DISEASE	CATEGORY	DRUG NAME	MOA	ADRs	CONTRAINDICATIONS
HYPERTENSION ❤	**ACEi**	Ramipril	- Inhibits ACE therefore inhibits conversion of AT1 to AT2	Dry cough	Pregnancy
			-Leads to less breakdown of bradykinin causing more vasodilation	Angioedema **(NOTE: more**	Renal stenosis
			-Leads to reduced ADH release	**common among black**	ACEi allergy
			-NO and PGI2 also cause vasodilation	**people so ARBs should be**	Chronic cough
			BP decreases	**used preferentially)**	
			- First line treatment for HTN with type 2 DM (or ARBs) or < 55 yrs	Headaches	
				Nausea/vomiting	
	ARBs	Losartan	- Blocks Angiotensin 2 receptor	Headache	Pregnancy
			-Prevents vasoconstriction and aldosterone activation	Hyperkalaemia	Bilateral renal stenosis
			-Leads to BP decrease	Nausea/vomiting	ARB allergy
			- First line treatment for HTN with type 2 DM (or ACEi) or < 55 yrs		
	Alpha-blockers	Doxazosin	- Inhibits contraction of vascular smooth muscle cells	Dizziness due to postural	
			-Leads to less vasoconstriction causing BP to decrease	hypotension	
			-Also decreases total body cholesterol which is beneficial for patients with	Headache	
			hyperlipidaemia	Fatigue	
			-Used with ARB/ACEi, CCB + Thiazide-like diuretic if blood K+>4.5 mmol/l	Oedoma **(especially when combined**	
				with dihydropyridines)	
	Beta-blockers	Bisoprolol	-Blocks sympathetic function in the heart by blocking β1 adrenoceptors	Can mask tachycardia **(sign of**	Asthma
			-Also decreases myocardial contraction	**insulin-induced hypoglycaemia**	COPD
			-Leads to decrease in HR causing a decrease in CO and BP	**in type 2 DM)**	2nd and 3rd degree heart block
			- Can be selective or non-selective	Bradycardia	
			-Used with ARB/ACEi, CCB + Thiazide-like diuretic if blood K+>4.5 mmol/l	Raynaud's	
				Bronchoconstriction	
				Reduced exercise tolerance	
	CCBs		-Block Ca2+ channels in the SA node and vascular smooth muscle cells		Congestive heart failure
			-Causes a prolonged action potential and refractory period		Heart block
			-Leads to a fall in BP		Ventricular tachycardia
			- First line treatment for HTN in black patients and patients 55 and above		
	1)Dihydropyridines	Amlodipine		Increases plasma conc. of simvastatin	
				Palpitations	
				Sympathetic activation (tachycardia)	
				Oedema	
	2)Benzothiazapines	Diltiazem		Risk of bradycardia	
				Can worsen heart failure (less than	
				verapamil)	
	3) Phenylalkylamines	Verapamil		Risk of bradycardia	
				Constipation	
				Can worsen heart failure due to -ve inotropic effect	
	Thiazide Diuretics	Bendroflumathiazide	-Inhibits Na+ reabsorption at the DCT	Hypokalaemia	
			-Leads to decrease in blood and extracellular volume causing lower TPR	Increased urea and uric acid levels	
			-Leads to decrease in BP	Impaired glucose tolerance with beta blockers	
			-Useful over CCBs in oedema	RAAS activation	
			-typically used with ACEi/ARB and/or CCB	Increased cholesterol and TGL levels	
HEART FAILURE	**ACEi and ARBs**		-Used to decrease BP to prevent worsening of the heart failure		
			-Same MOA as in HTN		
			-ARBs used if ACEi isn't tolerated		
			NOTE: keep initial dose low to prevent rapid fall in BP		
			-Used in HTN when blood K+ is 4.5 mmol/L or less		
	Aldosterone Receptor	Spironolactone	-Blocks aldosterone receptor		
	Antagonist		-Leads to reduced reabsorption of sodium causing BP to decrease		
			-Prevents heart failure from worsening		
			- Typically used with ACEi and a diuretic		
	Diuretics	Furosemide	- Used to treat oedema		
	Adjunct Therapeutics	Digoxin	-Provides symptomatic relief for patients with sinus rhythm		
		Ivabridine	-Funny current blocker		
			-Reduces HR		
			-Used for sinus rhythm with HR > 75 and ejection fraction < 35%		
		Sacubitril	- Inhibits natriuretic inactivating enzyme		
			-Potentiate the effects of ANP leading to more vasodilation		
			-Used as a replacement for ARB/ACEi if ejection fraction <35%		
		Hydralazine	- Balances venodilaton and arteriodilation		
			Reduces preload and afterload causing CO to increase		
			-Used especially in black patients with low RAAS activity		

TYPE OF ANAESTHETIC	ROUTE OF DELIVERY	EXAMPLES	MOA	USES	SIDE EFFECTS
GENERAL/ SYSTEMIC	**INTRAVENOUS (IV)**	PROPOFOL (rapid)	- Targets GABA A receptors (potentiates GABA		
	- induction	BARBITURATES (rapid)	activity)		
		ETOMIDATE	- Increased Cl- conductance		
			- Depressed CNS activity; sedation, anaesthesia		
			and anxiolysis.		- PONV; post operative nausea and
		KETAMINE (slow)	**- Non-competitive antagonists of NMDA receptors.**		vomiting.
