

Unit 5: Bone and Joint Disorders, Dermatological Diseases and Ophthalmology

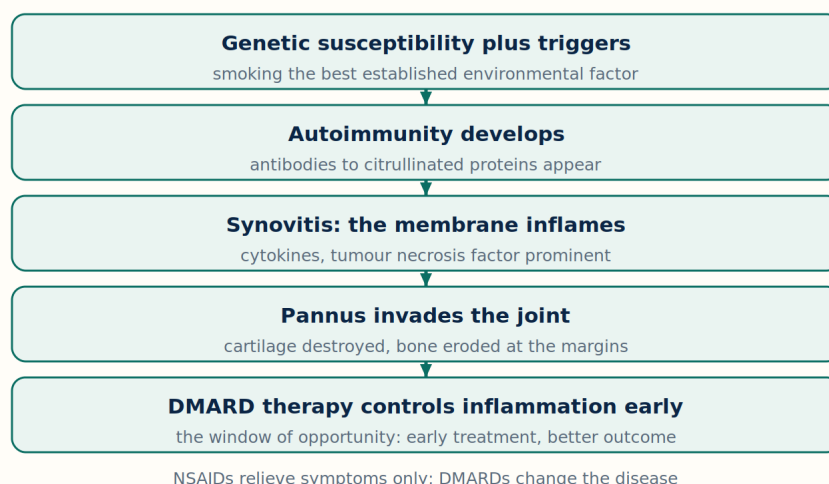
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5.1 Rheumatoid arthritis

Rheumatoid arthritis is a chronic systemic autoimmune disease whose central lesion is inflammatory synovitis: the synovial membrane thickens into an invasive tissue called pannus and destroys cartilage and erodes bone at the joint margins, producing deformity and loss of function if inflammation is not controlled. Its aetiology combines genetic susceptibility, notably shared epitope variants of the HLA system, with environmental triggers, smoking the best established, on a background in which autoimmunity to citrullinated proteins develops years before symptoms; rheumatoid factor and anti cyclic citrullinated peptide antibodies are its serological markers, the second the more specific. Synovial inflammation is driven by a cytokine network, tumour necrosis factor and interleukins prominent, recruiting inflammatory cells, activating fibroblasts and osteoclasts, and generating systemic features through circulating inflammation: fatigue, anaemia of chronic disease and accelerated cardiovascular disease, its major cause of premature death. Presentation is classically a symmetric polyarthritis of the small joints of the hands and feet with pain, swelling and prolonged morning stiffness evolving over weeks; over time come the characteristic deformities of ulnar deviation and swan neck and boutonniere fingers. Diagnosis is clinical, supported by inflammatory markers, autoantibodies and imaging showing erosions, and treatment is not delayed awaiting every test result, because early treatment changes the outcome: the window of opportunity principle.

Pharmacotherapy is early, disease modifying and target driven. Disease modifying antirheumatic drugs divide into conventional synthetic, targeted synthetic and biologic groups. Methotrexate is the anchor conventional agent, given weekly with folic acid supplementation, monitored by blood counts and liver tests, avoided in pregnancy, and combined or sequenced with sulfasalazine, leflunomide and hydroxychloroquine according to response and tolerance. Treatment escalates against a target of remission or low disease activity at regular reviews: where conventional therapy fails, biologic agents against tumour necrosis factor, interleukin 6 signalling, B cells or T cell costimulation, or targeted Janus kinase inhibitors, are added under specialist care after screening for latent infection, tuberculosis above all, and with vaccination reviewed. Glucocorticoids are short bridging therapy while DMARDs take effect and for flares, never the long term strategy where avoidance is possible. Non steroidal anti inflammatory drugs relieve symptoms and modify nothing; physiotherapy, occupational therapy and surgery for destroyed joints complete the multidisciplinary answer. Monitoring is the pharmacist visible part: the shared blood monitoring schedule of the DMARDs, infection vigilance under immunosuppression, and counselling on weekly methotrexate, whose accidental daily use is a recognised fatal error this subject expects students to name.

Rheumatoid arthritis: from autoimmunity to erosion



NSAIDs relieve symptoms only; DMARDs change the disease

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Figure: From autoimmunity to erosion. DMARD therapy works because it interrupts this cascade before the joint is destroyed.

