

### Unit 5: Oncology

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#### 5.1 General principles of cancer chemotherapy

Cancer is uncontrolled growth of abnormal cells that invade surrounding tissue and spread to distant sites, metastasis, arising from accumulated genetic damage to growth control: oncogenes activated, tumour suppressor genes lost, repair pathways failed, over years of carcinogen exposure, inherited susceptibility, infection and chance, with hallmarks of sustained proliferation, evasion of growth suppression and cell death, angiogenesis, immune evasion and metabolic reprogramming as its organising description.

Chemotherapy is drug treatment of cancer with cytotoxic and related agents, and its general principles govern every site specific section that follows. Cytotoxic agents injure dividing cells, by damaging DNA directly as the alkylating agents and platinum compounds do, by masquerading as building blocks and corrupting DNA and RNA synthesis as the antimetabolites do, by poisoning the mitotic spindle as the vinca alkaloids destabilising and the taxanes stabilising microtubules do, by trapping the topoisomerase enzymes that manage DNA topology as anthracyclines and their relatives do, anthracyclines adding free radical injury that explains their cardiac toxicity, and by hormonal manipulation, targeted small molecules and immunotherapy, which are chemotherapy in the wide teaching sense and dominate the site sections below. Because cytotoxic injury is proportional to division, therapy is given in cycles with recovery intervals for normal marrow and gut, in combinations of agents with different mechanisms and non overlapping toxicities that raise cure and suppress resistance exactly as in tuberculosis and HIV, and with intent declared before the first dose: curative, adjuvant after surgery to destroy micrometastases, neoadjuvant before surgery to shrink and assess, concurrent with radiotherapy for synergy, or palliative for symptom control and time, because intent decides how much toxicity is a fair price.

Cancer chemotherapy: from intent to cycle review

Diagnose and stage, declare the intent:

curative, adjuvant, neoadjuvant,  
concurrent or palliative

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v

Choose combination on mechanism, evidence  
and organ function, different toxicities  
paired, resistance suppressed

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v

Give in cycles, support through the  
expected marrow and gut injury,  
antiemetic cover planned in advance

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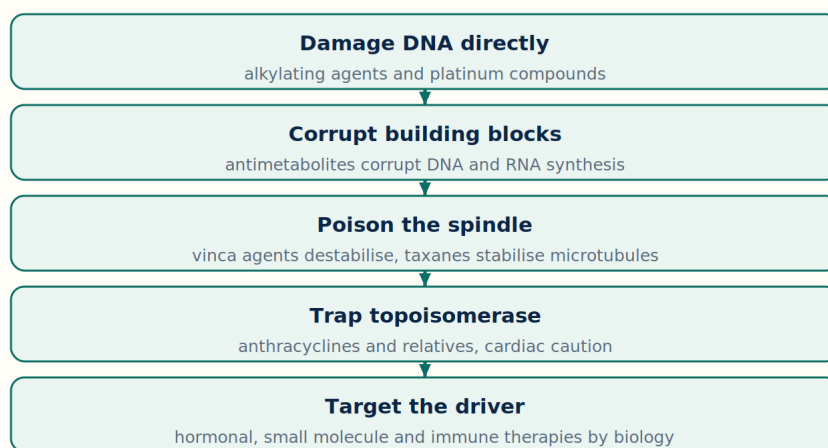
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Review response, toxicity and the  
patient's goals, continue, adjust  
or change intent honestly

Response and toxicity are the two ledgers of every cycle. Response is assessed clinically and by imaging and tumour markers where valid, complete and partial response, stable and progressive disease in the standard language, and resistance, primary or acquired by selection of resistant clones, efflux pumps, repair upregulation and target mutation, is planned against by combination and by switching class at progression. Toxicity divides into the shared and the signature: shared toxicities of marrow suppression with its infection, bleeding and anaemia nadirs, nausea and vomiting of Section 5.6, alopecia, mucositis,

diarrhoea and fatigue, infertility and second malignancy as late prices; and signature toxicities that monitoring is built around, anthracycline cardiomyopathy, cisplatin kidney, ear and nerve injury, vincristine neuropathy and constipation, bleomycin lung fibrosis of Part I, methotrexate liver, kidney and marrow injury rescued by folinic acid in its protocols, and the extravasation necrosis of vesicant agents as the administration emergency. Pharmacist practice in oncology is verification practice: protocol, body size and organ function checked, interactions hunted, antiemetic and growth factor support confirmed, and patient education on fever in the neutropenic window as an emergency instruction, not advice.