	INHALATION	HALOTHANE	- Targets GABA A receptors		- CVS effects; hypotension
	(VOLATILE)	ISOFLURANE	- Increased Cl- conductance		- PO cognitive dysfunction; risk increases
		DESFLURANE	- Depressed CNS activity; sedation, anaesthesia		with age.
			and anxiolysis.		- Chest infection
		N2O	- Mostly added to other volatile agents		- Post operative urinary retention.
			(reduced dosing).		
			- No potentiation of GABA activity		
			- Inhibition of NMDA glutamate receptors; removal		
			of excitatory effect in CNS.		
LOCAL	**IV**	LIDOCAINE	- Higher potency due to high lipid solublity.		
	- NO is used as a carrier	BUPIVACAINE	- Lower pKa means faster onset	- Bupivacaine infiltration for wound analgesia.	
			- Is an amide so longer acting		- Side effects for local or regional are mild
			- Blocks small myelinated afferent nerves; nociceptive		and rare.
			and sympathetic block.		- Local anaesthetics are Na+ channel
		ROPIVACAINE	- Less potent and shorter duration of action compared		blockers so CVS toxicity.
			to Bupivacaine.		- Allergic reactions and anaphylaxis.
			- Esterase metabolised so slower onset time.		
		PROCAINE	**- LOCAL ANAESTHETICS IN GENERAL TARGET**		
			VOLTAGE GATED Na+ CHANNELS.		
REGIONAL	**EXTRADURAL**	- OPIOIDS like Codaine,	- Block of a nerve hence the patient stays awake.		
	INTRATHECAL	Hydrocone and Methadone	**- Regional anaesthesia uses a local anaesthetic or an**		
	COMBINED (pregnancy)		**opioid.**		
		- LOCAL ANAESTHETICS	- For upper extremity; interscalene, supraclavicular,		
		like Lidocaine, Procaine...	infraclavicular and axillary nerves can be blocked.		
			- For lower extremity; femoral, sciatic, popliteal,		
			saphenous nerves can be blocked.		
ANAESTHETIC OF FUTURE*	**INHALATION**	XENON	- Rare gas from air; very expensive to produce.		
			- Low blood and tissue sollubility so rapid induction		
			and recovery.		
			- Potent with minimal side effects.		
			- Nonflammable and not metabolized.		

QUICK REVISION				
TRIAD OF ANAESTHESIA	**- Unconsciousness**			
	- Anaelgesia			
	- Muscle relaxation and			
	loss of reflexes.			
GENERAL ANAESTHETIC	**Guedel's classification**	Stage 1: anaelgesia and concsciousness		
CLASSIFICATION		Stage 2: unconscious, erratic breathing and delirium (avoided step)		
		Stage 3: surgical anaesthesia		
		Stage 4: respiratory paralysis and death		
VOLATILE ANAESTHETIC	**MAC (minimum alveolar**	- Level at which 50% of the subjects fail to move to surgical stimulus.	- MAC is increased by; hyperthermia, pregnancy, alcoholism, central stimulants	
POTENCY	**concentration)**	- At equilibrium; alveolar conc.= spinal cord conc.	- MAC is decreased by; age, sedatives and opioids.	
FACTORS AFFECTING	**PARTITION COEF.**	- Blood gas partition; low value means fast induction and recovery.		
INDUCTION& RECOVERY		- Oil gas partition; potency and slow ccummulation.		
FACTORS AFFECTING	**LIPID SOLUBILITY**	- High lipid solubility means high potency.		
