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**PARASYMPATHETIC NERVOUS SYSTEM**

<b>MUSCARINIC RECEPTOR AGONISTS</b>	<b>Examples:</b>	Pilocarpine, ACh, Carbachol, Bethanechol
	<b>Uses:</b>	Treat glaucoma, increase GI motility, relieve urinary tension, reduce tachycardia
	<b>Mechanism:</b>	Activate muscarinic receptors (M1-M5, dependant on area)
	<b>Action:</b>	Bradycardia, decreased C.O., vasodilation, increased secretions, ciliary constriction (Aqueous humour drainage), smooth muscle contraction
	<b>Side effects:</b>	N/A
<b>MUSCARINIC RECEPTOR ANTAGONISTS</b>	<b>Examples:</b>	Atropine (Long acting), Hycosine, Homatropine (Short acting), Ipratropium (Quaternary - no CNS effects)
	<b>Uses:</b>	Anaesthesia - inhibit secretions & prevent vagal inhibition of the heart, ophthalmology - dilate pupil & paralyse lens for examination, treatment of anticholinesterase poisoning, relief of smooth muscle spasm, anti-emetic, reduce Parkinson's tremor, incontinence
	<b>Mechanism:</b>	Reversible competition of muscarinic receptors
	<b>Action:</b>	Block secretions, tachycardia, pupil dilation, decreased GI motility, smooth muscle relaxation, CNS excitation, tremor suppression
	<b>Side effects:</b>	Tachycardia, urinary retention, blurred vision, increased intraocular pressure CNS effects: restlessness, disorientation/confusion, hallucinations, amnesia

**AUTONOMIC GANGLIA**

<b>GANGLION AGONISTS</b>	<b>Examples:</b>	Nicotine/ACh
	<b>Uses:</b>	Not used clinically
	<b>Mechanism:</b>	Activate nicotinic receptors
	<b>Action:</b>	Increase BP, tachycardia, decrease gastric motility, CNS stimulation, vasoconstriction, release of ADH
	<b>Side effects:</b>	Technically all actions, as usually undesirable
<b>GANGLION BLOCKERS</b>	<b>Examples:</b>	Hexamethonium, Trimetaphan, excess Nicotine
	<b>Uses:</b>	Reduce hypertension (Mainly in surgery)
	<b>Mechanism:</b>	Block sympathetic and parasympathetic transmission via nicotinic receptor antagonism
	<b>Action:</b>	Cardiovascular - decreased BP via vessel dilation, GI tract - decreased motility, genito-urinary - impaired micturition, erectile failure, inhibition of ejaculation, eyes - impaired accommodation
	<b>Side effects:</b>	Constipation, impotence, blurred vision

**NEUROMUSCULAR JUNCTION**

<b>COMPETITIVE NMJ BLOCKERS</b>	<b>Examples:</b>	Tubocurarine, Gallamine, Pancuronium
	<b>Uses:</b>	Muscle relaxation during surgery
	<b>Mechanism:</b>	Block ACh transmission by competing for nicotinic receptors - antagonism
	<b>Action:</b>	Muscle relaxation (No initial fasciculation),
	<b>Side effects:</b>	Hypotension, histamine release, antagonised by anticholinesterases, myasthenia gravis patients are sensitive, tetanic fade (Tetanic muscle response poorly sustained)
<b>DEPOLARISING BLOCKERS</b>	<b>Examples:</b>	Suxamethonium, Decamethonium
	<b>Uses:</b>	Muscle relaxation during surgery
	<b>Mechanism:</b>	Activate nicotinic receptors → depolarisation & inactivation of sodium channels in muscle membrane, inhibiting action potentials
	<b>Action:</b>	Initial fasciculation, followed by muscle relaxation
	<b>Side effects:</b>	Hyperkalaemia, post-operative muscle pain, block may be enhanced by anticholinesterases, myasthenia gravis patients insensitive
<b>ANTI-CHOLINESTERASES</b>	<b>Examples:</b>	Edrophonium (Short acting), Neostigmine (Medium acting), Dyflos, Parathion (Long acting/irreversible)
	<b>Uses:</b>	Treatment of glaucoma & myasthenia gravis, reversing NMJ block (E.g. following anaesthesia), *insecticide
	<b>Mechanism:</b>	Hydroxylation of cholinesterase, inhibition of ACh breakdown - sustain cholinergic transmission
	<b>Action:</b>	Increased secretions & GI motility, restoration of NMJ activity, pupillary constriction (Decreased intraocular pressure)
	<b>Side effects:</b>	Muscle fasciculation, bradycardia, hypotension, bronchoconstriction, respiratory failure. TOXICITY - demyelination
<b>CHOLINESTERASE REACTIVATORS</b>	<b>Examples:</b>	Pralidoxime (All oxime compounds)
	<b>Uses:</b>	Treatment of organophosphate poisoning
	<b>Mechanism:</b>	Reactivation of cholinesterases by moving phosphate group
	<b>Action:</b>	ACh breakdown
	<b>Side effects:</b>	N/A

<b>OTHER NMJ BLOCKERS</b>	<b>Examples:</b>	1) Hemicholinium 2) Botulinium toxin 3) Streptomycin/other aminoglycoside antibiotics
	<b>Uses:</b>	Pre-anaesthetic - muscle relaxation, reduce amount of general anaesthetic needed
	<b>Mechanism:</b>	1) Inhibits choline uptake by cholinergic nerve terminals 2) Blocks ACh release (Long lasting) 3) Block calcium entry and therefore transmitter release
	<b>Action:</b>	Block neuromuscular transmission
	<b>Side effects:</b>	3) Weakness

