

Unit 1: Nervous System

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Pharmacotherapeutics II, subject code MPP202T, is the second core theory subject of M.Pharm Pharmacy Practice, Semester 2, and the direct continuation of Pharmacotherapeutics-I (MPP102T) from Semester 1. It carries 4 credits and 60 hours of theory, taught at 4 hours per week, for 100 marks, and its five units are of equal length at 12 hours each in the PCI scheme. A code caution belongs at the head of the page: several copies of the PCI course of study table misprint this subject as MPP102T, duplicating the Semester 1 code; the correct code is MPP202T, as printed by Jamia Hamdard, Nitte, PTU, the RGPV Semester 2 unit text and the medipdf subject list, and MPP202T is used throughout these notes. Its scope, as the syllabus states it, is to enable students to understand the different treatment approaches in managing various disease conditions, and to impart skills in optimizing drug therapy by individualizing the treatment plan through evidence based medicine. Its objectives follow: to describe the rationale for drug therapy, to summarize the therapeutic approach for various disease conditions with reference to the latest evidence, to discuss clinical controversies and evidence based medicine, to prepare individualized therapeutic plans based on diagnosis, and to identify the patient specific parameters relevant in initiating and monitoring therapy. The subject head over the whole syllabus defines the method of every section: etiopathogenesis and pharmacotherapy of diseases of the following systems. The units follow the syllabus order: Unit 1 nervous system, Unit 2 psychiatric disorders and renal system, Units 3 and 4 infectious diseases with gynecological disorders closing Unit 4, and Unit 5 oncology. Every disease is therefore taught in the same order as in Part I: what it is, what causes it, how it develops, how it presents, how it is recognised, and how its drug therapy works in principle. One discipline runs through the whole subject: these notes teach diseases and their drug therapy at standard textbook level, naming the standard drug classes and their roles and describing stepped management qualitatively. No doses or dose schedules are stated, and no patient specific advice is given. Drug therapy is always individualised on diagnosis, severity, comorbidity and response, which is the habit this subject exists to teach.

1.1 The pharmacotherapeutics method, continued

The method of this subject is the method of Part I, applied to a new set of systems, and it is restated here because the nervous system, psychiatric and infectious topics test it in new ways. The method begins with etiopathogenesis: the causes of the disease and the mechanism by which those causes produce the disordered physiology, because rational drug therapy is therapy aimed at a mechanism. In this subject the mechanisms are often balances that have tipped: excitation against inhibition in epilepsy, dopamine against acetylcholine in Parkinson disease, transmitter excess or deficit in the psychiatric disorders, perfusion against demand in stroke, and host defence against microbial growth in the infectious units. The second stage is presentation and diagnosis: the symptoms, signs and investigations that establish which disease is present, how severe it is, and what else will condition therapy, including other diseases, other medicines, organ function, allergy and adherence. The third stage is the therapeutic plan itself, built in the stepped pattern most conditions in this subject follow: non drug measures and treatment of causes first and throughout, then drug therapy started with the class or classes the mechanism and the evidence put first, then review against stated goals, then addition, substitution or escalation where the response is inadequate, with long term therapy, prevention of complications and monitoring for adverse effects planned from the start rather than added later. Two cautions run through the whole subject. Many of its drug classes act on the brain and carry dependence, withdrawal and interaction burdens that must be planned for, not

discovered. And in the infectious units the patient is not the only unit of concern: every antimicrobial choice also acts on the microbial population and on future patients, which is why rational use opens those units.

