined or intrauterine routes; tibolone and tissue selective alternatives in defined niches; and non hormonal options, certain antidepressant class agents, gabapentinoids and the neurokinin antagonist class, for women in whom hormones are contraindicated or declined, after breast cancer among them, whose care is specialist shared. Osteoporosis prevention by hormone therapy is weighed against the dedicated bone classes of Part I and is not its usual modern indication where those serve better. Counselling states the balance numerically only from current guidance in the consultation itself; these notes teach the structure of that balance, which does not date.

*More free M.Pharm notes and unit wise PDFs at pharmacolearn.fyi
Free video lectures on the PharmacoLearn YouTube channel*