### SYMPATHETIC NERVOUS SYSTEM

<b><math>\alpha</math>-ADRENOCEPTOR AGONISTS</b>	<b>Examples:</b>	1) Adrenaline, noradrenaline. 2) Phenylephrine( $\alpha$ 1) 3) Clonidine( $\alpha$ 2). 4) Dipivephrine( $\alpha$ 2, adrenaline pro-drug), Brimonidine( $\alpha$ 2)
	<b>Uses:</b>	1) Anaphylactic shock, cardiac arrest, prolong action of local anaesthetics 2) Nasal decongestant, 3) Anti-hypertensive 4) Treatment of open angle glaucoma
	<b>Mechanism:</b>	$\alpha$ 1 - phospholipase C activation, IP3 & DAG formation, increase in calcium release. $\alpha$ 2 - inhibit adenylate cyclase, decrease cAMP and decrease protein kinase A
	<b>Action:</b>	$\alpha$ 1 - smooth muscle contraction, vasoconstriction, decrease GI motility, promote glycogenolysis $\alpha$ 2 - inhibit noradrenaline and decrease lipolysis, decrease aqueous humour production
	<b>Side effects:</b>	Hypertension, reflex bradycardia
<b><math>\alpha</math>-ADRENOCEPTOR ANTAGONISTS</b>	<b>Examples:</b>	Phentolamine, Phenoxybenzamine, Prazosin( $\alpha$ 1), Ergot alkaloids
	<b>Uses:</b>	Antihypertensives, treatment of phaeochromocytomas
	<b>Mechanism:</b>	Block $\alpha$ -adrenoceptor mediated effects by direct competition (Exception is Phenoxybenzamine - non-competitive and irreversible)
	<b>Action:</b>	Increased sympathetic drive (Indirect), Vasodilation, decreased BP
	<b>Side effects:</b>	Nasal stuffiness, reflex tachycardia, postural hypotension, sodium & water retention, miosis (Pupillary constriction)
<b><math>\beta</math>-ADRENOCEPTOR AGONISTS</b>	<b>Examples:</b>	Adrenaline, Noradrenaline, Isoprenaline, Dobutamine( $\beta$ 1) Bisoprolol( $\beta$ 1), Salbutamol( $\beta$ 2), Salmeterol( $\beta$ 2).
	<b>Uses:</b>	Treatment of asthma, inhibition of preterm labour, treatment of acute congestive heart failure
	<b>Mechanism:</b>	Increase cAMP, activation of protein kinase A - protein phosphorylation. Prolonged opening of calcium channels in the heart ( $\beta$ 1)
	<b>Action:</b>	Increase heart rate and contractile force ( $\beta$ 1), vasodilation, smooth muscle relaxation, renin release, inhibition of histamine, increased glycogenolysis and lipolysis
	<b>Side effects:</b>	Hypokalaemia, tremor, hypotension, reflex tachycardia

<b>β-ADRENOCEPTOR ANTAGONISTS</b>	<b>Examples:</b>	Atenolol(β <sub>1</sub> ), Propranolol, Metoprolol, Oxprenolol (Partial antagonist)
	<b>Uses:</b>	Treatment of angina, hypertension, heart failure, glaucoma, atrial dysrhythmias
	<b>Mechanism:</b>	Block β-adrenoceptor mediated effects by direct competition. Activation of baroreceptor reflex
	<b>Action:</b>	Decreased cardiac output and sympathetic activity. Decrease BP, vasoconstriction
<b>INDIRECTLY ACTING</b>	<b>Side effects:</b>	Bradycardia, cold extremities, bronchospasm (Contraindicated in asthmatic patients)
	<b>Examples:</b>	1) Carbidopa, Disulfiram, α-Methyl Dopa 2) Reserpine 3) Tyramine, Amphetamine 4) Clonidine, Guanethidine 5) Cocaine, Tricyclic Antidepressants, β-oestradiol 6) Phenelzine (Non-selective), Selegiline (MAO-B selective)
	<b>Uses:</b>	Treatment of hypertension, angina and glaucoma
	<b>Mechanism:</b>	1) Inhibits NA synthesis 2) Inhibits NA storage 3) Displaces NA from vesicles 4) Inhibits NA release 5) Inhibits NA uptake 6) Inhibits NA metabolism
<b>INDIRECTLY ACTING</b>	<b>Action:</b>	Decreased heart rate and cardiac output, reduced sympathetic drive to the heart, decreases production of aqueous humour
	<b>Side effects:</b>	Bronchoconstriction, bradycardia, cardiac failure

### ANTIHYPERTENSIVES & ANTI-ANGINAS (Also see above)

<b>THIAZIDE DIURETICS</b>	<b>Examples:</b>	Benchroflumethiazide, metolazone
	<b>Uses:</b>	Long term control of hypertension
	<b>Mechanism:</b>	Increase sodium excretion → water loss → decreased blood volume → decreased cardiac output → decreased BP
	<b>Action:</b>	Long term, peripheral resistance is decreased after cardiac output and blood volume return to normal → decreased BP
<b>ATP-SENSITIVE K<sup>+</sup> CHANNEL OPENERS</b>	<b>Side effects:</b>	Hypokalaemia, hyperglycaemia, gout, impotence
	<b>Examples:</b>	Diazoxide, Minoxidil, Nicorandil
	<b>Uses:</b>	Hypertensive emergencies, treatment of severe hypertension
	<b>Mechanism:</b>	Block binding of ATP to K <sup>+</sup> channels, keeping them open → K <sup>+</sup> influx → hyperpolarisation → closing of L-type Ca <sup>2+</sup> channels
<b>ATP-SENSITIVE K<sup>+</sup> CHANNEL OPENERS</b>	<b>Action:</b>	Vasodilation → fall in peripheral resistance → decreased BP
	<b>Side effects:</b>	Hypertrichosis, acute hypotension