POTENCY	**GABA ACTIVITY**	- High GABA A activity means high potency.		
ANAESTHETIC TARGETS	**MOLECULAR&**	- Balance between glutamate and GABA in the CNS.		
	CELLULAR			
	SYSTEMS	- Effects on brain circuitry.		
		- Depression of reticular formation; connectivity.		
		- Depression of hippocampus; memory.		
		- Depression of brainstem; CVS and respiratory.		
		- Depression of spinal cord dorsal horn; anaelgesic effects.		
		- Thalamus should NOT be depressed; CARDIAC CONTROL.		

DRUG TYPE	CATEGORY	EXAMPLES	MOA	USE	ADRs	DDIs
Anti-emetics	H1 Receptor Antagonists	**-zines:**	- Acts on H1 receptors in the vestibular nuclei	Motion sickness	Antimuscarinic - dry mouth,	
		Levomepromazine (Also a D2 & mAch antagonist)	-Inhibits transmission of histamineergic signals from the vestibular	Promethazine for morning sickness in	constipation, urinary retention	
		Cyclizine	nuclei to the CTZ in the medulla	pregnancy	Sedation	
		Promethazine	-Some also have aniitmuscarinic effects	Not good for little children & old ladies	Long QT interval	
	Muscarinic Receptor Antagonists	Hyoscine	-Competitively inhibits Ach receptors	People who can't take tablets	Sedation	
		Hydrobromide	-Acts on the vestibular nuclei and CTZ	Motion sickness	Memory problems	
				Bowel obstruction	Glaucoma	
					Dry mouth and constipation	
	5-HT3 Receptor Antagonists	**-setrons:**	-Acts on the 5-HT3 receptors in the GI tract and CTZ	Chemotherapy patients	Extra-pyramidal effects- dystonia,	
		Ondansetron	-Inhibits the CTZ	Radiation-induced emesis	parkinsonism	
		Granisetron	-Reduces GI motility and secretions	Post-operative nausea & vomiting (PONV)	Long QT syndrome	
		Palonosetron	-Inhibits vagal afferents leading to decreased peristalsis		Elevated liver enzymes	
					Constipation	
					Headache	
	D2 Receptor Antagonists	Metacloperamide (also a 5HT4 agonist)	- Acts on the CTZ to prevent it sending signals to the medulla		Galactorrhoea	
		Domperidone	-Metacloperamide also acts on gut to promote gastric emptying	Metacloperamide - GORD & Ileus	Extra-pyramidal effects- dystonia,	
		Levomepromazine	and peristalsis	Levomepromazine - Motion sickness and	parkinsonism	
		Haloperidol		vertigo	Long QT syndrome	
				Haloperidol - Chemotherapy and palliation	Hypotension	
				Domperidone also good for promoting	**NOTE: Domperidone doesn't cross**	
				lactation in breastfeeding mothers	**THE BBB therfeore has less CNS**	
					effects	
	Corticosteroids	Methylprednisolone	- Assumed to act on the CTZ	Chemotherapy	Refer to immunosuppressants drug	
		Dexamethasone	-May also have properties of D2 receptor antagonists	PONV	profile	
			-Has to be used with another drug	Palliation		
	Cannabinoids	Nabilone	Assumed to act on the CTZ	Last-line for chemo	Drowsiness	
					Dizziness	
					Dysphoria	
					Dry motuh	
					Visual disturbances	
	NK-1 Receptor Antagonists	**-prepitants:**	-Prevents the action of substance P in the CTZ and	Chemotherapy	Headache	
		Aprepitant	in peripheral nerves		Dirrhoea	
		Fosaprepitant	-Also used for anxiety and depression		Constipation	
			Usually given with 5-HT3 antagonists and dexamethasone		Stevens-Johnson syndrome	
Laxatives	Bulk-forming laxatives	Isphagula Husk	-Absorbs water and swells	First-line for simple short-duration	Bloating	Interferes with Warfarin
		Methylcellulose	-Leads to distention of the GI wall	constipation	Flatulence	absorption
			-Stimulates peristalsis		Blockage therefore needs to be taken	
					with fluids	
	Osmotic laxatives	Lactulose	-Increases the amount of water in the large bowel		Cramping	
		Macrogols	-Causes faecal amtter to be less dry and hard		Bloating	
					Flatulence	
					Higher risk of dehydration in the elderly	
					and patients with renal failure	
	Stool softeners	Liquid paraffin	- Allows water and lipids to penetrate stools more easily making it	Used in patients who have to avoid straining		
		Glycerin	softer and easier to pass	while passing stools e.g post-surgical,		
			-Decreases the surface tension	post-natal or haemorrhoid patients		
	Stimulant laxatives	Co-Danthromer	Directly stimulates the enteric nervous system		Glycerin suppositories cause rectal irritation	
		Senna				
		Glycerin				