5.2 Osteoarthritis

Osteoarthritis is the commonest joint disease, a disorder of the whole joint in which articular cartilage progressively fails, with changes in underlying bone, synovium, ligaments and muscle. It differs in kind from rheumatoid arthritis: its primary process is cartilage breakdown and failed repair, not autoimmune synovitis. Risk factors are age, obesity, previous joint injury, malalignment, occupational overload and genetic predisposition. Cartilage loses proteoglycan and its collagen network, chondrocyte repair cannot keep pace, the joint space narrows, subchondral bone thickens and forms osteophytes, and pain arises from bone, synovium, ligament and muscle rather than from the aneural cartilage. Presentation is pain on use relieved by rest, brief inactivity stiffness, reduced movement, crepitus and bony enlargement, in the hand the Heberden and Bouchard nodes at the finger joints; inflammatory markers are normal and imaging shows joint space narrowing, osteophytes and subchondral sclerosis. Diagnosis is clinical in the typical older patient, with inflammatory arthritis, infection and malignancy excluded by pattern.

Pharmacotherapy is symptomatic only, no agent modifies structure, and the non drug core takes first place: education, exercise and physiotherapy to strengthen the muscles protecting the joint, weight reduction where overweight, footwear, walking aids and occupational adaptations. Paracetamol has a modest, limited effect. Topical non steroidal anti inflammatory preparations are effective for knee and hand joints with little systemic exposure and are an early choice in older patients. Oral non steroidal anti inflammatory drugs are the most effective symptomatic class, used at the lowest effective exposure for the shortest time, with proton pump inhibitor gastroprotection where gastrointestinal risk requires it, and cardiovascular, renal and gastrointestinal risk assessed in every patient; cyclo oxygenase 2 selective agents trade some gastrointestinal advantage against cardiovascular caution, as in Section 3.1. Intra articular corticosteroid injection gives short term relief of an inflamed flare. Opioids have little place in chronic disease. Joint replacement for end stage knee and hip disease is the definitive treatment, and recognising when conservative therapy has reached its limit is part of pharmacotherapeutics.

5.3 Gout

Gout is arthritis caused by deposition of monosodium urate crystals in and around joints, the clinical expression of chronic hyperuricaemia. Uric acid is the end product of purine metabolism in humans, who lack uricase, and its level is set by production against excretion: most hyperuricaemia is renal under

excretion, worsened by kidney disease, diuretics, especially thiazides, low dose aspirin, alcohol, obesity and purine and fructose rich diets; overproduction dominates in rapid cell turnover states and rare enzyme defects. When urate exceeds its solubility, crystals form in cooler peripheral joints and trigger an intense innate immune response through inflammasome activation and interleukin 1 release, producing the acute attack. The acute attack is dramatic: abrupt, exquisitely painful, hot, swollen arthritis, classically of the first metatarsophalangeal joint, podagra, often at night and after a dietary, alcohol, illness or drug provocation, settling over days to weeks even untreated; between attacks the patient is well until the pattern accelerates. Diagnosis is by crystal identification in aspirated joint fluid, the gold standard that also excludes septic arthritis, the dangerous mimic that must always be considered, supported by the clinical pattern and urate measured away from the acute attack, since it may be normal during one.

Pharmacotherapy separates into two questions that must never be merged: treating the acute attack, and lowering urate to prevent attacks and dissolve deposits. The acute attack is treated promptly with anti inflammatory therapy: non steroidal anti inflammatory drugs, colchicine, which disrupts microtubule dependent inflammatory activation and works best early, limited by gastrointestinal intolerance and interactions, or corticosteroids where the first two are unsuitable, with interleukin 1 blockade reserved for refractory cases under specialist care. Urate lowering therapy has its own indications: recurrent attacks, tophi, joint damage, stones and very high urate. Allopurinol, a xanthine oxidase inhibitor blocking uric acid production, is first line, started low and titrated against urate with renal function and its hypersensitivity syndrome in mind; febuxostat is the alternative xanthine oxidase inhibitor, with a cardiovascular caution in its history; uricosuric agents increase renal excretion where under excretion dominates and kidney function and stone risk allow; uricase therapy converting urate to soluble allantoin is a specialist last line for refractory tophaceous disease. Established urate lowering therapy is continued through acute attacks in current teaching, and prophylactic anti inflammatory cover accompanies its initiation, when urate shifts provoke flares.