<b>CALCIUM CHANNEL BLOCKERS</b>	<p><b>Examples:</b> Nifedipine (L-type selective), verapamil, diltiazem</p> <p><b>Uses:</b> Long term control of hypertension, angina and arrhythmias</p> <p><b>Mechanism:</b> Antagonise calcium channels, preventing calcium influx</p> <p><b>Action:</b> Vasodilation</p> <p><b>Side effects:</b> Facial flushing, headache, dizziness</p>
<b>ORGANIC NITRATES</b>	<p><b>Examples:</b> 1) Sodium Nitroprusside 2) GTN (Short acting), ISMN (Long acting)</p> <p><b>Uses:</b> 1) Severe hypertensive crises 2) Angina</p> <p><b>Mechanism:</b> Activate guanylate cyclase → increase cGMP → activation of protein kinase G → phosphorylation of myosin light chains</p> <p><b>Action:</b> Relaxes vascular smooth muscle</p> <p><b>Side effects:</b> Headache, reflex tachycardia, nausea, vomiting, dizziness, increased CSF &amp; intraocular pressure, methanemoglobinaemia</p>
<b>ANGIOTENSIN CONVERTING ENZYME (ACE) INHIBITORS</b>	<p><b>Examples:</b> Captopril, Enalapril, Ramapril</p> <p><b>Uses:</b> Long term control of hypertension, treatment of congestive heart failure</p> <p><b>Mechanism:</b> Inhibit the renin-angiotensin pathway - prevent the conversion of angiotensin I to angiotensin II. Prevent bradykinin breakdown</p> <p><b>Action:</b> Vasodilation</p> <p><b>Side effects:</b> Can cause precipitous decrease in BP in patients with renal impairment, cough, hypokalaemia</p>
<b>ANGIOTENSIN II AT1 RECEPTOR ANTAGONISTS</b>	<p><b>Examples:</b> Losartan</p> <p><b>Uses:</b> Long term control of hypertension, treatment of congestive heart failure</p> <p><b>Mechanism:</b> Competitive antagonist at AT1 receptors</p> <p><b>Action:</b> Vasodilation</p> <p><b>Side effects:</b> Similar to ACE inhibitors, less likely to cause cough Still being evaluated.</p>
<b>RENIN INHIBITORS</b>	<p><b>Examples:</b> Aliskiren</p> <p><b>Uses:</b> Long term control of hypertension</p> <p><b>Mechanism:</b> Prevents the conversion of angiotensinogen to angiotensin I by binding to renin</p> <p><b>Action:</b> Vasodilation</p> <p><b>Side effects:</b> Dose-related GI effects (E.g. diarrhoea)</p>

**TREATMENT OF CONGESTIVE HEART FAILURE (Also see above)**

<b>LOOP DIURETICS</b>	<b>Examples:</b>	Furosemide, Bumetanide
	<b>Uses:</b>	Treatment of hypertension, pulmonary oedema, hypercalcaemia & heart failure. Reduce peripheral and pulmonary oedema.
	<b>Mechanism:</b>	Inhibition of Na/K/Cl transporter on thick ascending loop of Helme → loss of Na <sup>+</sup> and Cl <sup>-</sup> → water loss
	<b>Action:</b>	Reduce preload, vasodilation
	<b>Side effects:</b>	Hypokalaemia, alkalosis, gout, deafness
<b>CARDIAC GLYCOSIDES</b>	<b>Examples:</b>	Digoxin/Digitoxin
	<b>Uses:</b>	Treatment of congestive heart failure (Usually when associated with atrial arrhythmia)
	<b>Mechanism:</b>	Inhibits Na <sup>+</sup> /K <sup>+</sup> ATPase, causes an accumulation of intracellular Na <sup>+</sup> and Ca <sup>2+</sup>
	<b>Action:</b>	Increased force of cardiac contraction. Slowed A-V conduction. Decreased refractory period of atrial muscle (As they have more cholinergic innervation)
	<b>Side effects:</b>	Usually associated with excessive dosage, as they have a low therapeutic index. Anorexia, nausea, vomiting, diarrhoea, visual disturbances (Colour vision) cardiac arrhythmias
<b>PHOSPHODIESTE RASE (PDE) III INHIBITORS</b>	<b>Examples:</b>	Milrione, Enoximone
	<b>Uses:</b>	Treatment of congestive heart failure
	<b>Mechanism:</b>	Increase intracellular levels of cAMP, increase Ca <sup>2+</sup> influx
	<b>Action:</b>	Increase myocardial contractility
	<b>Side effects:</b>	Pro-dysrhythmic

**TREATMENT OF ARRHYTHMIAS (Also see above)**

<b>CLASS Ia ANTI-DYSRYHTHMICS</b>	<b>Examples:</b>	Quinidine, Disopyramide
	<b>Uses:</b>	Treatment of supraventricular and ventricular arrhythmias
	<b>Mechanism:</b>	Block voltage sensitive Na <sup>+</sup> channels
	<b>Action:</b>	Slow conduction velocity, prolong repolarisation
	<b>Side effects:</b>	-