## Cancer chemotherapy: classes by mechanism



Combinations pair different mechanisms with non overlapping toxicities

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Figure: Classes by mechanism. Combinations pair different mechanisms with non overlapping toxicities.

## 5.2 Breast cancer

Breast cancer is the commonest cancer of women worldwide in the teaching record, arising chiefly in the ductal epithelium of the breast, and its pharmacotherapy is the model of biology directed treatment, because the tumour receptor pattern divides it into diseases treated differently. Risk factors are hormonal and reproductive, early menarche, late menopause, nulliparity and late first pregnancy, hormone replacement of Section 4.9 among them, family history and the inherited BRCA mutations, alcohol, obesity after the menopause and radiation exposure. Tumours classify by expression of oestrogen and progesterone receptors, by amplification of the HER2 growth factor receptor, and by proliferation rate: hormone receptor positive disease driven by oestrogen signalling, HER2 positive disease driven by that receptor and aggressive untreated, and triple negative disease with neither target and the fewest drug options. Presentation is a breast lump, skin or nipple change, nipple discharge or an axillary node, or a screening detected lesion; staging by imaging and sentinel node assessment, and metastatic survey where indicated, divides early from advanced disease and curative from palliative intent. Diagnosis is triple assessment: clinical, imaging and core biopsy, the biopsy carrying the receptor pattern on which all systemic therapy depends.

Pharmacotherapy follows the pattern and the stage together. Endocrine therapy is the backbone of hormone receptor positive disease: tamoxifen, a selective oestrogen receptor modulator blocking the receptor in breast while acting as agonist in uterus and bone, with venous thrombosis and endometrial cautions; aromatase inhibitors blocking peripheral oestrogen synthesis in postmenopausal patients, with bone loss and arthralgia as their price; ovarian suppression added in higher risk premenopausal disease; and the cyclin dependent kinase inhibitors combined with endocrine therapy in advanced and defined early disease, with fulvestrant degrading the receptor in later lines. HER2 positive disease is transformed by

HER2 directed therapy: trastuzumab and the monoclonal and conjugate and small molecule family that followed it, combined with chemotherapy in early disease and sequenced in advanced disease, with cardiac monitoring as its signature duty since HER2 blockade and anthracyclines injure the same heart. Chemotherapy serves triple negative disease, high risk and node positive disease and HER2 combinations: anthracycline and taxane containing regimens in the classical backbone, with immunotherapy added in defined triple negative settings and PARP inhibitors for the BRCA mutated. Surgery and radiotherapy are the local partners, systemic therapy given before or after them by stage, and bone protection and survivorship care, endocrine adherence over years being the adherence challenge of this section, complete the plan.