The pharmacotherapeutics method used in every section

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graph TD; A[Etiopathogenesis: cause and mechanism of the disease] --> B[Presentation and diagnosis: severity, comorbidity, organ function, other medicines, allergy, adherence]; B --> C[Plan: non drug measures and causes treated throughout, first drug classes matched to the mechanism]; C --> D[Review against goals: response, adverse effects, clinical and laboratory indices over time]; D --> E[Adjust: add, substitute or escalate in steps, prevent complications, plan long term monitoring];
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Etiopathogenesis: cause and mechanism of the disease

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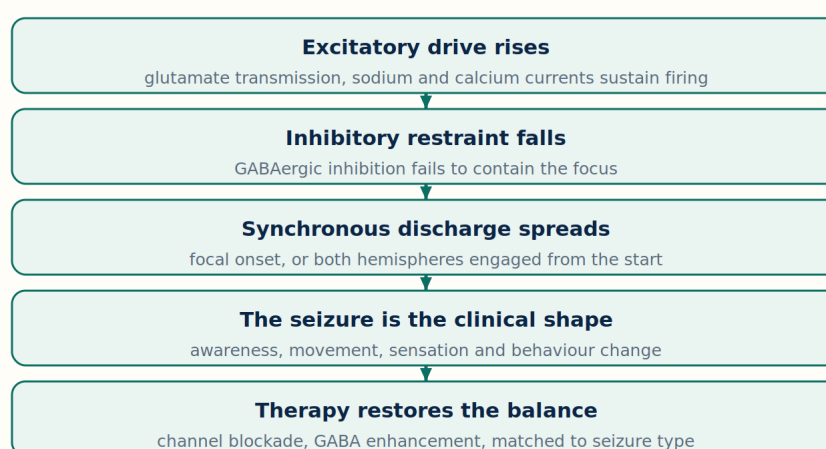
1.2 Epilepsy

Epilepsy is a chronic disorder of the brain characterised by an enduring predisposition to generate epileptic seizures, which are transient episodes of abnormal, excessive or synchronous neuronal activity producing changes in awareness, movement, sensation or behaviour. A single seizure is not epilepsy: seizures provoked by fever in young children, by metabolic disturbance, by alcohol withdrawal or by acute brain injury are acute symptomatic events, and the diagnosis of epilepsy rests on recurrence risk and on the pattern over time. The aetiology is classified by cause: structural causes such as cortical malformation, head injury, stroke, tumour and hippocampal sclerosis; genetic causes, prominent in the childhood onset syndromes; infectious causes, including the parasitic and viral infections of Unit 4; metabolic causes; immune causes; and a large group in which no cause is found. The pathophysiology is an imbalance between excitation and inhibition in a neuronal network: excitatory glutamatergic transmission and voltage gated sodium and calcium currents that sustain firing outweigh inhibitory GABAergic restraint, a focus of hyperexcitable neurons recruits its neighbours, and the seizure is the clinical shape of that synchronous discharge spreading or generalising. Seizures classify by onset: focal seizures begin in one hemisphere and may preserve or impair awareness, with motor, sensory, autonomic or psychic features pointing to the site of origin, and may evolve to bilateral convulsive seizures; generalised seizures engage both hemispheres from the start and include tonic clonic, absence, myoclonic, tonic and atonic forms; combined and unknown onset forms complete the scheme. Presentation follows the type: the generalised tonic clonic seizure with loss of consciousness, tonic stiffening, clonic jerking, tongue biting and incontinence and a postictal phase of confusion and sleep is the form the public recognises, while absence seizures are brief staring spells in a child and focal seizures may be no more than a rising epigastric sensation, a smell that is not there, or rhythmic jerking of one hand. Diagnosis is first a witness history, which outweighs every test, supported by electroencephalography showing epileptiform activity, brain imaging seeking a structural cause, and blood tests excluding metabolic provocation; the differential diagnosis of transient loss of consciousness, syncope and psychogenic non epileptic events above all, must be settled before lifelong therapy is built on the label.

Pharmacotherapy aims at complete seizure freedom without intolerable adverse effects, and its first principle is that the drug is chosen for the seizure type and syndrome, not for epilepsy in the abstract, because some classes that control focal seizures aggravate absence and myoclonic seizures. Antiseizure medicines act by a small set of mechanisms that map onto the pathophysiology: blockade of voltage gated