CLASS Ib ANTI-DYSRHYTHMICS	<b>Examples:</b>	Lidocaine
	<b>Uses:</b>	Treatment of ventricular tachycardia, particularly following infarction
	<b>Mechanism:</b>	Rapidly associate with and dissociate from Na <sup>+</sup> channels (Brief block)
	<b>Action:</b>	Abort premature beats. Selectively block depolarised regions
	<b>Side effects:</b>	-
CLASS Ic ANTI-DYSRHYTHMICS	<b>Examples:</b>	Flecainide, Encainide
	<b>Uses:</b>	Treatment of atrial fibrillation and re-entrant ventricular dysarrhythmia
	<b>Mechanism:</b>	Slow association and disassociation from Na <sup>+</sup> channels (Constant block)
	<b>Action:</b>	Reduce general excitability
	<b>Side effects:</b>	Flecainide can cause death in patients with heart failure
CLASS III ANTI-DYSRHYTHMICS	<b>Examples:</b>	Amiodarone, sotalol
	<b>Uses:</b>	Treatment of arrhythmias
	<b>Mechanism:</b>	K <sup>+</sup> channel blocker
	<b>Action:</b>	Increase refractory period, prolong cardiac action potential
	<b>Side effects:</b>	Thyroid and pulmonary toxicity - long half life
ADENOSINE RECEPTOR AGONISTS (DYSRHYTHMIC)	<b>Examples:</b>	Adenosine
	<b>Uses:</b>	Treatment of paroxysmal supraventricular tachycardia
	<b>Mechanism:</b>	Activation of A <sub>1</sub> receptors in cardiac muscle, inhibits Ca <sup>2+</sup> influx, increases K <sup>+</sup> permeability
	<b>Action:</b>	Slows AV transmission, prolongs refractory period
	<b>Side effects:</b>	Dyspnoea, headache, flushing, chest tightness

**THE KIDNEY (Also see above)**

<b>ADH</b>	<p><b>Examples:</b> Vasopressin (ADH), Desmopressin</p> <p><b>Uses:</b> Treatment of diabetes insipidus</p> <p><b>Mechanism:</b> Binds to V2 receptors in collecting ducts, increases water reabsorption</p> <p><b>Action:</b> Antidiuresis</p> <p><b>Side effects:</b> Headaches, facial flushing, nausea. ADH may cause hypertension via V1 receptors in blood vessels</p>
<b>POTASSIUM SPARING DIURETICS</b>	<p><b>Examples:</b> 1) Spironolactone 2) Amiloride, Triamterene</p> <p><b>Uses:</b> Treatment of Conn's syndrome, severe heart failure, liver disease with ascites</p> <p><b>Mechanism:</b> 1) Antagonises aldosterone in kidney, increases Na<sup>+</sup> excretion. 2) Blocks Na<sup>+</sup> channels and decreases its permeability in distal nephron, increasing its excretion.</p> <p><b>Action:</b> Reduces K<sup>+</sup> excretion. Increases water loss.</p> <p><b>Side effects:</b> Severe hyperkalaemia in patients with renal impairment/those taking ACE inhibitors</p>
<b>CARBONIC ANHYDRASE INHIBITORS</b>	<p><b>Examples:</b> Acetazolamide</p> <p><b>Uses:</b> Diuretic (Not clinically used), treat glaucoma, reduce CSF pressure</p> <p><b>Mechanism:</b> Inhibits carbonic anhydrase. Increases HCO<sub>3</sub><sup>-</sup> excretion</p> <p><b>Action:</b> Diuresis</p> <p><b>Side effects:</b> Acidosis</p>
<b>OSMOTIC DIURETICS</b>	<p><b>Examples:</b> Mannitol</p> <p><b>Uses:</b> Treatment of cerebral oedema and high intraocular pressure</p> <p><b>Mechanism:</b> Filtered and excreted with an osmotic equivalent of water</p> <p><b>Action:</b> Water loss</p> <p><b>Side effects:</b> Hyponatremia</p>

URINE ACIDIFICATION	<b>Examples:</b>	Ammonium chloride, ascorbic acid
	<b>Uses:</b>	Promote excretion/retention of drugs
	<b>Mechanism:</b>	Weak acid; excreted via homeostasis
	<b>Action:</b>	Makes urine more acidic
	<b>Side effects:</b>	-
URINE ALKALISATION	<b>Examples:</b>	Sodium bicarbonate, potassium citrate
	<b>Uses:</b>	Promote excretion/retention of drugs
	<b>Mechanism:</b>	Metabolised to bicarbonate. Cations are excreted with bicarbonate
	<b>Action:</b>	Makes urine more alkaline
	<b>Side effects:</b>	-

### ANAESTHETICS

LOCAL ANAESTHETICS	<b>Examples:</b>	Lidocaine, prilocaine, bupivacaine*, benzocaine**, cocaine
	<b>Uses:</b>	Localised pain prevention. *Spinal anaesthesia (Epidural), **Surface anaesthesia only.
	<b>Mechanism:</b>	Penetrate nerves and block Na <sup>+</sup> channels
	<b>Action:</b>	Prevent generation of action potentials
	<b>Side effects:</b>	Hypersensitivity, light-headedness. Toxicity - respiratory/cardiac depression
GENERAL ANAESTHETICS	<b>Examples:</b>	Inhaled - Nitrous oxide, halothane, isoflurane, enflurane, desflurane. IV - Thiopentone, Propofol
	<b>Uses:</b>	Depress excitable tissues in surgery
	<b>Mechanism:</b>	Bind to hydrophobic regions of proteins; GABA-A receptors (Enhance), glutamate receptors (Inhibit), ion channels.
	<b>Action:</b>	Cause unconsciousness, respiratory depression, decreased BP (Via myocardial depression and vasodilation)
	<b>Side effects:</b>	Nausea and vomiting upon regaining consciousness. Serious side effects include cardiac dysrhythmia, but are rare