5.4 Osteoporosis

Osteoporosis is reduced bone mass and microarchitectural deterioration leading to fragility, defined operationally by low bone mineral density on dual energy X ray absorptiometry and clinically by its fractures: vertebral, hip and wrist fractures from falls from standing height or less that healthy bone would withstand. Bone is remodelled continuously, osteoclasts resorbing and osteoblasts forming, coupled under hormonal and cytokine control, the RANKL pathway being the key osteoclast driver that one drug class targets. Peak mass is reached in young adult life; thereafter resorption gradually exceeds formation, and loss of oestrogen at the menopause sharply accelerates resorption, postmenopausal osteoporosis being the commonest form. Other causes must be sought: glucocorticoid therapy, the commonest secondary cause and a classic drug induced disease, hypogonadism, hyperthyroidism, hyperparathyroidism, alcohol, smoking, immobility, low body weight, vitamin D deficiency and malabsorption. The disease is silent until fracture: vertebral crush fractures present with back pain, height loss and kyphosis, and hip fracture dominates its mortality in the elderly. Diagnosis is by bone density measurement, by fracture risk assessment combining density with clinical risk factors, and by blood tests excluding secondary causes; a fragility fracture in an older patient establishes the diagnosis clinically and treatment follows without waiting for density confirmation.

Pharmacotherapy aims to reduce fractures, and its classes act on the remodelling balance from both sides. Foundation measures are adequate calcium and vitamin D, corrected where deficient, weight bearing and muscle strengthening exercise, fall prevention, smoking cessation and alcohol moderation.

Bisphosphonates are the first line antiresorptive class: they bind bone mineral, are taken up by resorbing osteoclasts and disable them, reducing spine and hip fractures. The oral agents carry the famous

administration discipline, taken fasting with a full glass of water, remaining upright, nothing else by mouth for a period after, because of oesophageal injury risk and near zero absorption with food, a counselling set examinations follow; intravenous preparations serve patients who cannot follow or tolerate the oral route. Denosumab, a monoclonal antibody against RANKL, is a potent antiresorptive given by injection at long intervals, with the warning that stopping it without follow on antiresorptive therapy produces rebound bone loss and vertebral fractures. Anabolic agents that build bone, the parathyroid hormone analogues and the sclerostin inhibitor, serve severe and high risk disease under specialist care, followed by an antiresorptive to hold the gain.

5.5 Psoriasis

Psoriasis is a chronic immune mediated skin disease in which keratinocyte proliferation is greatly accelerated and disordered, producing well demarcated, red, scaly plaques. Genetic susceptibility and immune dysregulation are its basis, T cell and cytokine pathways, the interleukin 17 and interleukin 23 axis centrally, driving epidermal hyperplasia so that cells reach the surface in days rather than weeks and pile as silvery scale. Triggers include streptococcal throat infection, classically before guttate psoriasis, skin trauma localising lesions to injury (the Koebner phenomenon), stress, alcohol, smoking and medicines, lithium, beta blockers and antimalarials among the recognised provocations. Patterns include chronic plaque psoriasis of elbows, knees, scalp and lower back, guttate, flexural, pustular and erythrodermic forms, nail disease and associated psoriatic arthritis. Psoriasis is also a systemic inflammatory state with increased cardiovascular and metabolic risk and a heavy psychological burden. Diagnosis is usually clinical, severity graded by extent and by impact on quality of life, which together decide the treatment ladder.

Pharmacotherapy is stepped by severity and site. Emollients are universal foundation therapy. Topical corticosteroids and vitamin D analogues, which normalise keratinocyte proliferation, are the mainstay for limited plaque disease, combined in single preparations for adherence; coal tar and dithranol are older effective agents limited by mess and irritation, and calcineurin inhibitors serve face and flexures where steroids thin skin. Phototherapy treats widespread disease failing topicals. Systemic therapy for moderate to severe disease uses methotrexate, ciclosporin, rapid but limited by kidney and blood pressure effects, and acitretin, a retinoid for pustular forms, teratogenic with a prolonged contraception requirement. Biologic therapies against tumour necrosis factor, interleukin 17 and interleukin 23 pathways, and the oral phosphodiesterase 4 inhibitor for moderate disease, have transformed severe psoriasis under specialist supervision with infection screening. Counselling covers a long term, relapsing course: control, not cure.