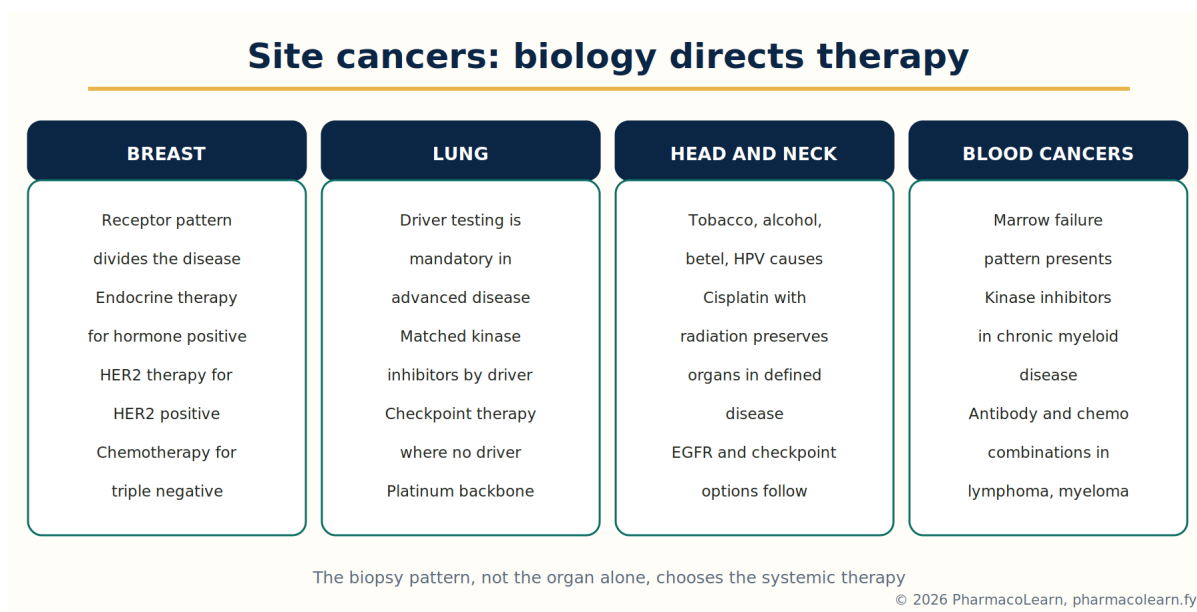


Figure: Biology directs therapy. The biopsy and molecular pattern, not the organ alone, chooses systemic treatment.

## 5.3 Lung cancer

Lung cancer is the leading cause of cancer death in the teaching record, caused overwhelmingly by tobacco smoking, with occupational exposures including asbestos, indoor and outdoor air pollution, and radon completing the causes, and it divides into two families with different biology and therapy. Non small cell lung cancer, the large majority, comprises adenocarcinoma, now commonest and the form of never smokers, squamous cell carcinoma tied closest to smoking, and large cell carcinoma. Small cell lung cancer is a neuroendocrine, rapidly doubling, early metastasising disease divided simply into limited and extensive stage and treated as systemic from diagnosis. Presentation is cough, haemoptysis, breathlessness, chest pain, recurrent infection behind an obstruction, weight loss and the paraneoplastic syndromes, syndrome of inappropriate antidiuretic hormone secretion and hypercalcaemia prominent, with metastases to brain, bone, liver and adrenal declaring advanced disease. Diagnosis is by imaging, biopsy or cytology with molecular profiling of non small cell tumours as a mandatory step in advanced disease, and staging that decides resectability, because surgery cures only the early minority and every other patient is a systemic therapy patient.

Pharmacotherapy of non small cell disease is the showcase of targeted therapy. Tumours are tested for driver alterations, and each found driver has its matched class: EGFR mutation with its tyrosine kinase inhibitors across generations matched to resistance mutations, ALK and ROS1 rearrangements with their inhibitors, and KRAS, BRAF, MET, RET and NTRK alterations with theirs in smaller groups; patients without a driver receive chemotherapy, platinum doublet combinations as the classical backbone, with immunotherapy, checkpoint inhibitors blocking the PD1 and PDL1 axis that releases T cell attack on the

tumour, alone or combined with chemotherapy by tumour PD L1 expression and stage, and antiangiogenic agents in defined combinations. Early disease adds adjuvant and neoadjuvant systemic therapy around surgery in modern practice. Small cell disease is treated with platinum and etoposide chemotherapy combined with immunotherapy in extensive stage and with concurrent radiotherapy in limited stage, responds dramatically and relapses almost universally, and is the starkest teaching example of chemosensitivity without cure in advanced disease. Palliative radiotherapy, pleural and airway symptom care and smoking cessation at every stage, cessation improving even advanced outcomes, complete the section.

## 5.4 Head and neck cancer

Head and neck cancers are the squamous cell cancers of the oral cavity, pharynx, larynx and related sites, caused classically by tobacco and alcohol acting synergistically, by betel quid and areca nut in the populations where they are chewed, and, for oropharyngeal cancer in rising proportion, by human papillomavirus infection, whose tumours carry a distinctly better prognosis and a younger patient. Presentation follows site: a non healing oral ulcer or patch, persistent hoarseness as the larynx warning that must always trigger referral, dysphagia and odynophagia, a neck mass as first sign in nodal spread, ear pain referred from pharynx and throat. Diagnosis is by examination including flexible endoscopy, biopsy, HPV testing of oropharyngeal tumours, and imaging for stage and planning, with dental, nutrition, speech and swallowing assessment before treatment because every modality injures those functions. Staging divides early disease, curable by a single local modality, from locally advanced disease needing combined treatment, and metastatic disease treated systemically.