**ANALGESIA**

<b>OPIOIDS</b>	<b>Examples:</b>	Morphine, Heroin (Metabolised to morphine), codeine, fentanyl, methadone, tramadol*
	<b>Uses:</b>	Pain relief
	<b>Mechanism:</b>	Bind to MU receptor, activate G-protein, open K <sup>+</sup> channels, hyperpolarisation, inhibition of neurotransmitter release. *Also blocks re-uptake of neurotransmitter and 5-HT
	<b>Action:</b>	Analgesia, anxiety relief. Antagonised by Naloxone (Used to treat overdose)
	<b>Side effects:</b>	Nausea, constipation, dependence
<b>NSAIDS</b>	<b>Examples:</b>	Ibuprofen, Aspirin
	<b>Uses:</b>	Pain relief, anti-inflammatory
	<b>Mechanism:</b>	Block prostanoid (Inflammatory mediator) production via inhibition of cyclo-oxygenase. Decrease PGE formation, reducing sensory neural sensitisation.
	<b>Action:</b>	Decrease inflammation and therefore associated pain
	<b>Side effects:</b>	Indigestion, stomach ulcers
<b>NMDA ANTAGONISTS</b>	<b>Examples:</b>	Ketamine
	<b>Uses:</b>	Relief of severe pain
	<b>Mechanism:</b>	Blocks NDMA channels in spinal cord
	<b>Action:</b>	Blocks central sensitisation
	<b>Side effects:</b>	Also blocks NDMA receptors in brain - loss of memory and cognitive function
<b>TRIPTANS</b>	<b>Examples:</b>	Sumatriptan
	<b>Uses:</b>	Treatment of headaches
	<b>Mechanism:</b>	Blocks 5-HT receptors in cranial blood vessels
	<b>Action:</b>	Blood vessel constriction
	<b>Side effects:</b>	Migraine recurrence?

**PARKINSON'S DISEASE (Also see Muscarinic Antagonists)**

<b>DOPAMINE PRECURSOR</b>	<b>Examples:</b>	L-DOPA
	<b>Uses:</b>	Improve muscle rigidity and akinesia (& resting tremor to an extent)
	<b>Mechanism:</b>	Crosses blood-brain barrier. Converted to dopamine by DOPA decarboxylase
	<b>Action:</b>	Activates dopamine receptors
	<b>Side effects:</b>	Central: Dyskinesia, psychotic effects, decreased prolactin release, "on-off" effects. Peripheral: Hypotension, nausea
<b>DOPA DECARBOXYLASE INHIBITORS</b>	<b>Examples:</b>	Carbidopa
	<b>Uses:</b>	L-DOPA adjunct
	<b>Mechanism:</b>	Inhibits DOPA-decarboxylase - prevents peripheral dopamine formation
	<b>Action:</b>	Prevents peripheral side effects
<b>MAO-B INHIBITORS</b>	<b>Examples:</b>	Selegiline
	<b>Uses:</b>	L-DOPA adjunct
	<b>Mechanism:</b>	Inhibits monoamine oxidase
	<b>Action:</b>	Reduces dopamine breakdown in the CNS
<b>DOPAMINE RECEPTOR AGONISTS</b>	<b>Examples:</b>	Bromocriptine (D2), Apomorphine (D1&D2)
	<b>Uses:</b>	Useful in older patients/where L-DOPA is no longer effective
	<b>Mechanism:</b>	Activates dopamine receptors
	<b>Action:</b>	Reduces symptoms of Parkinson's disease
	<b>Side effects:</b>	Domperidone (A D2 antagonist and anti-emetic) must be taken with these drugs - does not cross the blood-brain barrier

**OTHER MOVEMENT DISORDERS**

<b>DOPAMINE ANTAGONISTS</b>	<b>Huntingdon's Chorea</b>	Uncontrolled movement as a result of a loss of striated neurones and therefore a lack of inhibition of dopamine neurones. Treated with dopamine antagonists.
	<b>Tics</b>	Sudden, stereotyped movements of unknown origin. Improved with the administration of dopamine antagonists.

**SCHIZOPHRENIA**

<b>TYPICAL NEUROLEPTICS</b>	<b>Examples:</b>	Chlorpromazine, Thioridazine, Flupenthixol, Haloperidol
	<b>Uses:</b>	Relieve positive symptoms of schizophrenia
	<b>Mechanism:</b>	Antagonise D2 receptors. Flupenthixol also antagonises histamine, muscarinic and $\alpha$ 1 receptors
	<b>Action:</b>	Reduce dopaminergic transmission in the brain
	<b>Side effects:</b>	Extrapyramidal - dystonia, akathisia. Tardive dyskinesia, hyperprolactinaemia, pseudo-parkinsonism, aplastic anaemia.
<b>ATYPICAL NEUROLEPTICS</b>	<b>Examples:</b>	1) Clozapine, Olanzapine, Quetiapine 2) Risperidone
	<b>Uses:</b>	Relieve both positive and negative symptoms of schizophrenia
	<b>Mechanism:</b>	1) Antagonise 5-HT <sub>2A/2C</sub> receptors. 2) Weak partial agonists at 5-HT <sub>1A</sub> receptors. Muscarinic and $\alpha$ 2 antagonists.
	<b>Action:</b>	1) Anti-psychotic action. 2) Increased dopamine release from neurones projecting to frontal cortex thought to relieve -ve symptoms
	<b>Side effects:</b>	Marked dribbling, weight gain, prolonged cardiac QT interval, agranulocytosis (Clozapine)
<b>3RD GENERATION ANTI-PSYCHOTIC</b>	<b>Examples:</b>	Aripiprazole
	<b>Uses:</b>	Improved control of symptoms of schizophrenia
	<b>Mechanism:</b>	1) D2 antagonists, 5-HT <sub>2A</sub> antagonist. 2) Weak 5-HT <sub>1A</sub> partial agonist - increased dopamine production.
	<b>Action:</b>	1) Anti-psychotic action. 2) Relief of negative symptoms
	<b>Side effects:</b>	Hypercholesterolaemia, hyperprolactinaemia