Diarrhoea drugs	Opioid Receptor Agonist	Loperamide	-Loperamide acts on μ receptors in the myenteric plexus	-Diarrhoea		
		Codeine	-Codeine & Morphine act on both μ and δ receptors	-Codeine and Morphine usually uased when		
		Morphine	-Decreases the tone of the longitudinal and smooth muscles	the patient also experiences pain		
			Reduces peristalsis but increases segmental contractions			
			-Decreases colonic mass movement by suppressing gastrocolic			
			reflex			
	Dietary		Bananas - high in K+ and fibre			
			White rice - binds stool			
			White bread/pasta - low in fibre			
			Limit fruits to 3 times/day			
			Avoid Caffeine, sorbitol, fatty or spicy foods and fzzy drinks			
			Consider probiotics			

DRUG ACTION	TYPE OF DRUG	EXAMPLE	ACTION	USES	EFFECTS ON ECG	SIDE EFFECTS
SAN AP GENERATION	**BETA AGONISTS**	DOBUTAMINE	- Increased opening probability of If channels - Decreases the time it takes to reach threshold	- Used to treat bradycardia - Tachycardia		
	MUSCARINIC AGONIST	ADENOSINE (given as IV bolus, enters heart and transiently blocks AV node)	- Decreases the opening probability of If channels - Increases the time it takes to reach threshold - Natural nucleoside that binds to a1 and activates K+ currents in SAN and AVN. It slows AV conduction. - Hyperpolarisation; heart rate drops.	- To convert reenterant supraventricular arrhythmias - Diagnosis of coronary artery disease (scans).		
VENTRICULAR AP GENERATION	**CLASS IA** **(moderate Na+ blockade)**	PROCAINAMIDE (IV) **QUINIDINE** DISOPYRAMIDE	- Decreases conduction and automaticity - Increases refractory period and threshold - Increases AP duration	**- Quinidine used to maintain sinus rhythm in AF, flutter and Brugada syndrome** - Procainamide used to treat (IV) acute supraventricular and ventricular arrhythmias	- Increased QRS complex - Moderate effects on PR interval - Increased QT interval	- Hypotension - Torsades de Pointes (increased QT interval) - High dose related dizziness, confusion - GI effects - Lupus like symptoms
	CLASS IB **(weak Na+ blockade)**	**LIDOCAINE (IV)** MEXILETINE (oral)	- Decreased phase 0 conduction in fast beating or ischaemic tissue.	**- Lidocaine used to treat ischaemic ventricular tachycardia; scar related reentry.** - Mexiletine is used to prolong effects of Lidocaine. - Really useful post MI; arrhythmia treatment. - Not used in atrial arrhythmias or AV junctional arrhythmias.	- Class IB does not change phase 0 effectively; but it can in ischaemic tissue. - Increased QRS complex	- Less QT effect than Class IA. - CNS effects; dizziness, drowsiness - Abdominal upset.
	CLASS IC **(strong Na+ blockade)**	**FLECAINIDE** PROPAFENONE	- Decreases phase 0 and automaticity - Increases threshold and refractory period especially in rapidly depolarising atrial tissue. - Flecainide also inhibits opening of K+ channels (not recommended in patients with structural heart disease)	- Can be used in acute phase ischaemia. - Can be used in patients of arrhythmias with abnormal impulse FORMATION. **- Used for supraventricular arrhythmias (fibrillation and flutter)** - Premature ventricular contractions - Wolff Parkinson White syndrome	- Increased PR interval - Increased QRS complex - Increased QT interval	- Proarrhythmia and sudden cardiac death - Increased ventricular response to supraventricular arrhyhtmia - CNS and GI effects. - When using Flecainidine (atrial flutter treatment), combination therapy is needed to block some action of AV to make sure atrial rate is reduced to a level that can be captured by ventricle
	CLASS II **(Beta blockers)**	PROPRANOLOL BISOPROLOL METOPROLOL ESMOLOL	- Increased AP duration and refractory period within the AV node. - Slowed ventricular APs. - Slowed spontaneous depolarisation of the pacemaker potential in the SAN due to their beta blocking action. - Slowed AV conduction velocity.	- Treating sinus and catecholamine dependent tachcardia. - Protection of ventricles from high atrial rates. - Converting reenterant arrhythmias at AV nodes.	- Increased PR interval. - Decreased heart rate.	- Bronchospasms (since it acts on beta adrenoceptors). - Hypotension - Do not use in partial AV block or acute heart failure (used in stable heart failure).