sodium channels stabilising the inactive state and limiting sustained firing, the mechanism of carbamazepine, phenytoin, lamotrigine and lacosamide among others; enhancement of GABAergic inhibition, by benzodiazepines, barbiturates, vigabatrin and tiagabine; blockade of calcium channels, ethosuximide acting on the thalamic currents of absence seizures; and mixed or novel mechanisms, valproate acting broadly across several targets, levetiracetam acting on synaptic vesicle protein, and perampanel antagonising glutamate receptors. Broad spectrum agents, valproate, lamotrigine and levetiracetam prominent among them, serve where the syndrome is generalised or unsettled, while narrower agents serve focal epilepsy. Treatment begins with a single drug, monotherapy, reviewed against seizure frequency and tolerability, and substitution precedes addition where the first drug fails; rational polytherapy combining complementary mechanisms follows in resistant epilepsy under specialist care, alongside the non drug options of epilepsy surgery for a resectable focal cause, vagus nerve stimulation and dietary therapy in defined settings. Status epilepticus, continuous or rapidly repeating seizures without recovery, is the emergency of the topic, treated in hospital by a staged sequence of benzodiazepine therapy, then a loading antiseizure medicine, then anaesthetic infusion under critical care. Counselling is pharmacotherapy in this subject: adherence above all, since missed therapy is the commonest cause of breakthrough seizures; the teratogenicity of valproate and the enzyme inducing agents and the folate and contraception planning that accompany prescribing to women of childbearing potential; enzyme induction and inhibition interactions, which reach contraceptives, anticoagulants and many other medicines; gradual withdrawal only under supervision after sustained freedom, never abrupt cessation; and safety advice on driving, bathing, heights and supervision proportionate to seizure control.

Epilepsy: the excitation and inhibition balance



The right class follows the seizure type and syndrome, not the label alone

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Figure: Epilepsy as a tipped balance. Antiseizure classes restore the balance, and the right class follows the seizure type.

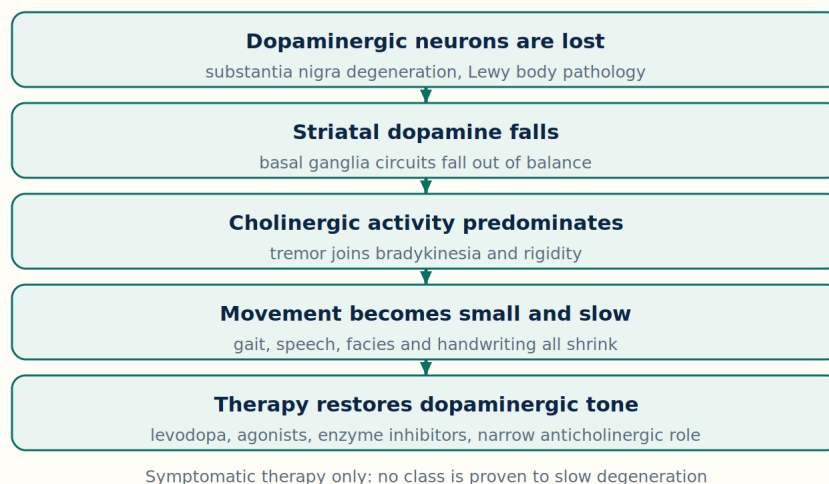
1.3 Parkinson disease

Parkinson disease is a progressive neurodegenerative disorder whose core pathology is the loss of dopaminergic neurons of the substantia nigra pars compacta, with Lewy bodies, aggregates rich in alpha synuclein, as its pathological signature in surviving neurons. Its cause is incompletely understood and is taught as a convergence: ageing as the strongest risk factor, genetic forms in a minority, environmental exposures including certain pesticides, and protective associations such as caffeine intake noted in epidemiology, over a long prodromal phase in which non motor features precede the motor diagnosis by years. The pathophysiology is a striatal transmitter imbalance: dopamine depletion in the putamen removes the normal dopaminergic modulation of the basal ganglia circuits, the direct and indirect pathways fall out of balance, thalamic drive to cortex is excessively inhibited, and movement becomes small, slow and hard to