**ANTI-DEPRESSANTS**

<b>MAO INHIBITORS</b>	<b>Examples:</b>	Phenelzine (Non-selective), Moclobemide (MAO-B selective)
	<b>Mechanism:</b>	Inhibits monoamine oxidase. Intra-neuronal accumulation of noradrenaline and 5-HT. Increased amount of transmitter available for release.
	<b>Action:</b>	Increased monoaminergic transmission
	<b>Side effects:</b>	"The Cheese Reaction," postural hypotension, sympathetic and CNS effects

<b>TRICYCLIC ANTIDEPRESSANT (UPTAKE INHIBITORS)</b>	<p><b>Examples:</b> Imipramine, Clomipramine</p> <p><b>Mechanism:</b> Block re-uptake of noradrenaline. Clomipramine also blocks 5-HT</p> <p><b>Action:</b> Prevents inactivation of noradrenaline</p> <p><b>Side effects:</b> Anti-muscarinic activity, postural hypotension, convulsions, confusion</p>
<b>SELECTIVE UPTAKE BLOCKERS</b>	<p><b>Examples:</b> 1) Maprotiline 2) Fluoxetine</p> <p><b>Mechanism:</b> 1) Noradrenaline uptake blocker 2) SSRI</p> <p><b>Action:</b> 1) Prevent inactivation of noradrenaline 2) Prevent inactivation of 5-HT</p> <p><b>Side effects:</b> Severe serotonin toxicity if given with MAO inhibitor - fatal</p>
<b>SNRIs</b>	<p><b>Examples:</b> Venlafaxine</p> <p><b>Mechanism:</b> Inhibit re-uptake of both 5-HT and noradrenaline</p> <p><b>Action:</b> Prevent inactivation of 5-HT and noradrenaline</p> <p><b>Side effects:</b> Fewer side effects as do not have anti-muscarinic activity</p>
<b>ATYPICAL ANTIDEPRESSANT</b>	<p><b>Examples:</b> Mianserin, Mirtazepine</p> <p><b>Mechanism:</b> <math>\alpha_2</math> antagonists. Prevent feedback inhibition of noradrenaline release. Also bind to 5-HT receptors</p> <p><b>Action:</b> Prevent inactivation of 5-HT and noradrenaline</p> <p><b>Side effects:</b> Hepato and renal toxicity, postural hypotension</p>
<b>OTHER ANTIDEPRESSANT</b>	<p><b>Examples:</b> Trazodone</p> <p><b>Mechanism:</b> 5-HT re-uptake inhibitor. 5-HT<sub>2</sub> receptor antagonist</p> <p><b>Action:</b> Prevents inactivation of 5-HT</p> <p><b>Side effects:</b> Postural hypotension</p>

**ANTI-EPILEPTICS**

<b>Na<sup>+</sup> CHANNEL INHIBITORS</b>	<b>Examples:</b>	Carbamazepine, Phenytoin
	<b>Uses:</b>	Carbamazepine used to treat partial seizures
	<b>Mechanism:</b>	Inhibits Na <sup>+</sup> channels in hyper-excitabile cells
	<b>Action:</b>	Reduces cell excitability and firing
	<b>Side effects:</b>	Sedation, ataxia, changes in oral contraceptive metabolism
<b>Ca<sup>2+</sup> CHANNEL INHIBITOR</b>	<b>Examples:</b>	Ethosuximide
	<b>Uses:</b>	Treatment of absence seizures
	<b>Mechanism:</b>	Inhibit Ca <sup>2+</sup> channels in excitable cells
	<b>Action:</b>	Reduces cell excitability and firing
	<b>Side effects:</b>	Sedation, ataxia, changes in oral contraceptive metabolism
<b>GENERAL ANTI-EPILEPTIC</b>	<b>Examples:</b>	Valproate
	<b>Uses:</b>	Can be used in most forms of epilepsy
	<b>Mechanism:</b>	Inhibits GABA metabolism. Blocks Na <sup>+</sup> channels in hyper-excitabile cells
	<b>Action:</b>	Reduces cell excitability and firing
	<b>Side effects:</b>	Sedation, ataxia, changes in oral contraceptive metabolism
<b>GABA-A RECEPTOR MODIFIERS</b>	<b>Examples:</b>	Clonazepam, Diazepam
	<b>Uses:</b>	Clonazepam used to treat status epilepticus
	<b>Mechanism:</b>	Allosteric modification of GABA-A receptors
	<b>Action:</b>	Reduces cell excitability and firing
	<b>Side effects:</b>	Tolerance likely - used in emergencies. Sedation, ataxia, changes in oral contraceptive metabolism.

**DIABETES & OBESITY**

<b>SULPHONYLUREAS</b>	<b>Examples:</b>	Tolbutamine (Short acting), Glibenclamide (Long acting)
	<b>Uses:</b>	Treatment of type II diabetes mellitus
	<b>Mechanism:</b>	Block ATP-sensitive K <sup>+</sup> channels. $\beta$ -cells depolarise. Ca <sup>2+</sup> channels open; influx
	<b>Action:</b>	Stimulation of insulin release
	<b>Side effects:</b>	Hypoglycaemia, increased appetite - weight gain
<b>BIGUANIDES</b>	<b>Examples:</b>	Metformin
	<b>Uses:</b>	Treatment of type II diabetes mellitus
	<b>Mechanism:</b>	Increase glucose uptake by tissues
	<b>Action:</b>	Suppress appetite. Decrease plasma LDLs
	<b>Side effects:</b>	Lactic acidosis, decreased vitamin B12 absorption
<b>THIAZOLEDIONES</b>	<b>Examples:</b>	Piaglitazone
	<b>Uses:</b>	Treatment of type II diabetes mellitus
	<b>Mechanism:</b>	Activate PPAR $\gamma$ receptors
	<b>Action:</b>	Sensitise peripheral tissues to insulin
	<b>Side effects:</b>	-
<b>OBESITY TREATMENTS</b>	<b>Examples:</b>	1) d-Amphetamine 2) Sibutramine 3) Orlistat 4) Liraglutide
	<b>Uses:</b>	Reduce weight in obese patients
	<b>Mechanism:</b>	1) Competitive inhibition of NA re-uptake. 2) Inhibits NA & 5-HT re-uptake. 3) Inhibits pancreatic lipase. 4) GLP1 agonist.
	<b>Action:</b>	1)& 2) Prevents breakdown & reduces food intake. 3) Prevents fat breakdown - excreted instead. 4) Promotes insulin release.
	<b>Side effects:</b>	1) Dependence. 2) Sympathetic effects. 3) Faecal fat; patients must adhere to low fat diet.