	CLASS III **(K+ blockade)**	AMIODARONE	- Increased refractory period and threshold. - Decreased phase 0 and phase 4 conduction. - Decreased speed of AV conduction.	- Effective for most arrhythmias.	- Increased PR interval - Increased QRS complex - Increased QT - Decreased heart rate	- Pulmonary fibrosis - Hepatic injury - Increased LDL cholesterol - Thyroid disease - Photosensitivity and optic neuritis (transient blindness).
		SOTALOL	- Also demonstrates some class II activity. - It mostly alters AP duration and refractory period in atrial and ventricular tissue. - Increased refractory period. - Slowed phase 4 and AV conduction.	- Widesprectrum; supraventricular and ventricular tachcardia.	- Increased QT interval - Decreased heart rate	- Proarrhythmia - Fatigue - Insomnia
	CLASS IV **(Ca2+ blockade)**	VERAPAMIL DILTIAZEM (oral)	- Slow conduction through AV - Slow down heart rate; effect on L type Ca+2 channels in SAN - Increased refractory period in AV node	- Control the ventricles during supraventricular tachycardia. - Convert supraventicular tachycardia (reentry around AV).	- Increased PR interval - Changes in heart rate depending on baroreflex and blood pressure change	- Caution when partial AV block is present - Caution when hypotension, decreased cardiac output or sick sinus. - Some GI problems; constipation.
ADDITIONAL DRUGS		VERNAKALANT	- Block atrial specific K+ channels; outward channel class 3. - Slows atrial conduction. - Increases potency with higher heart rates.	- Used to convert recent onset atrial fibrillation to normal sinus rhythm.		- Hypotension due to AV block - Sneezing and taste disturbance
		IVABRADINE	- Blocks HCN channels; drive funny currents responsible for pacemaker potential in the SAN. - Slows sinus node without altering blood pressure (like beta blockers and Ca+2 channels do.	- Used to reduce inappropriate sinus tachycardia - Used to reduce heart rate in heart failure and angina (avoiding blood pressure drops).		- Increased risk of atrial fibrillation due to deveopment of circus currents in the atrial wall.
	CARDIAC GLYOSIDE	DIGOXIN	- Inhibits Na+/ K+ ATPase and results in a rise in intracellular Ca+2 concentration. - Increased contractility; positive inotropic effect. - Enhances vagal activity; increased K+ currents and decrease Ca+2 currents. - Slows AV conduction and heart rate; negative chronotropic effect.	- Used to reduce ventricular rate in atrial fibrillation and flutter. - Used to reduce heart rate in combination with BISPROLOL		
	MUSCARINIC RECEPTOR ANTAGONIST	ATROPINE	- Inhibits the effect of excessive vagal stimulation of the heart. - Blocks vagal activity to speed AV conduction and increase heart rate.	- Used to TREAT BRADYCARDIA.		- Metabolised really quickly so used as a short term drug.