initiate, while relatively preserved cholinergic activity in the striatum contributes the tremor, which is why the two classical drug strategies are to restore dopaminergic transmission and to restrain cholinergic excess. Degeneration is not confined to the nigra: autonomic, limbic and cortical involvement explains the non motor burden, constipation, urinary symptoms, orthostatic hypotension, depression, sleep disturbance and, late, dementia and psychosis. Presentation is classically asymmetric at onset: rest tremor, bradykinesia as the defining feature without which the diagnosis is not made, rigidity, and postural instability appearing later, with a shuffling, festinating gait, reduced arm swing, masked facies and soft, monotonous speech; drug induced parkinsonism, from the dopamine blocking agents of Section 2.5, and the atypical parkinsonian syndromes must be distinguished from it, since their course and treatment differ. Diagnosis is clinical, supported by a clear and sustained response to dopaminergic therapy, with imaging reserved for atypical features, and red flags of early falls, poor levodopa response, prominent early autonomic failure or dementia redirecting the diagnosis.

Pharmacotherapy replaces or mimics the missing dopamine and is symptomatic: no class is proven to slow the underlying degeneration, and examinations reward that honesty. Levodopa, the dopamine precursor that crosses the blood brain barrier where dopamine itself cannot, given always with a peripheral decarboxylase inhibitor that prevents its breakdown outside the brain, is the most effective symptomatic therapy and the benchmark against which the other classes are judged; its long term complications define the advanced disease, wearing off as each dose effect shortens, unpredictable on and off fluctuations, and dyskinesias, involuntary movements at peak effect. Dopamine agonists mimic dopamine at its receptors and carry a longer, smoother action, used early in younger patients and as adjuncts later, with somnolence, ankle oedema, hallucinations and the impulse control disorders, gambling, hypersexuality and compulsive shopping, as their distinctive counselling burden. Monoamine oxidase B inhibitors slow central dopamine breakdown and serve early mild disease and wearing off. Catechol O methyltransferase inhibitors block peripheral levodopa breakdown and extend its effect in fluctuation. Amantadine serves dyskinesia and mild early symptoms. Anticholinergic agents retain a narrow place for tremor in younger patients, limited by confusion, urinary retention, constipation and glaucoma risk, and are avoided in older patients. Advanced therapies for disabling fluctuation include infusion and intestinal gel delivery of dopaminergic therapy, apomorphine by injection or infusion, and deep brain stimulation in selected patients. Management throughout treats the whole disease: exercise and physiotherapy, speech and swallowing review, treatment of depression, constipation, sleep disturbance and orthostatic symptoms, and review of medicines that block dopamine or worsen confusion, because in this condition the prescription list is part of the pathology.

Parkinson disease: striatal transmitter balance



Symptomatic therapy only: no class is proven to slow degeneration

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Figure: The striatal balance. Dopaminergic therapy restores tone, anticholinergic therapy restrains the other side in a narrow role.

