

2.1 Asthma

Asthma is a chronic inflammatory disease of the airways characterised by variable airflow obstruction and bronchial hyperresponsiveness, in which the airways narrow in response to triggers that leave normal airways unaffected, and narrow reversibly, spontaneously or with treatment. The pathophysiology is inflammation first and bronchospasm second, the order examinations test. In the allergic pattern, inhaled allergen cross links IgE on mast cells, releasing histamine, prostaglandins and leukotrienes that produce the early bronchoconstriction, oedema and mucus; hours later a late response recruits eosinophils and Th2 lymphocytes and their cytokines, sustaining inflammation. Presentation is episodic wheeze, breathlessness, chest tightness and cough, worse at night and early morning and varying with triggers, with a normal examination between attacks in mild disease; severe attacks bring inability to complete sentences, exhaustion and, ominously, a silent chest.

Pharmacotherapy has two arms that must never be confused: reliever therapy that reverses bronchoconstriction, and controller therapy that treats inflammation and prevents attacks. Inhaled corticosteroids are the foundation controller class, suppressing inflammation, reducing hyperresponsiveness and preventing exacerbations, and modern stepped care places anti inflammatory treatment at every step rather than relying on reliever alone. Short acting beta 2 agonists are the classic rapid relievers; long acting beta 2 agonists are used only in combination with inhaled corticosteroids in asthma. Escalation adds or raises controller therapy: increased inhaled corticosteroid, the combination inhaler used as both maintenance and reliever, long acting muscarinic antagonists, leukotriene receptor antagonists blocking the cysteinyl leukotriene arm, and, in severe allergic or eosinophilic asthma under specialist care, biologic therapies against IgE, interleukin 5 pathways or related Th2 targets, with oral corticosteroids a last step whose long term harms are a standing warning. Acute severe asthma is emergency therapy: oxygen where needed, repeated inhaled bronchodilators, systemic corticosteroids, inhaled ipratropium and intravenous magnesium in severe cases under hospital care.

Asthma stepped care, in principle

Every step: inhaled corticosteroid based controller,
a reliever plan, technique and adherence checked,
triggers addressed, action plan in the patient's hands

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Step up if control is poor: raise the controller,
add a long acting bronchodilator in combination,
consider leukotriene or muscarinic additions

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Severe asthma: specialist review, phenotype assessed,
biologic therapy matched to the inflammatory type

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Step down only when control has been sustained,
never by withdrawing anti inflammatory treatment

2.2 Chronic obstructive airways disease

Chronic obstructive airways disease, universally taught as chronic obstructive pulmonary disease or COPD, is progressive airflow limitation that is not fully reversible, encompassing chronic bronchitis, defined clinically by chronic productive cough, and emphysema, defined anatomically by destruction of alveolar walls and enlargement of air spaces. Tobacco smoking is the dominant cause, with occupational dusts and fumes, biomass smoke and air pollution the other inhaled causes, and inherited alpha 1 antitrypsin deficiency the genetic cause to name, producing early emphysema. The pathophysiology is chronic inflammation of airways, parenchyma and vessels driven by the inhaled insult, with a protease and antiprotease imbalance destroying alveolar tissue, small airway fibrosis narrowing the bronchioles, and mucus gland hypertrophy producing the bronchitis picture. Presentation is progressive exertional breathlessness, chronic cough and sputum, wheeze and frequent infective exacerbations in a patient with a significant exposure history. Diagnosis requires spirometry showing persistent airflow obstruction after a bronchodilator, distinguishing COPD from asthma by history, variability and reversibility, though the two may coexist; severity and exacerbation history guide therapy.

Pharmacotherapy relieves symptoms and reduces exacerbations, but only stopping the exposure, above all smoking cessation, changes the underlying course, and cessation support is a pharmacotherapeutic intervention in its own right. Bronchodilators are the symptomatic foundation, the reverse emphasis from asthma: short acting beta 2 agonists and short acting muscarinic antagonists for relief, long acting beta 2 agonists and, centrally, long acting muscarinic antagonists for maintenance, combined where symptoms persist. Inhaled corticosteroids are added only for patients with frequent exacerbations and an eosinophilic pattern, weighed against pneumonia risk in this population. Exacerbations are treated with increased bronchodilators, a short course of systemic corticosteroids, antibiotics where infection criteria are met, controlled oxygen and ventilatory support where required.