5.6 Eczema and scabies

Eczema, or dermatitis, is a pattern of skin inflammation whose commonest important form is atopic eczema, a chronic relapsing itchy dermatosis of childhood onset associated with the atopic family, asthma of Section 2.1 among them. Its pathophysiology combines a defective skin barrier, filaggrin gene defects prominent among its causes, which lets water out and irritants and allergens in, with Th2 type immune inflammation; other eczemas are irritant and allergic contact dermatitis, where patch testing identifies allergic culprits, seborrhoeic, discoid and varicose patterns. Management is barrier repair plus anti inflammatory control: liberal emollients as continuing maintenance and soap substitutes, avoidance of identified triggers, topical corticosteroids stepped by potency to site and severity in defined short courses with fingertip unit guidance, topical calcineurin inhibitors for sensitive sites and steroid sparing maintenance, antibacterials where infection supervenes, and for severe disease phototherapy, systemic immunosuppressants and the biologic and targeted therapies against interleukin 4 and 13 signalling and Janus kinase pathways.

Scabies shares the printed syllabus head with eczema but is a different kind of disease: infestation by the mite *Sarcoptes scabiei*, transmitted by prolonged skin contact, the female mite burrowing into the stratum

corneum and laying eggs, the itching being a hypersensitivity response to the mite and its products. Diagnosis is clinical, supported by mite or egg demonstration on scraping or dermoscopy. Treatment is scabicide and social at once: permethrin cream, the standard topical agent, applied to the whole body surface as directed and repeated per its schedule, or oral ivermectin in crusted disease, outbreaks and where topical application is impractical, and, decisively, simultaneous treatment of all close contacts with hot washing of bedding and clothing, because untreated contacts reinfest the treated patient. Itch persists for weeks after successful treatment as hypersensitivity settles, must be explained in advance so it is not read as failure, and is treated symptomatically.

5.7 Impetigo

Impetigo is a superficial, highly contagious bacterial skin infection, commonest in children, caused by *Staphylococcus aureus*, *Streptococcus pyogenes*, or both. Non bullous impetigo is the classic form, honey coloured crusted erosions around the nose, mouth and limbs arising where minor trauma, insect bites or underlying eczema break the skin; in bullous impetigo, staphylococcal exfoliative toxins split the superficial epidermis into flaccid blisters. Diagnosis is usually clinical, swabs settling doubtful or recurrent cases. Its importance exceeds its depth: it spreads through families, schools and teams, and streptococcal impetigo is associated with post streptococcal glomerulonephritis, a reason to treat rather than merely reassure.

Pharmacotherapy follows extent. Limited lesions in a well patient are treated topically with an antiseptic or a topical antibacterial such as fusidic acid or mupirocin, with crust removal and hygiene. Widespread, bullous, recurrent or outbreak disease, and disease in the immunocompromised, is treated with oral antibacterials active against both organisms per local guidance. Recurrence prompts review for nasal staphylococcal carriage and untreated contacts.