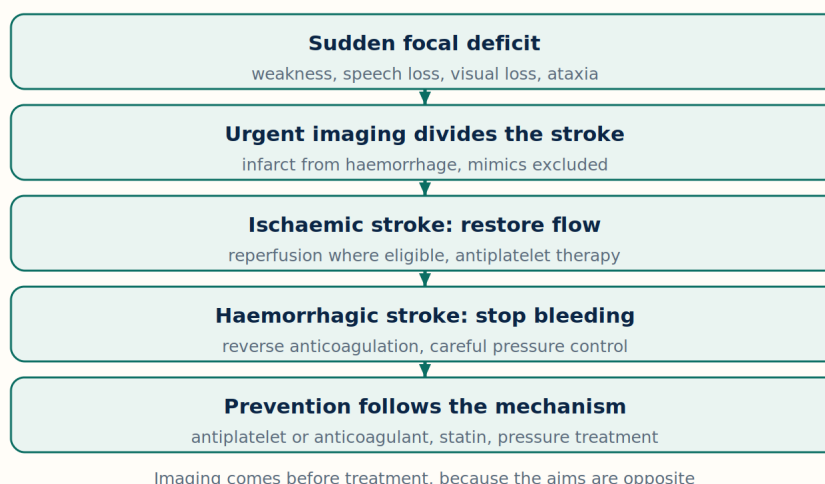
1.4 Stroke

Stroke is an acute focal neurological deficit of vascular cause, lasting because brain tissue has been injured, and it divides into two diseases with opposite treatments, which is why the division governs the whole topic. Ischaemic stroke, the large majority, follows occlusion of a cerebral artery, by thrombosis on an atherosclerotic plaque in a large artery, by embolism from the heart, atrial fibrillation the cardinal source, or from the carotid arteries, or by small vessel disease occluding a penetrating artery and producing a lacunar infarct. Haemorrhagic stroke follows rupture of a cerebral vessel, intracerebral haemorrhage from chronic hypertension damaging small deep vessels or from amyloid angiopathy in older patients, and subarachnoid haemorrhage from a ruptured aneurysm presenting as a thunderclap headache. The pathophysiology of ischaemia is a perfusion gradient: a core of tissue below the flow level needed for survival infarcts rapidly, surrounded by the penumbra, tissue that is electrically silent but still viable and salvageable if flow is restored in time, while excitotoxic glutamate release, calcium entry, free radical injury and inflammation extend the damage over the hours that follow; in haemorrhage the injury is the haematoma itself, raised intracranial pressure and, in subarachnoid bleeding, delayed vasospasm. Presentation is sudden and focal: weakness or numbness of face, arm or leg, speech disturbance, visual loss, ataxia and, in haemorrhage, headache and vomiting with reduced consciousness; transient ischaemic attack is the same mechanism with symptoms resolving without infarction, and is a warning event demanding urgent secondary prevention, not reassurance. Diagnosis in the acute phase is imaging first: urgent brain scanning separates infarct from haemorrhage and excludes mimics such as hypoglycaemia, seizure and tumour, and vascular and cardiac investigation then seeks the mechanism, rhythm monitoring for atrial fibrillation, carotid imaging and echocardiography among them, because prevention is mechanism specific.

Pharmacotherapy divides by phase. In acute ischaemic stroke the aim is reperfusion: thrombolytic therapy dissolving the occluding clot in eligible patients assessed in a specialist pathway, mechanical thrombectomy retrieving clot from a large vessel occlusion in selected patients, and early antiplatelet therapy with aspirin once haemorrhage is excluded; anticoagulation is not acute infarct therapy, and blood pressure is managed cautiously under protocol because the penumbra depends on perfusion. In acute haemorrhagic stroke the aims reverse: stop the bleeding and protect the brain, with urgent reversal of anticoagulation where present, careful blood pressure control under protocol, neurosurgical assessment and nimodipine in subarachnoid haemorrhage against vasospasm as its specific measure. Secondary prevention is the long pharmacotherapy of the topic and is built from the mechanism: antiplatelet therapy for non cardioembolic

ischaemic stroke, dual therapy for a limited early period in defined minor stroke and high risk transient attack under guideline direction, then single agent therapy long term; anticoagulation for atrial fibrillation, the heparins and warfarin tradition now largely succeeded by the direct oral anticoagulants in most patients, begun after the infarct at a timing set by its size and haemorrhage risk; high intensity statin therapy for atherosclerotic disease; treatment of hypertension as the single most important long term measure once the acute phase has passed, with the classes of Part I; diabetes and smoking addressed as in Part I; and carotid intervention for significant symptomatic stenosis in selected patients. Rehabilitation, swallowing assessment, prevention of venous thromboembolism and of aspiration, and treatment of post stroke depression and spasticity complete the plan.

Stroke: two diseases, opposite treatments



Imaging comes before treatment, because the aims are opposite

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Figure: Two diseases, opposite treatments. Imaging divides them before any therapy is chosen.