**INFLAMMATION (Also see above)**

<b>STEROIDS</b>	<b>Examples:</b>	Hydrocortisone, prednisolone, betamethosone
	<b>Uses:</b>	Reducing inflammation (& associated oedema and pain), asthma
	<b>Mechanism:</b>	Bind glucocorticoid receptor. Loss of hsp complex. Receptor enters nucleus and binds glucocorticoid regulatory element. Inhibits cyclo-oxygenase.
	<b>Action:</b>	Transcription of anti-inflammatory proteins. Suppression of pro-inflammatory gene transcription. Decreases cyclo-oxygenase products.
	<b>Side effects:</b>	Suppression of hypothalamic/pituitary/adrenal function. Immunosuppression. Salt & water retention.
<b>ANTI-TNF<math>\alpha</math> ANTIBODIES</b>	<b>Examples:</b>	Infliximab, Adalimumab
	<b>Uses:</b>	Reduce inflammation
	<b>Mechanism:</b>	Prevent TNF $\alpha$ from binding to its receptors
	<b>Action:</b>	Prevent inflammation
<b>IMMUNO-SUPPRESSANTS</b>	<b>Examples:</b>	Leflunomide, ciclosporin, methotrexate
	<b>Uses:</b>	Reduce inflammation
	<b>Mechanism:</b>	Suppress immune response
	<b>Action:</b>	Decrease inflammatory mediators
	<b>Side effects:</b>	Decreased immune system
<b>HISTAMINE RECEPTOR ANTAGONISTS (ANTI-HISTAMINES)</b>	<b>Examples:</b>	H1 - Astemizole, terfenadine, ceterizine. H2 - Cimetidine, ranatidine. H3 - Thioperamide
	<b>Uses:</b>	H1 - Treat allergies. H2 - treat GI conditions (E.g. gastric ulcers). H3 - Undergoing trials
	<b>Mechanism:</b>	Inverse agonists at histamine receptors
	<b>Action:</b>	H1 - Smooth muscle relaxation (Except arterial). Reduced secretions. Reduced pain (Decreased stimulation of C-fibres). H2 - Reduced acid secretion
<b>OTHER</b>	<b>Examples:</b>	Auranofin
	<b>Uses:</b>	Anti-rheumatic drug
	<b>Mechanism:</b>	Unknown
	<b>Action:</b>	Reduces IL-1 & TNF $\alpha$

**THROMBOSIS**

<b>ANTI-COAGULANTS</b>	<b>Examples:</b>	1) Heparin, Dalteparin (Low molecular weight heparin - longer duration of action) 2) Warfarin
	<b>Uses:</b>	Reduce thrombus formation
	<b>Mechanism:</b>	1) Inactivates thrombin 2) Interferes with vitamin K reduction
	<b>Action:</b>	Reduce blood coagulation
	<b>Side effects:</b>	Haemorrhage
<b>FIBRINOLYTICS</b>	<b>Examples:</b>	Streptokinase, Urokinase, Tissue Plasminogen Activator (TPA)
	<b>Uses:</b>	Reduce thromboses
	<b>Mechanism:</b>	Activate plasminogen
	<b>Action:</b>	Encourage breakdown of fibrin
	<b>Side effects:</b>	Bleeding
<b>ANTI-FIBRINOLYTICS</b>	<b>Examples:</b>	1) Tranexamic acid, Aminocaproic acid (Less potent) 2) Aprotinin
	<b>Uses:</b>	Reduce bleeding
	<b>Mechanism:</b>	1) Inhibition of plasminogen activation 2) Inhibits proteolytic enzymes
	<b>Action:</b>	Promote blood coagulation
	<b>Side effects:</b>	Thrombosis

**ASTHMA (Also see above)**

<b>ANTI-ALLERGY DRUGS</b>	<b>Examples:</b>	Cromoglycate, Nedocromil
	<b>Uses:</b>	Prevent asthma attacks
	<b>Mechanism:</b>	Unknown. Possibly inhibit release of inflammatory mediators or inhibit bronchoconstrictor reflexes
	<b>Action:</b>	Prevent inflammation or bronchoconstriction

<b>METHYLXANTHINES</b>	<b>Examples:</b>	Theobromine, Theophylline
	<b>Uses:</b>	Asthma
	<b>Mechanism:</b>	Inhibit cAMP & cGMP phosphodiesterases, preventing their catabolism. Competitive antagonism of adenosine.
	<b>Action:</b>	Smooth muscle relaxation, bronchodilation.
	<b>Side effects:</b>	Tachycardia and arrhythmia (+ve inotropic & chronotropic effect), tremor
<b>LEUKOTRIENE RECEPTOR ANTAGONISTS</b>	<b>Examples:</b>	Montelukast
	<b>Uses:</b>	Asthma
	<b>Mechanism:</b>	Antagonise action of LTD4