Pharmacotherapy holds three defined places. Concurrent chemoradiation is the organ preserving and post operative standard in defined locally advanced disease: cisplatin given with radiotherapy as a radiosensitiser, its kidney, ear, nerve and emetic burdens of Section 5.1 accepted for a cure directed gain, with carboplatin based and cetuximab combinations, the EGFR antibody serving patients unfit for cisplatin, as alternatives. Induction chemotherapy before definitive local treatment serves defined presentations under specialist selection. Recurrent and metastatic disease is treated with platinum based combinations with fluorouracil in the classical regimen, cetuximab combinations, and checkpoint immunotherapy as first or later line by pattern, the modern sequence improving a historically bleak outlook. Supportive care is unusually heavy in this section and is pharmacotherapy too: feeding support through mucositis, pain of Section 1.7, antifungal vigilance of Section 4.7, thyroid replacement after neck irradiation, and alcohol and tobacco cessation without which second primaries harvest the survivors.

## 5.5 Hematological malignancies

The hematological malignancies are cancers of blood forming and lymphoid tissue: the leukaemias of marrow and blood, acute lymphoblastic and acute myeloid, chronic myeloid and chronic lymphocytic; the lymphomas, Hodgkin and non Hodgkin, of lymph nodes and lymphoid organs; multiple myeloma of plasma cells; and the myeloproliferative and myelodysplastic family around them. Their presentations rhyme because the marrow fails or overflows: anaemia, infection from absent or dysfunctional white cells, bleeding from thrombocytopenia, lymph node enlargement, splenomegaly, bone pain and lytic lesions in myeloma, and B symptoms of fever, drenching sweats and weight loss in lymphoma. Diagnosis is by blood count and film, marrow examination, flow cytometry immunophenotyping, cytogenetics and molecular testing that now classify and direct therapy together, imaging and staging for lymphoma, and protein studies for myeloma; these are the cancers in which the laboratory is the staging department.

Pharmacotherapy is the triumph and the textbook of modern targeted and combination therapy, and each disease carries one teaching exemplar. Chronic myeloid leukaemia, driven by the BCR ABL fusion kinase,

is controlled for decades in most patients by its tyrosine kinase inhibitors, imatinib and its successors matched to resistance and tolerance, the founding proof that a molecular target can convert a fatal leukaemia into a chronic condition. Acute leukaemias are treated with intensive multi agent chemotherapy in phases, induction, consolidation and, in lymphoblastic disease, prolonged maintenance on the tuberculosis pattern, with targeted additions by mutation, stem cell transplantation as consolidation for high risk disease, and differentiation therapy in acute promyelocytic leukaemia, where retinoic acid and arsenic force the malignant clone to mature, as the most elegant mechanism in oncology. Hodgkin lymphoma is cured in most patients by its classical combination chemotherapy with radiotherapy in early disease and antibody and checkpoint therapy in relapse; non Hodgkin lymphoma divides by aggressiveness, anti CD20 antibody rituximab combined with chemotherapy as the B cell backbone, cellular therapies including CAR T cells in defined relapses. Multiple myeloma, incurable but increasingly controlled, is built from proteasome inhibitors, immunomodulatory agents and anti CD38 antibodies in combinations sequenced across relapses, with transplantation in eligible patients and bone and infection protection throughout. Tumour lysis syndrome, the metabolic emergency of rapid malignant cell death releasing potassium, phosphate and uric acid into a failing kidney, prevented by hydration and urate lowering therapy and treated as Section 2.6 in the worst case, is the safety lesson of the section.

## 5.6 Management of nausea and vomiting

Nausea and vomiting in oncology divide into chemotherapy induced, radiotherapy induced, anticipatory, and the nausea of advanced disease itself, and the chemotherapy induced form is classified by timing: acute within the first day of treatment, delayed over the days that follow, breakthrough despite prophylaxis, refractory across cycles, and anticipatory, a conditioned response to the sights and smells of treatment in patients whose earlier cycles were badly controlled, prevented only by controlling those cycles well. The physiology is a set of emetic pathways that the drug classes map one to one: serotonin released from gut enterochromaffin cells by cytotoxic injury activating vagal and central serotonin receptors and driving acute emesis; substance P acting at neurokinin receptors centrally and driving delayed emesis; dopamine, histamine, acetylcholine and cannabinoid pathways serving vestibular, cortical and advanced disease nausea. Chemotherapy agents are graded by emetogenic potential, high, moderate, low and minimal, and prophylaxis is prescribed by that grade before the cycle, not after vomiting starts, which is the single rule of the section.