1.5 Headache

Headache is pain arising from the pain sensitive structures of the head, the vessels, meninges, muscles and nerves, the brain parenchyma itself being insensitive, and its pharmacotherapeutics begins with the division into primary headaches, in which the headache is the disease, and secondary headaches, in which it is the symptom of a cause that must be found. The secondary causes that must never be missed frame every assessment: subarachnoid haemorrhage, meningitis and encephalitis of Unit 4, raised intracranial pressure from tumour or thrombosis of the cerebral veins, giant cell arteritis in older patients, acute glaucoma of Part I, uncontrolled hypertension and medication overuse itself. Their warning pattern is the red flag history: thunderclap onset, fever with neck stiffness, new headache in later life, progressive pattern, headache with focal signs, papilloedema, seizure, immune suppression or pregnancy, and any such feature converts the consultation from symptomatic treatment to investigation. Migraine, the primary headache that dominates pharmacotherapy teaching, is a disorder of brain excitability and trigeminovascular activation: cortical spreading depression underlies the aura of transient focal symptoms, most often visual, and activation of the trigeminal system with release of calcitonin gene related peptide and other mediators produces vasodilation, neurogenic inflammation and the throbbing, usually unilateral pain with nausea, vomiting and sensitivity to light, sound and smell that defines the attack; triggers include hormonal change, missed meals, sleep disturbance, stress and its release, and specific foods and alcohol in susceptible patients. Tension type headache, the commonest headache of all, is a pressing, bilateral, non disabling pain linked to pericranial muscle tenderness and central sensitisation in its chronic form. Cluster headache is the severe, strictly unilateral pain around the eye with autonomic features, tearing, nasal congestion and restlessness, occurring in clusters of attacks, most often in men, and is separated here because its acute

and preventive therapies are distinctive.

Feature	Migraine	Tension type headache	Cluster headache
Site and character	Often unilateral, throbbing	Bilateral, pressing	Strictly unilateral, around the eye, severe
Accompaniments	Nausea, light and sound sensitivity, aura in some	Few accompaniments	Tearing, nasal congestion, restlessness
Course	Attacks lasting hours to days	Episodic or chronic, non disabling pattern	Attacks in clusters over weeks

Diagnosis of the primary headaches is clinical, from a structured history of onset, site, character, duration, associated features, triggers and medicine use; imaging is for red flags and atypical patterns, not for the typical migraineur, and a headache diary is the practical instrument that settles pattern, triggers and overuse. Pharmacotherapy of migraine divides, as gout did in Part I, into two questions that must never be merged: treating the attack, and preventing attacks. Acute treatment is stepped: simple analgesia with paracetamol or a non steroidal anti inflammatory drug taken early in the attack, with an antiemetic where nausea and gastric stasis impair absorption; then the migraine specific triptans, serotonin receptor agonists that constrict the dilated cranial vessels and block trigeminal mediator release, contraindicated in ischaemic heart disease and stroke by their vasoconstrictor action; and the newer non vasoconstrictor acute classes, the gepants antagonising the calcitonin gene related peptide receptor and the ditans acting on a serotonin receptor subtype without vasoconstriction, serving patients in whom triptans fail or are unsafe. Preventive therapy is offered where attacks are frequent, disabling or poorly controlled, and is judged over months, not attacks: the established oral classes are beta blockers, the antiseizure medicines topiramate and valproate, the latter carrying the teratogenicity caution of Section 1.2, amitriptyline, and candesartan among the renin angiotensin agents, while the calcitonin gene related peptide monoclonal antibodies and preventive gepants are the modern targeted option for patients failing the older classes, and botulinum toxin serves defined chronic migraine under specialist care. Cluster headache is treated acutely with high flow oxygen and a rapidly acting triptan preparation and prevented during the cluster principally with verapamil under cardiac monitoring. Medication overuse headache closes the topic and is its pharmacist centrepiece: frequent use of acute headache medicines, triptans, combination analgesics and opioids above all, perpetuates and chronifies headache, and its treatment is education, withdrawal of the overused medicine with a bridge plan, and prevention, a cure that begins with recognising that the treatment has become the cause.