Sunny is Cutie	DRUG CLASS		MOA	ADRs/Side effects	CONTRAINDICATION	NOTES
Inhibition of Cell wall synthesis	**Penicillins**	**Bactericidal**	Beta lactam ring - needed for their antimicrobial action	Amoxicillin & Penicillin generally safe for children & pregnancy but		
				may cause renal failure		
	eg. Benzopenicillins		Resistance become problematic due to bacteria producing beta-lactamase enzymes to destroy the ring	**Hypersensitivity** - Utricaria, Fever, Rashes, Anaphylaxis		
	eg. Amoxicillin		Prevent cross linkage between linear peptidoglycan polymer chains that make up cell walls	Encephalopathy with seizures from high doses in severe **renal failure**		
	eg. Ampicillin			Diarrhoea & C.diff - disturbed normal gut flora		
	Cephalosporins		Broad spectrum	**allergic** reactions - cross sensitivity known in 10% of patients		
			commonly used in treatment of Meningitis, Pneumonia,	Skin rashes		
			Septicaemia, Biliary tract infections, Peritonitis, UTI	Nausea & vomiting		
			Similar action to Penicillins	Diarrhoea		
				Hypersensitivity allergic reactions		
	Meropenem	**Bactericidal**	beta lactam similar to penicillin but resistant to beta-lactamase			IV
			used both against Gram +ve & Gram -ve			
			Used against MRSA			
	Vancomycin	**Bactericidal**	inihibit the peptidoglycan formation			IV
			used in Septicemia & Endocarditis by MRSA			
inhibition of Protein synthesis	**Aminoglycosides**	**Bactericidal**	binding to the 30s unit irreversly.	**Autotoxicity** (reduced/loss of hearing)	Myasthenia Gravis = reduce doses if any known renal impairment	no against anaerobes
mammal ribosome = 60&40s	eg. Gentamicin, Tobramycin,		Stops transferring mRNA to protein.	Irreversible **disturbed balance,** deafness, renal toxicity, nausea, vomiting		Narrow therapeutic window & toxicity development is easy
bacterial ribosome = 50 & 30s	Streptomycin,neomicin		Active against Gram -ve & pseudomonas & some Gram +ve			Close monitor
so only works on bacterial ribosome	**Macrolides**	**Bacteriostatic**	Reversly binding to 50s unit of bacterioa ribosome to interfere with bacterial protein synthesis	GI upset, Skin rash, **Prolonged QT interval**		
	eg. Azithromycin		Active against Respiratory infection = eg. Whooping cough, Chalmydia			
			Erythromycin & clarithromycin = P450 enzyme inhibitors & can increase the level of			
	eg. Erythromycin & Clarithromycin		carbamazepine & cyclosporin. so need to keep an eye on the patients			
	Chloramphenicol		Protein synthesis inhibition by competing with mRNA for bacterioal ribosme binding	**Bone marrow toxicity, anaemia**		
			inhibit peptidyl transferase so inhibit additional amino acid chain formation			
	Oxalizidonone (Linezolid)		inhibit protein synthesis by preventing association with mRNA with ribosome	**Neurotoxicity**		
			effective against MRSA, but ineffective against Gram-ve			
	Streptogramins		only used Gram +ve infection			
	Tetracyclines		used for prolonged term infection like Acne, bind to 30s			
inhibition of nucleic acid synthesis	**Sulphonamides & Trimethoprim**		inhibit enzyme di-hydrofolate reducatse in the synthetic pathway to folic acid.	Nausea, Vomiting, Skin rash, Diarrhoea, **folate deficiency**		Increase resistance to Trimethoprim
			Bacteria unable to use external folic acid - which is needed for cell growth			given to UTI, Acute/Chronic bronchitis
			combination of trimethoprim + Sulfamethoxazole ('Co-trimoxazole')			
			used for Stevens-Johnson syndrome, Bone marrow suppression			
	Quinolones	**bactericidal**	inhibit DNA synthesis/replication in bacteria	GI effect, Nausea, Vomiting, Upset stomach, CNS, dizziness, tremor, tendon damage		
	eg. Ciprofloxacin					
	Metronidazole	**bactericidal**	active against anerobic bacteria	Nausea, Vomiting, metalic taste on mouth,		
			commonly used hydropylory & C.diff & dental infection	React with alcohol (and cause tangle) intake so warn patients to not drink. LFT level drop		

Lab cultures & sensitivity testing	
Blood testing	Malaria
Urine sample	all UTI - fresh urine sample is needed; collect middle stream of the urine
Faeces	C.Diff
Throat swab	Bacterial infections
Sputum	Chest infections
	COPD excerbation by steroid
Long term prescribing for antibiotics	eg. 3-6month
Acne	2-3month
Prophylaxis	Recurrent UTIs- elderly gets prophylactic antibiotics in care homes. They could get recurrent UTIs & go to the hospital. So provide antibiotics as preventative medicine
Immunodeficiency	Splenectomy - eg. Patients with splenectomy, organ transplantation = will have preventative antibiotics

TYPE OF DRUG	CLASS OF DRUG	DRUG NAME	MOA	USES	PHARMACOKINETICS	SIDE EFFECTS	ADRs	DISADVANTAGES
ANTIPLATELET AGENTS	METABOLIC INHIBITORS	ASPIRIN	- Non-selective irreversible COX inhibitor.	- Moderate doses have antipyretic and	- Hydrolyzed to salicylate by esterases	- GI ulceration and haemorrhage	- Aspirin resistance; higher than expected	
	COX-1 INHIBITOR		- Irreversible acetylation of serine 530 of COX-1	anaelgesic effects due to PG inhibition.	in the GI mucosa, RBCs, synovial fluid	- Bronchospasms	platelet reactivity despite aspirin treatment	
			- Inhibition of production of thromboxane A2 by	- High doses effective as anti- inflammatory	blood.	- Urticaria	- It can be due to (1) poor compliance (2)	
			platelets.	agents in rheumatic disorders.		- Anaphylactic reactions	COX-1 polymorphism (3) reduced platelet	
			- Moderate doses inhibit both COX-1 and COX-2,	- Management of unstable angina and MI.			recovery time.	
			blocking PG production	- Transient ischaemic attack.				