5.8 Drug induced skin disorders

The skin is the most visible target of adverse drug reactions, and drug induced skin disorders range from the common and benign to the rare and life threatening, a range the examination answer must span in order. Exanthematous or maculopapular eruptions are the commonest pattern, a symmetric red rash appearing days into a new drug, most often an antibacterial, and settling on withdrawal, its main task being not to miss the early signs of a severe reaction within it. Urticaria and angioedema follow histamine release by immune and non immune mechanisms, penicillins and NSAIDs prominent, angioedema with airway involvement being an emergency, and ACE inhibitor angioedema, bradykinin mediated and antihistamine resistant, its own named entity. Fixed drug eruption recurs at exactly the same site on each exposure, leaving pigmented patches, a history pattern that makes the diagnosis. Photosensitivity reactions divide into phototoxic, dose related exaggerated sunburn, and photoallergic patterns, with tetracyclines, fluoroquinolones, thiazides and amiodarone among the culprits, managed by withdrawal and photoprotection. The severe cutaneous adverse reactions stand at the head of the topic and must be recognised on sight: Stevens Johnson syndrome and toxic epidermal necrolysis, a spectrum of epidermal necrosis with mucosal involvement and skin detachment, caused above all by allopurinol, sulfonamides, aromatic anticonvulsants and lamotrigine and certain NSAIDs, with high mortality, treated by immediate withdrawal of every suspect drug and specialist burn level supportive care; DRESS, drug reaction with eosinophilia and systemic symptoms, a delayed eruption with fever, facial swelling, eosinophilia and hepatitis and other organ injury, classically from anticonvulsants, allopurinol and sulfonamides, treated by withdrawal and corticosteroids; and acute generalised exanthematous pustulosis, a rapid sterile pustular eruption, usually antibacterial driven, settling on withdrawal. Pharmacogenetic screening prevents some, HLA testing before carbamazepine and abacavir and in defined allopurinol risk groups being the standard examples. The pharmacist discipline across all patterns is the timeline, every new eruption matched against every drug started in the preceding weeks, immediate withdrawal of suspects in severe patterns,

documentation and reporting, allergy records that distinguish rash histories precisely, and counselling that a patient who has survived a severe reaction must never receive the culprit again.

5.9 Conjunctivitis

Conjunctivitis is inflammation of the conjunctiva, presenting as a red eye with discharge, irritation and itching, usually with normal vision and little pain, the features that separate it from the dangerous causes of red eye. Infective conjunctivitis is viral, most often adenoviral, watery and highly contagious, or bacterial, with purulent discharge and lids stuck on waking; chlamydial and gonococcal forms demand specific systemic therapy and referral. Allergic conjunctivitis is itchy above all, bilateral, seasonal or perennial with rhinitis. Irritant forms follow chemicals, smoke and medicines themselves, including preservative reactions. Diagnosis is clinical from discharge character, itch, laterality and associated features, with pain, photophobia, reduced vision, corneal involvement, contact lens wear and failure to settle as referral triggers, since keratitis, uveitis and acute glaucoma share the red eye headline and none of them is conjunctivitis.

Pharmacotherapy is cause matched and mostly restrained. Viral conjunctivitis is self limiting, treated supportively with lubricants and hygiene; antibacterials have no action on it. Bacterial conjunctivitis often settles without treatment, topical antibacterial drops or ointment, chloramphenicol and fusidic acid preparations being standard examples, shortening the course where discharge is heavy or return to school or work requires it. Allergic conjunctivitis is treated by allergen avoidance, topical antihistamine and mast cell stabilising drops, often combined in dual acting agents, and oral antihistamines where rhinitis accompanies; topical steroids are specialist directed only, because unsupervised ocular steroids aggravate herpes simplex keratitis and raise intraocular pressure, the caution that links this section to the next. Hygiene counselling, hand washing, no shared towels and no lens wear until settled, completes most episodes.