Feature	Asthma	COPD
Typical onset	Often childhood or young adult, atopic background	Later life, after prolonged inhaled exposure
Airflow obstruction	Variable and reversible	Persistent and incompletely reversible
Core pathology	Airway inflammation with hyperresponsiveness	Airway and parenchymal inflammation, alveolar destruction
Controller foundation	Inhaled corticosteroids	Long acting bronchodilators
Disease modifying step	Control of inflammation, trigger avoidance	Stopping the exposure, above all smoking cessation

Asthma and COPD: opposite foundations

ASTHMA	SHARED GROUND	COPD
Airway inflammation with hyperresponsiveness Variable, reversible obstruction Foundation: inhaled corticosteroids	Wheeze, breathlessness and cough Inhaler technique and adherence decide real world control Exacerbations treated promptly in both	Airway and alveolar damage from inhaled exposures Persistent, incompletely reversible obstruction Foundation: long acting bronchodilators

Smoking cessation is the one step that changes the course of COPD

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Figure: Opposite foundations. Asthma therapy is built on anti inflammatory control, COPD therapy on bronchodilation and stopping the exposure.

2.3 Drug induced pulmonary diseases

Drug induced pulmonary disease is lung injury caused by a medicine, and it belongs in this unit because the respiratory system is a recognised target of adverse drug reactions and because the pharmacist reviewing therapy is often the professional who connects a new lung picture with a drug timeline. The mechanisms are several and should be classified in an examination answer. Direct cytotoxic injury to alveolar and capillary cells, classically by cytotoxic agents such as bleomycin, whose pulmonary toxicity is its defining dose limiting harm, and by agents including methotrexate and cyclophosphamide, produces pneumonitis and, if injury continues, pulmonary fibrosis. Oxidative injury is the mechanism of bleomycin and of nitrofurantoin chronic lung toxicity and of amiodarone lung injury, amiodarone accumulating in lung tissue over long therapy. Immune and hypersensitivity mechanisms produce eosinophilic pneumonias and hypersensitivity pneumonitis patterns, with nitrofurantoin, sulfonamides and many other agents reported. Drugs also injure the lung functionally rather than structurally: beta blockers and aspirin and other non steroidal anti inflammatory drugs provoke bronchospasm, aspirin sensitive respiratory disease combining asthma, nasal polyps and NSAID sensitivity; opioids and sedatives depress ventilatory drive; ACE inhibitors produce the familiar chronic dry cough by bradykinin accumulation, a benign but adherence destroying effect whose recognition prevents needless investigation; and several agents cause pleural effusions or pulmonary oedema by fluid or permeability mechanisms. Presentation is non specific, new cough, breathlessness, fever, infiltrates on imaging or falling gas exchange appearing during therapy, and that non specificity is the diagnostic problem: drug induced disease is a diagnosis reached by the timeline of exposure, by exclusion of infection, cardiac failure and the underlying disease, and by improvement on withdrawal of the suspect drug, the dechallenge, with lung function tests, imaging, bronchoscopy and occasionally biopsy in support. Management is withdrawal of the offending drug as the essential step, corticosteroid therapy where inflammation is significant and progressive, supportive oxygen and ventilation as needed, documentation and reporting of the reaction, and a permanent record that prevents re exposure; fibrosis established before recognition may not reverse, which is why baseline and interval monitoring is standard for the highest risk agents.

2.4 Diabetes mellitus

Diabetes mellitus is chronic hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. Type 1 diabetes is autoimmune destruction of the pancreatic beta cells, usually presenting in the

young with absolute insulin deficiency and a liability to ketoacidosis. Type 2 diabetes, the large majority, combines insulin resistance in liver, muscle and adipose tissue with progressive beta cell failure: the resistant liver overproduces glucose, muscle takes up less, adipose tissue releases free fatty acids that worsen resistance, incretin responses are blunted, and beta cells that first compensate gradually fail. Acutely, marked hyperglycaemia causes osmotic diuresis, dehydration and the emergencies of diabetic ketoacidosis, chiefly in type 1, and the hyperosmolar hyperglycaemic state in type 2. Chronically, hyperglycaemia damages vessels, producing the microvascular complications, retinopathy, nephropathy and neuropathy, and accelerating macrovascular disease, the main cause of death. Type 1 presents rapidly with thirst, polyuria, weight loss and fatigue, sometimes first as ketoacidosis; type 2 is often silent for years and found on testing or through its complications. Diagnosis is by the standard blood glucose and glycated haemoglobin criteria, followed by classification, complication screening and cardiovascular risk assessment.