				- Coronary bypass surgery				
				- Pyrexia				
				- Ischaemic stroke that is NOT associated				
				with AF.				
		DIPYRIDAMOLE	- Phosphodiesterase inhibitor.	- Adjunctive therapy for prophylaxis of		- GI symptoms		- Short lived effect; repeated dosing and slow
			- Prevents inactivation of cAMP.	thromboembolism with cardiac valve replacement		- Dizziness		release preparations are required.
			- Inhibition of thromboxane synthase; reduced	- Used along with aspirin in secondary prevention		- Rash		- Precaution needed in presence of rapidly
			platelet activation.	of stroke and transient ischaemic attack.		- Tachycardia		worsening angina, recent MI, HF, hypotension,
			- Rise in level of prostacyclin so vasodilation and			- Worsening symptoms of coronary		LV outflow obstruction.
			platelet inhibition.			heart disease.		
	P2Y12/ ADP ANTAGONISTS	CLOPIDOGREL (prodrug)	- Blockage of P2Y12 component of ADP receptor					- Ticlodipine no longer used due to its haematological
		PRASUGREL (prodrug)	on platelet surface.					effects
		TICAGRELOR	- Prevent activation of the GPIIb/IIIa receptor					
		CANGRELOR	complex					
			- Reduced platelet aggregation.					
	GLYCOPROTEIN IIb/IIIa	ABCIXIMAB	- Blocks the final common pathway of platelet	- Added to aspirin with or without an oral P2Y12	- Must be given injection or infusion.	- Bleeding		
	INHIBITORS	EPTIFIBATIDE	aggregation; achieves 80% inhibition of	inhibitor for acute coronary syndrome (ACS).		- Thrombocytopenia		
	(also called integrin inhibitors)	TIROFIBAN	platelet aggregation.					
	ATP ANALOGUE	CANGRELOR	- Noncompetitive, reversible P2Y12 receptor					
ANTICOAGULANT AGENTS	VITAMIN K ANTAGONISTS	WARFARIN	- Inhibition of vitamin K epoxide reductase and		- Has to be taken at the same time ever	- Has drug-food interactions; acute ethanol		- The therapeutic range is narrow.
		ACENOCOUMAROL	vitamin K reductase		- Avoid drastic changes in diet.	decrases metabolism of oral anticoagulants		- Dosing is affected by may factors such as genetic
		FLUINDIONE	- Accummulation of vitamin K epoxide in the liver		- Consistent intake of vit K is essential.	and increases PT/INR.		variation, drug interactions and diet.
		PHENPROCOUMON	and plasma and depletion of reduced vit K.			- Chronic ethanol use increases metabolism		- AF patients have high risk of thromboembolic and
			- Vitamin K is required for synthesis of coagulation			and decreases PT/INR.		bleeding complications.
			factors II, VII, IX and X as well as protein C and S.			- Anticoagulant effects of warfarin is decreased		- Frequent monitoring required; cost and burdens.
						if taken with vit K rich food; banana, green leafy		
						vegetable, fish, liver, meat and eggs.		
						- Vitamin E increases warfarin effect.		
						- Cranberry juice increases warfarin effect.		
	DIRECT ACTING ORAL							
	ANTICOAGULANTS							
	DIRECT ACTING Xa	RIVAROXABAN	- Reversal agent is Andexanet alfa					
	INHIBITORS	APIXABAN						
		EDOXABAN						
		BETRIXABAN						
	DIRECT THROMBIN	DABIGATRAN	- Selective direct competitive thrombin inhibitors, both		- Monitoring by antifactor Xa			
	INHIBITORS	BIVALIRUDIN	circulating and thrombus bound IIa active metabolites					
		ARGATROBAN						
	PARENTERAL	HEPARIN	- Potentiates action on antithrombin III and inactivates	- Rapid onset and offset of action so allows for more		- Heparin induced thrombocytopenia.		- Short half life; administration via continuous infusion
	ANTICOAGULANTS		thrombin and other coagulation factors.	flexible dose titration or discontinuation.		- Skin reactions.		for therapeutic levels of anticoagulation.