5.10 Glaucoma

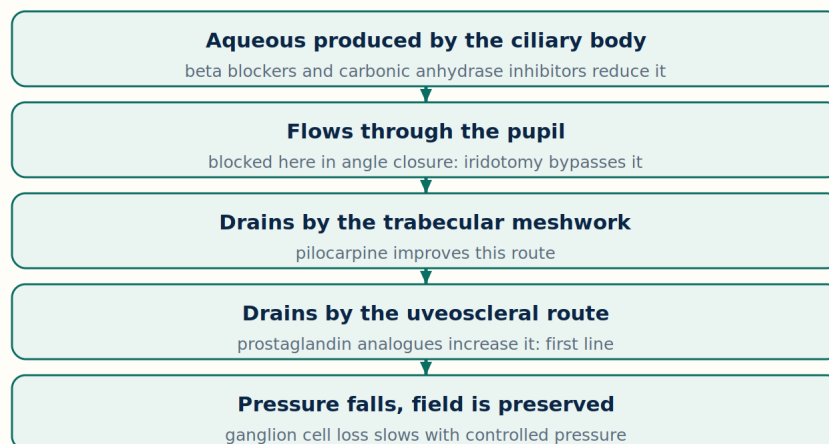
Glaucoma is a group of optic neuropathies in which retinal ganglion cells and their axons are progressively lost, producing characteristic optic disc excavation and visual field loss beginning in the periphery, with raised intraocular pressure the major modifiable risk factor, though damage also occurs at statistically normal pressures. Aqueous humour physiology is the pharmacology of the whole topic: aqueous is produced by the ciliary body, flows through the pupil into the anterior chamber, and drains chiefly through the trabecular meshwork into the canal of Schlemm, with a secondary uveoscleral route; pressure is the balance of production against outflow, and every drug class either reduces production or increases outflow. In primary open angle glaucoma, the commonest form, the angle is open but trabecular resistance rises, pressure climbs slowly and painlessly, and the patient notices nothing until field loss is advanced, so detection is by opportunistic examination of pressure, disc and fields in at risk patients: older age, family history, African ancestry, diabetes and myopia among them. In primary angle closure glaucoma, a shallow anterior chamber lets the iris block the meshwork when the pupil dilates, precipitating an acute attack of severe eye pain, blurred vision with haloes, a red eye, a mid dilated unreactive pupil and nausea, an ophthalmic emergency in which pressure must be lowered at once or sight is lost within days. Diagnosis is by tonometry, gonioscopy, disc assessment and formal visual field testing in combination.

Pharmacotherapy of open angle glaucoma lowers pressure with topical agents in a stepped sequence, laser trabeculoplasty and surgery standing beside drugs in the modern pathway. Prostaglandin analogues are the usual first line class: they increase uveoscleral outflow, are used once daily in the evening, and have the distinctive local effects of iris darkening, lash growth and periorbital fat loss that patients must be warned of so they do not stop a working drug. Beta blocker drops reduce aqueous production and carry

their class cautions in miniature, bronchospasm in asthma and bradycardia, since drops drain through the nasolacrimal duct into the systemic circulation, which punctal occlusion after instillation reduces, a practical counselling technique. Alpha agonists reduce production and increase outflow, allergic conjunctivitis limiting them. Topical carbonic anhydrase inhibitors reduce production and are common additions, the oral form reserved for short term and emergency use. Pilocarpine increases trabecular outflow and survives mainly in angle closure management. The acute angle closure attack is an emergency sequence: rapid pressure lowering by combined topical agents and systemic carbonic anhydrase inhibition, analgesia and antiemetics, then the definitive laser peripheral iridotomy that bypasses the blocked pupil pathway, also performed in the fellow eye. Adherence in a symptomless, lifelong, sight preserving therapy is the closing counselling theme of the unit: patients stop the drops that sting, and keep their sight only if the bargain is explained plainly.

Glaucoma: the aqueous balance and where drugs act
 Ciliary body produces aqueous humour
 (beta blockers, alpha agonists and carbonic anhydrase inhibitors reduce production here)
 ↓
 ↓
 Posterior chamber, through the pupil
 (blocked here in angle closure: iridotomy bypasses it)
 ↓
 ↓
 Anterior chamber drains two ways:
 trabecular meshwork (pilocarpine improves this route)
 uveoscleral outflow (prostaglandin analogues increase this route, first line therapy)
 ↓
 ↓
 Pressure falls, ganglion cell loss slows, visual field is preserved

Glaucoma: aqueous flow and where drugs act



A symptomless disease: adherence keeps the sight the drops protect

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Figure: Aqueous flow and drug action. Every glaucoma class either reduces production or increases one of the two drainage routes.

Frequently Asked Questions

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 is hypertension treated in principle, and which drug classes are first line? Lifestyle measures apply to every patient: weight reduction, salt restriction, a healthy diet, regular exercise, alcohol moderation and stopping smoking. The first line classes are thiazide and thiazide like diuretics, calcium channel blockers, ACE inhibitors and angiotensin receptor blockers, with beta blockers first line chiefly where a compelling cardiac indication coexists. Most patients need complementary classes in combination, and resistant hypertension adds agents such as aldosterone antagonists under specialist care. See Section 1.2.