**FERTILITY**

<b>GnRH</b>	<b>Examples:</b>	Gonadorelin
	<b>Uses:</b>	Pulsatile dosing - stimulate ovulation OR Continuous dosing - treat endometriosis, precocious puberty and sex-hormone-dependent tumours
	<b>Action:</b>	Simulates effects of GnRH. Pulsatile dosing - stimulates LH/FSH production. Continuous dosing - inhibits LH/FSH production
<b>GONADATROPHINS</b>	<b>Examples:</b>	Human chorionic gonadotrophin, menotrophin
	<b>Uses:</b>	Treat female infertility (Due to pituitary insufficiency). Treat cryptorchidism (Undescended testis)
	<b>Action:</b>	Simulates effects of gonadotrophins
<b>OESTROGENS</b>	<b>Examples:</b>	Ethinylestradiol, mestranol
	<b>Uses:</b>	HRT, oral contraceptives, hormone-dependent tumours, menopausal symptoms
	<b>Action:</b>	Simulates effects of oestrogens
<b>OESTROGEN ANTAGONISTS</b>	<b>Examples:</b>	1) Clomiphene 2) Tamoxifen
	<b>Uses:</b>	1) Induce ovulation 2) Breast cancer?
	<b>Action:</b>	Antagonise oestrogen receptors. Tamoxifen acts on peripheral oestrogen receptors

<b>PROGESTOGENS</b>	<b>Examples:</b>	Progesterone (Injection only), Norethisterone (Oral), Danazol
	<b>Uses:</b>	Contraceptives
	<b>Action:</b>	Simulate effects of progesterone. Danazol inhibits gonadotrophin secretion
<b>ANDROGENS</b>	<b>Examples:</b>	Testosterone, Fluoxymesterone, Nandrolone
	<b>Uses:</b>	HRT, treatment of certain tumours
	<b>Action:</b>	Simulate effects of androgens

**ANTI-VIRALS**

<b>INHIBITORS OF ATTACHMENT TO/ PENETRATION OF HOST CELL</b>	<b>Examples:</b>	Amatidine
	<b>Uses:</b>	Influenza A
<b>INHIBITION OF REVERSE TRANSCRIPTASE</b>	<b>Examples:</b>	DDC, AZT (Zidovudine), DDL, 3TC (Lamivudine)
	<b>Uses:</b>	HIV
<b>INHIBITION OF DNA POLYMERASE</b>	<b>Examples:</b>	1) Idoxuridine 2) Acyclovir, Vidaribine 3)Ganciclovir
	<b>Uses:</b>	1) HSV 2) HSV & Varicella-Zoster viruses 3) CMV retinitis
<b>INHIBITION OF PROTEIN TRANSLATION &amp; VIRUS ASSEMBLY</b>	<b>Examples:</b>	Interferons
<b>INHIBITION OF VIRAL RELEASE</b>	<b>Examples:</b>	Oseltamavir, Zanamavir
	<b>Uses:</b>	Influenza A

**ANTIBIOTICS**

<b>PENICILLINS</b>	<b>Examples:</b>	Benzympenicillin, Flucloxacillin (Basically anything ending in -cillin)
	<b>Uses:</b>	Broad spectrum. Gram positive bacteria.
	<b>Mechanism:</b>	Act on peptidoglycan. Inhibit transpeptidase.

<b>β-LACTAMS</b>	<p><b>Examples:</b> Penams, Cephalosporins, Monobactams, Carbapenams (Broad antibiotic class)</p> <p><b>Uses:</b> Broad spectrum. Usually given with a β-lactamase inhibitor such as clavulanic acid</p> <p><b>Mechanism:</b> Act on peptidoglycan. Prevent cross-linking.</p>
<b>TETRACYCLINES</b>	<p><b>Examples:</b> Tetracycline, Doxycycline</p> <p><b>Uses:</b> Broad spectrum</p> <p><b>Mechanism:</b> Inhibit protein synthesis. Prevent binding of tRNA to amino acids.</p>
<b>ANTI-METABOLITES</b>	<p><b>Examples:</b> Trimethoprim, Sulphonamides</p> <p><b>Uses:</b> Broad spectrum</p> <p><b>Mechanism:</b> Prevent DNA replication. Decrease nucleotide generation.</p>
<b>QUINOLONES</b>	<p><b>Examples:</b> Levofloxacin, Trovafloxacin</p> <p><b>Uses:</b> Broad spectrum</p> <p><b>Mechanism:</b> Prevent DNA replication</p>
<b>COMPLEX GLYCOPEPTIDES</b>	<p><b>Examples:</b> Vancomycin</p> <p><b>Uses:</b> Gram positive bacteria</p> <p><b>Mechanism:</b> Acts on peptidoglycan. Prevents incorporation of NAM &amp; NAG peptide subunits</p>
<b>MACROLIDES</b>	<p><b>Examples:</b> Erythromycin</p> <p><b>Uses:</b> Gram positive bacteria</p> <p><b>Mechanism:</b> Inhibit protein synthesis. Prevent translocation along ribosomes</p>
<b>AMINOGLYCOSIDES</b>	<p><b>Examples:</b> Entamicin, Tobramycin, Streptomycin, Neomycin</p> <p><b>Uses:</b> Gram negative bacteria</p> <p><b>Mechanism:</b> Inhibit protein synthesis. Prevent codon-anticodon recognition of tRNA</p>
<b>POLYMYXINS</b>	<p><b>Examples:</b> Polymyxin B</p> <p><b>Uses:</b> Gram negative bacteria</p> <p><b>Mechanism:</b> Act on cell membrane/envelope. Allow free access through ("Puncture")</p>