Pharmacotherapy is built on the physiology, and the classes are best presented as mechanism groups. Insulin replaces the missing hormone, is obligatory and lifelong in type 1 as regimens combining background and mealtime insulin, and is used in type 2 when other therapy no longer controls glycaemia or acutely in illness and pregnancy. Metformin, the usual first drug in type 2, reduces hepatic glucose production, does not cause weight gain or hypoglycaemia alone, and is limited by gastrointestinal intolerance and caution in advanced renal impairment. Sulfonylureas stimulate insulin secretion from functioning beta cells, carrying hypoglycaemia and weight gain as class cautions. Incretin based therapies work through the gut hormone pathway: DPP 4 inhibitors prolong endogenous incretin action and are weight neutral, while GLP 1 receptor agonists enhance glucose dependent insulin secretion, suppress glucagon, slow gastric emptying and reduce appetite, producing weight loss and, for several agents, proven cardiovascular benefit. SGLT2 inhibitors block renal glucose reabsorption, lowering glucose independently of insulin, and have transformed care through heart failure and kidney protection, with genital mycotic infection and volume effects as counselling points. Therapy in type 2 is stepped and individualised: lifestyle and metformin at the foundation, then classes added according to response, weight, hypoglycaemia risk, kidney function and, decisively, the presence of cardiovascular or kidney disease, which directs choice toward the classes with proven organ protection.

Diabetes: drug classes matched to physiology

LESS GLUCOSE MADE	RAISE INSULIN ACTION	REMOVE GLUCOSE
<p>Metformin: less hepatic glucose output</p> <p>Thiazolidinediones: better insulin sensitivity</p> <p>Alpha glucosidase inhibitors: slower carbohydrate digestion</p>	<p>Sulfonylureas: more insulin secreted</p> <p>DPP 4 inhibitors: longer incretin action</p> <p>GLP 1 agonists: insulin up, glucagon down, appetite reduced</p> <p>Insulin: replacement</p>	<p>SGLT2 inhibitors: glucose lost in urine</p> <p>Heart failure and kidney protection beyond glycaemia</p> <p>Choice follows weight, kidney function and cardiovascular disease</p>

Therapy is stepped and individualised, never one drug for every patient

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Figure: Diabetes classes matched to physiology. The right combination follows which defect dominates and which organs need protection.

2.5 Thyroid diseases

The thyroid produces thyroxine and triiodothyronine under pituitary thyroid stimulating hormone control by negative feedback, and its pharmacotherapeutic diseases are the overactive and underactive states. Hyperthyroidism is excess thyroid hormone action, most commonly from Graves disease, an autoimmune disorder in which stimulating antibodies activate the TSH receptor, driving overproduction and enlargement, often with Graves eye disease; other causes are toxic multinodular goitre and toxic adenoma, where autonomous nodules escape control, and thyroiditis, where inflamed follicles release stored hormone transiently, a distinction that matters because antithyroid drugs do not treat the release phase. Hypothyroidism is hormone deficiency, most commonly from Hashimoto thyroiditis, autoimmune gland destruction, and otherwise from iodine deficiency, thyroid surgery or radioiodine, and medicines including amiodarone and lithium. Diagnosis in both directions reads TSH and free thyroxine together through the feedback logic, suppressed TSH with raised hormone in primary hyperthyroidism and raised TSH with low hormone in primary hypothyroidism, with antibody tests for the autoimmune causes and uptake scanning where the cause of hyperthyroidism is in doubt.

Pharmacotherapy of hyperthyroidism offers three routes to compare. Thionamide antithyroid drugs, carbimazole and its active metabolite methimazole, and propylthiouracil, block new hormone synthesis; a course may induce lasting remission in Graves disease, and the class carries rash and, rarely, agranulocytosis, whose warning symptoms of sore throat and fever every patient must know, and liver injury, the greater concern with propylthiouracil, reserved mainly for the first trimester of pregnancy and thyroid storm. Beta blockers control the adrenergic symptoms, tremor, palpitations and anxiety, while definitive therapy takes effect. Radioactive iodine destroys thyroid tissue, usually resulting in hypothyroidism that is then replaced; surgery is chosen for large goitres, suspicious nodules and patient preference after preparation with antithyroid drugs and iodide. Hypothyroidism treatment is replacement: levothyroxine restores the hormone pool, is titrated against TSH over weeks because of the long half life, and is lifelong in established primary failure, with counselling on consistent timing in relation to food and on absorption interactions with iron, calcium and other medicines, and dose review in pregnancy and changing therapy. Drug induced thyroid disease, from amiodarone above all, is a recognised monitoring duty linking this section back to Section 2.3.

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