Q: Which drug classes improve survival in heart failure with reduced ejection fraction, and which only relieve symptoms? Renin angiotensin inhibition, including the angiotensin receptor and neprilysin inhibitor combination, the beta blockers proven in heart failure, aldosterone antagonists and SGLT2 inhibitors improve outcomes, because they block or counter the maladaptive neurohormonal responses. Loop diuretics relieve congestion by removing fluid but are symptom therapy, adjusted to fluid state. See Section 1.3.

Q: What are the two drug aims in acute coronary syndrome, and which classes serve each? To limit necrosis by restoring flow, by reperfusion with intervention or fibrinolysis supported by anticoagulation, and to prevent thrombus extension and recurrence, by dual antiplatelet therapy with aspirin plus a P2Y₁₂ blocker in the acute phase and long term secondary prevention with continued antiplatelet therapy and a statin. Nitrates relieve pain, and beta blockers reduce oxygen demand where not contraindicated. See Section 1.4.

Q: How do asthma and COPD differ in their drug therapy foundations? In asthma the pathology is airway inflammation with hyperresponsiveness, so inhaled corticosteroids are the foundation at every step and long acting bronchodilators are used only in combination with them. In COPD the pathology is persistent airflow limitation from airway and parenchymal damage, so long acting bronchodilators are the foundation, inhaled corticosteroids are added only for selected patients, and smoking cessation is the one intervention that changes the course. See Sections 2.1 and 2.2.

Q: How are the diabetes drug classes matched to the physiology of type 2 diabetes? Metformin reduces hepatic glucose production. Sulfonylureas stimulate insulin secretion and risk hypoglycaemia and weight gain. DPP 4 inhibitors prolong incretin action gently, GLP 1 receptor agonists enhance glucose dependent insulin secretion, reduce appetite and weight and include agents with cardiovascular benefit, and SGLT2 inhibitors lower glucose by renal glucose excretion and protect heart and kidneys. Insulin replaces the hormone and is obligatory in type 1 diabetes. See Section 2.4.

Q: What decides drug therapy in peptic ulcer disease? The balance between aggressive factors, acid, pepsin, *Helicobacter pylori* and NSAID injury, and defensive factors, the mucus bicarbonate barrier, blood flow and prostaglandins. Proton pump inhibitors suppress acid at the final common step for healing, eradication therapy removes *H. pylori* so ulcers do not recur, and NSAID ulcers require stopping or reducing the NSAID where possible with proton pump inhibitor healing and protection. See Section 3.1.

Q: How do ulcerative colitis and Crohn disease differ, and what follows for therapy? Ulcerative colitis is continuous mucosal inflammation of the colon only, presenting chiefly with bloody diarrhoea, and colectomy removes the colonic disease. Crohn disease is transmural, discontinuous and may affect any part of the gut, producing strictures and fistulae, and surgery is bowel preserving because recurrence is common. Both are treated by inducing remission, corticosteroids having that role but never the maintenance role, and maintaining remission with aminosalicylates in ulcerative colitis, immunomodulators, and biologic or targeted therapies in moderate to severe disease. See Section 3.3.

Q: What are the principles of anticoagulation in deep vein thrombosis? Anticoagulants prevent extension, embolisation and recurrence while the body removes the clot; they do not dissolve it directly, thrombolysis being reserved for massive pulmonary embolism and a few limb threatening cases. Options are heparins, warfarin with INR monitoring and a parenteral overlap at initiation, and the direct oral anticoagulants, now usual for most patients. Duration is individualised by provoking factors and bleeding risk, and thromboprophylaxis of at risk inpatients is the preventive half of the subject. See Section 4.5.

Q: How do drugs treat glaucoma, in terms of aqueous humour? Aqueous is produced by the ciliary body and drains through the trabecular meshwork and the uveoscleral route, and every class acts on that balance. Prostaglandin analogues increase uveoscleral outflow and are first line. Beta blockers, alpha agonists and carbonic anhydrase inhibitors reduce production, alpha agonists also increase outflow, and pilocarpine increases trabecular outflow. The acute angle closure attack is an emergency treated by rapid pressure lowering followed by laser iridotomy. See Section 5.10.

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