Pharmacotherapy combines classes by phase and grade. Serotonin receptor antagonists are the foundation for acute emesis; neurokinin receptor antagonists add the delayed phase cover that transformed highly emetogenic regimens; dexamethasone is combined across both phases for its central antiemetic contribution; and olanzapine in combination serves highly emetogenic and breakthrough settings in modern guidance, the four class combination being the full prophylaxis of the highest grade. Breakthrough treatment adds a class not already used, reviews hydration, obstruction, brain metastasis, opioids and other causes of Section 5.7, and refractory failure redesigns the next cycle rather than repeating a failed plan. Non chemotherapy nausea in advanced disease is treated by cause and mechanism: dopamine antagonists for chemical and drug induced nausea, antihistamine and anticholinergic agents for vestibular and obstruction patterns, prokinetics for gastric stasis, corticosteroids for raised pressure and liver stretch, and octreotide and decompression measures in malignant bowel obstruction under palliative direction. The pharmacist owns the pre cycle antiemetic check, because prophylaxis omitted cannot be recovered retrospectively.

## 5.7 Palliative care

Palliative care is the active total care of patients whose disease is not responsive to curative treatment, affirming life, regarding dying as a normal process, integrating physical, psychological, social and spiritual

care, and offered alongside disease directed therapy from the diagnosis of advanced illness in modern practice, not only in its last days; Unit 5 ends here because every oncology plan in this subject eventually asks what treatment is for. Its pharmacotherapy is symptom pharmacotherapy of the highest precision, and its core symptoms repeat the whole syllabus in miniature. Pain is treated on the ladder of Section 1.7 in its cancer form: oral morphine as the standard strong opioid, titrated against pain and function with laxatives always and antiemetics as needed, opioid rotation and alternative routes, subcutaneous infusion where swallowing fails, for intolerance or uncontrolled toxicity, adjuvants for neuropathic and bone pain including bisphosphonates of Part I, and radiotherapy as an analgesic for bone metastasis. Breathlessness is treated by its causes where reversible, by opioids at symptom directed exposure, oxygen only where hypoxaemia makes it a drug and not a comfort object, and positioning and airflow measures. Nausea follows Section 5.6, delirium is assessed for reversible drug and metabolic causes before any sedative, antipsychotics serving hyperactive delirium with risk, constipation is prevented rather than rescued, and secretions, agitation and anxiety in the last days are treated by the anticipatory medicines prescribed in advance in good services, the just in case medicines whose presence prevents crisis calls.

The prescribing philosophy is as examinable as any class list: regular review with deprescribing of every medicine whose purpose has expired, statins, preventive agents and tight control medicines relaxed as prognosis shortens, because pill burden is a symptom too; routes simplified; families taught what each medicine is for and what dying usually looks like; and goals of care discussed, documented and revisited, including resuscitation and place of care decisions, as clinical acts equal to any prescription. Palliative care in this syllabus is not confined to cancer in principle, its heart failure, lung and dementia applications echoing Part I and Unit 1, but its pharmacotherapy is taught here, at the end of the oncology unit, as the discipline that returns every drug decision to the patient it serves.

## Frequently Asked Questions

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**Q: What is the pharmacotherapeutics method this subject teaches for every disease?** Begin with etiopathogenesis, then presentation and diagnosis including severity, comorbidity, organ function, other medicines and adherence, then an individualised plan in steps: non drug measures and treatment of causes throughout, first drug classes matched to the mechanism, review against stated goals using clinical and laboratory indices, and adjustment by addition, substitution or escalation, with long term monitoring planned from the start. See Section 1.1.

**Q: How are antiseizure medicines chosen, and what counselling does epilepsy therapy require?** The medicine is chosen for the seizure type and syndrome, since some classes that control focal seizures aggravate absence and myoclonic seizures. Mechanisms include sodium channel blockade, enhancement of GABAergic inhibition, calcium channel blockade for absence seizures and mixed mechanisms, with broad spectrum agents where the syndrome is generalised or unsettled. Treatment starts as monotherapy, substitution precedes addition, and counselling covers adherence, teratogenicity and enzyme interaction planning, gradual supervised withdrawal only, and safety advice. See Section 1.2.