1.6 Alzheimer disease

Alzheimer disease is the commonest cause of dementia, a progressive neurodegenerative disorder in which memory and then all cognitive domains fail as neurons and synapses are lost, and it is taught here as the exemplar of the dementias, with vascular dementia, dementia with Lewy bodies and frontotemporal dementia as the comparison set. Its pathology carries two signature lesions: extracellular amyloid plaques formed from amyloid beta peptide cleaved abnormally from its precursor protein, and intracellular neurofibrillary tangles of hyperphosphorylated tau protein, with loss of cholinergic neurons of the basal forebrain an early and therapeutically important feature, since acetylcholine underpins attention and memory encoding. Risk is dominated by age, with genetic risk carried by the apolipoprotein E variants and rare deterministic mutations in the familial early onset forms, and vascular and lifestyle factors, hypertension, diabetes, smoking, inactivity and low cognitive engagement, shaping incidence at population level. The clinical course is insidious: early failure of episodic memory, losing recent events while remote memory survives, then language, visuospatial and executive failure, disorientation, and in later stages behavioural and psychological symptoms, agitation, apathy, wandering, sleep reversal and psychosis, with

dependence for all daily activities at the end. Diagnosis is a clinical synthesis of history from patient and informant, cognitive testing, exclusion of reversible causes by blood tests including thyroid function and vitamin B12, and brain imaging, with specialist biomarker assessment in atypical and early onset cases; the reversible mimics, depression presenting as pseudodementia, delirium, hypothyroidism of Part I and drug effects from the anticholinergic and sedative burden of the medicine list, must be excluded first, and reviewing that list is again a pharmacist act.

Pharmacotherapy is modest and must be presented honestly: the established classes improve symptoms for a time and do not halt the underlying degeneration. Cholinesterase inhibitors, donepezil, rivastigmine and galantamine, increase cholinergic transmission by blocking acetylcholine breakdown and serve mild to moderate disease, with gastrointestinal effects, bradycardia and syncope risk as their cautions and a cholinergic rationale that forbids casual co prescription of strongly anticholinergic medicines. Memantine, an NMDA receptor antagonist moderating glutamatergic excitotoxicity, serves moderate to severe disease and may be combined with a cholinesterase inhibitor. The anti amyloid monoclonal antibodies are the first class aimed at the pathology itself, offered in some health systems to carefully selected early patients under specialist programmes with imaging monitoring for their distinctive brain swelling and bleeding risks; their place is presented here as emerging and selective, not as standard therapy for established dementia. Much of the real pharmacotherapy of the topic is subtraction and symptom care: deprescribing anticholinergics and sedatives that worsen cognition, treating depression, pain, constipation and sleep disturbance that present as behaviour, reserving antipsychotic medicines for severe behavioural symptoms with risk, time limited and reviewed, because of their stroke and mortality warnings in dementia, and supporting carers, whose health determines how long home care survives. Vascular risk factor treatment protects the vascular contribution that so often coexists.

1.7 Neuralgias and pain pathways and pain management

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, and this closing Unit 1 head teaches first the pathways, then the neuralgias as its named clinical patterns, then the general management framework that Units 3 to 5 will reuse for cancer pain. Nociceptive pain begins in peripheral nociceptors responding to mechanical, thermal and chemical stimuli, intensified by the inflammatory mediators of Part I; signals travel in fast myelinated fibres carrying sharp, localised pain and slow unmyelinated fibres carrying dull, diffuse pain, synapse in the dorsal horn where transmission is modulated by descending pathways from the brainstem using serotonin, noradrenaline and endogenous opioids, and ascend to thalamus and cortex where pain is perceived and given emotional colour.

Neuropathic pain is different in kind: pain arising from a lesion or disease of the somatosensory system itself, generated by ectopic firing in damaged nerves, peripheral and central sensitisation, and loss of inhibitory control, described by patients as burning, shooting, electric or stabbing, with allodynia, pain from a stimulus that should not hurt, as its signature sign. The neuralgias are the named neuropathic syndromes: trigeminal neuralgia, paroxysms of severe electric pain in a trigeminal division triggered by touch, chewing or a breeze, most often from vascular compression of the nerve root and sometimes secondary to multiple sclerosis or tumour; postherpetic neuralgia, persistent burning pain in the dermatome of a healed shingles eruption, commonest in older and immune suppressed patients and preventable in part by vaccination and early antiviral treatment of the acute eruption; painful diabetic neuropathy, the length dependent burning and lancinating pain of feet and legs in long standing diabetes; and occipital and glossopharyngeal neuralgias completing the named set. Diagnosis is by the pain history and its descriptors, distribution in a nerve or dermatomal territory, sensory examination, and investigation for the underlying lesion where the pattern demands it; pain assessment itself, site, character, intensity on a scale the patient can use, timing, aggravating and relieving factors and effect on sleep, mood and function, is the diagnostic foundation of all pain management.