**Q: What are the drug strategies in Parkinson disease, and what limits levodopa over time?** Therapy is symptomatic: levodopa with a peripheral decarboxylase inhibitor is the most effective agent, dopamine agonists mimic dopamine, monoamine oxidase B and catechol O methyltransferase inhibitors prolong dopamine or levodopa action, amantadine serves dyskinesia, and anticholinergics hold a narrow tremor role in younger patients. Long term levodopa use is limited by wearing off, on and off fluctuations and dyskinesias, which advanced infusion therapies and deep brain stimulation address in selected patients. See Section 1.3.

**Q: Why does stroke pharmacotherapy divide so sharply, and what is built on the division?** Ischaemic stroke follows arterial occlusion and haemorrhagic stroke follows vessel rupture, and their acute treatments are opposite in aim: reperfusion, antiplatelet therapy and mechanism specific prevention after ischaemia, against bleeding control, anticoagulant reversal and pressure management after haemorrhage, which is why urgent imaging precedes treatment. Secondary prevention is built from the mechanism: antiplatelet therapy for non cardioembolic disease, anticoagulation for atrial fibrillation, statin therapy, and blood pressure treatment as the most important long term measure. See Section 1.4.

**Q: How do the antidepressant and antipsychotic classes trade their harms?** Antidepressants share similar average efficacy and are chosen on harm profile and patient factors: selective serotonin reuptake inhibitors first line for tolerability and safety in overdose, serotonin and noradrenaline agents adding pain and blood pressure considerations, tricyclics limited by anticholinergic and cardiac effects and overdose lethality. Antipsychotics divide the same way: first generation agents carry extrapyramidal and prolactin harms, second generation agents carry metabolic harms, and clozapine alone serves resistant schizophrenia under mandatory blood monitoring. See Sections 2.1 and 2.2.

**Q: What are the principles of managing acute and chronic renal failure with drugs?** Acute injury is managed supportively: treat the cause, restore perfusion where depletion is the mechanism, relieve obstruction, stop or adjust nephrotoxic and renally cleared medicines, and treat hyperkalaemia, overload and acidosis in order of danger, with dialysis for refractory complications. Chronic disease is managed to slow progression with renin angiotensin blockade where proteinuria is present, glycaemic and pressure control and SGLT2 inhibitor therapy, and to treat anaemia, mineral and bone disorder and acidosis, with every dose reviewed against filtration. See Sections 2.6 and 2.7.

**Q: What are the rules of rational antibiotic use, and how does surgical prophylaxis express them?** Confirm bacterial infection, find the source, take cultures first where possible, choose the narrowest spectrum covering likely organisms from local guidance, review against cultures for narrowing, oral switch and stopping, and document indication, review date and duration. Surgical prophylaxis is the same discipline compressed: cover only where infection is likely or grave, against expected site organisms, timed for incision, and stopped at closure rather than continued as a course. See Section 3.1.

**Q: Why is tuberculosis treated with combinations for months, and what does each first line agent demand in monitoring?** Combination therapy kills multiplying bacilli rapidly, sterilises dormant persisters and prevents resistance, in an intensive phase followed by a continuation phase. Isoniazid demands liver and neuropathy vigilance with pyridoxine in at risk patients, rifampicin demands liver vigilance and interaction review for its enzyme induction, ethambutol demands visual monitoring, and pyrazinamide demands liver vigilance. Adherence support and public health supervision protect both the patient and the regimen. See Section 3.5.

**Q: How is HIV treated in principle, and how are its opportunistic infections handled?** Antiretroviral therapy combines classes attacking successive life cycle steps, nucleoside backbone with an integrase inhibitor as the usual modern anchor, suppressing viral load, restoring immunity and preventing transmission when suppressed; it is lifelong and adherence is its effectiveness. Opportunistic infections are prevented by prophylaxis at defined immune depths and treated specifically when established, with interactions and immune reconstitution planned for. See Section 4.2.

**Q: What are the general principles of cancer chemotherapy, and how is its nausea prevented?** Cytotoxic classes injure dividing cells by distinct mechanisms and are combined, in cycles with recovery intervals, with intent declared in advance, curative, adjuvant, neoadjuvant, concurrent or palliative, because intent decides acceptable toxicity; response and signature toxicities are reviewed every cycle. Chemotherapy induced nausea is prevented by grade before the cycle: serotonin antagonists for acute

emesis, neurokinin antagonists for the delayed phase, dexamethasone across phases and olanzapine in the fullest combinations, never by waiting for vomiting to start. See Sections 5.1 and 5.6.

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