Pharmacotherapy follows the pain type, and confusing the two ladders is the examination trap. Nociceptive pain is treated on the analgesic ladder principle in its general form: non opioid analgesia first, paracetamol and non steroidal anti inflammatory drugs matched to inflammatory contribution and to the gastrointestinal, renal and cardiovascular cautions of Part I; then opioid therapy for moderate to severe pain uncontrolled by those measures, weak and then strong opioids in the classical language, always with a laxative planned against opioid constipation, an antiemetic available, and review against function rather than pain score alone; with adjuvants for specific pains, corticosteroids for raised pressure and inflammatory compression, antispasmodics for colic, and the bone pain measures of Unit 5. Neuropathic pain does not answer reliably to that ladder, opioids included, and its first line classes are the adjuvants borrowed from Sections 1.2 and 2.2: tricyclic antidepressants, the serotonin and noradrenaline reuptake inhibitor duloxetine, and the gabapentinoids gabapentin and pregabalin binding calcium channel subunits and reducing transmitter release, with topical lidocaine and capsaicin preparations for localised peripheral pain, tramadol and strong opioids reserved and specialist guided, and carbamazepine holding a specific first place in trigeminal neuralgia, where its sodium channel blockade matches the paroxysmal mechanism. Non drug measures are co therapy, not garnish: physiotherapy and activity pacing, psychological therapy for the mood and catastrophising that amplify chronic pain, and interventional procedures and neurosurgery, microvascular decompression for trigeminal neuralgia among them, in selected refractory cases. The pharmacist duties that close the unit are safety duties: opioid stewardship, dependence and diversion awareness, interaction and sedation review, naloxone awareness in high risk co prescription, and the standing question of whether long term opioids are still earning their place.

Pain management: match the ladder to the pain type

Assess: type, site, character, intensity,
effect on sleep, mood and function

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Nociceptive pain: paracetamol and NSAID first,
opioid steps where severity demands, laxative
and antiemetic planned, review against function

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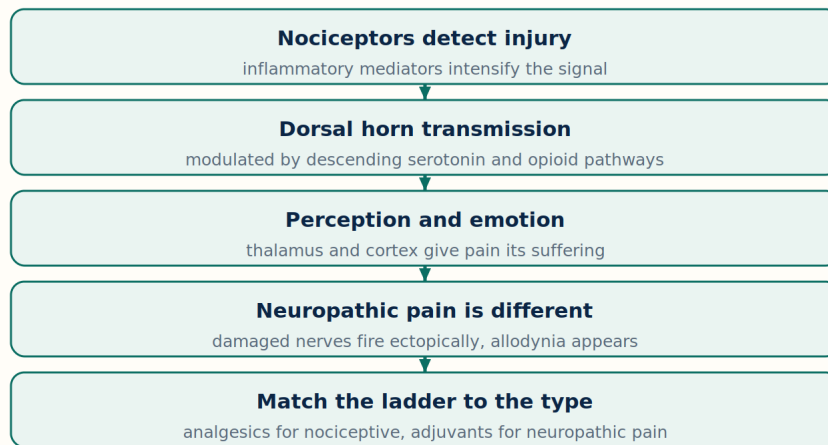
Neuropathic pain: adjuvant classes first,
tricyclic, duloxetine or gabapentinoid,
topical agents for localised pain

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Review: benefit against harm, dependence
and interactions watched, plan adjusted

Pain pathways and where therapy acts



Confusing the two ladders is the classic examination trap

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Figure: Pathways and ladders. Nociceptive and neuropathic pain travel differently and are treated differently.

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