			- Prevents conversion of fibrinogen to fibrin.	- Ability to monitor using aPTT, anti factor Xa activity		- Osteoporosis in long term use.		- Non-linear kinetics; highly variable dose response
			- BOTH UF AND LMW HEPARIN inhibits (1) thrombin	or ACT.		- Increased risk of haemorrhagic complications		relationship.
			and (2) factor Xa.	- Activity can be reversed by protamine sulfate.				
	Low MW heparin	ENOXAPARIN	- Inhibits Factor Xa more than thrombin by potentiating		- More predictable anticoagulant effect.	- Slightly delayed onset of action.		
	- inhibits factor Xa more than	DALTEPARIN	antithrombin.		- Greater bioavailability than UF hepari	- Longer duration of action so hard to stop therapy		
	thrombin.	TINZAPARIN			- Better correlation between dose and	- Prolonged half life in patients with renal failure.		
		NADROPARIN			anticoagulant response.			
					- Subcutaneous administration.			
	INDIRECT Xa INHIBITOR	FONDAPARINUX	- Selective inhibition of factor Xa.			- Indirectly inhibits Factor Xa through its interaction		
						with antithrombin.		
						- Bioavailability is 100%		
						- Contraindicated in patients with severe renal		
						impairment.		
						- The effect of Fondaparinux persists for 2-4 days		
						after being discontinued.		
FIBRINOLYTICS	TISSUE PLASMINOGEN	ALTEPLASE			- Alteplase administered as IV bolus foll	- Bleeding		
	ACTIVATOR	RETEPLASE			by an infusion; has shortest duration of	- Intracranial haemorrhage		
		TENECTEPLASE			- Tenecteplase as single IV bolus	- Anaemia		
	FIBRINOLYTIC PROTEINS	STREPTOKINASE	- Not clot specific and causes generalised lytic stage.			- Cerebral haemorrhage		
		UROKINASE				- Allergic reactions		

Antiviral drugs		USES (most likely)		
immunoglobulins		- Cold/ runny nose		
Amantadine		- Flu		
Neuraminidase inhibitors		- Sore throat		
DNA polymerase inhibitors		- Bronchitis/ chest infections		
Viral reverse transcriptase inhibitors		- Otitis media with effusion		
Protease inhibitors				
Integrase inhibitors				
Immunomodulators				
Types	**MOA**	**Side effects**	**Which viral**	**NOTES**
Enfuvirtide & Immunoglobulins	Stop the virus **cell penetrationin/fusion the host cell** which needs to survive as they are intracellular parasite			
Amantadine	**Inhibiting uncoating** of Influenza A virus	Insomnia, Dizziness, Headache	Influenza A	
Aiclovir	Nucleoside analogue = Interfering viral **nucleic acid synthesis,** Herpes **DNA synthesis (DNA polymerase) terminator**	vomiting, abdo pain, epistaxis	Herpes, Shingles	oral aciclovir should be given 3-5times/day. **Compliance!!**
	no known benefits of using orally/topically post 72hours of initiation of the symptoms			High dose for Shingles (800mg/day)
	low toxicity			
Zanamivir	inhibiting **exit of viral particles.**			
	has low bioavailability			
Oseltamivir (aka, Tamiflu)	**Neuramidase inhibitor. (inhibit exit)**		Influenza A & B	Reduce duration of illness by lowering viral load by inhibiting replication
	blocks the **exit of the influenza** virus from the host cell -> prevent replication in other than a few host cells			
	pro-drug. 80% of bioavailability. Mean onset of treatment 64hours.			

Type of anti cancer drug	Class of drug	Name of drug	Mechanism of Action	Side effects
Cell cycle non-specific	**Alkylating agents**	Cyclophosphamide	Form adducts with DNA that prevents the cell from reproducing.	May cause leukaemia- this is dose dependent
		Ifosfamide		Arrythmias(ifofasmide), myocardial necrosis causing dilated cardiomyopathy (cyclophosphamide)
			Same as above and it works in nucleus and mitochondria. In nucleus it inhibits DNA rep./mRNA transcription.	
	Platinum agents	Carboplatin	In mitochondria, it iinhibits mDNA rep./transcription and	Supraventricular tachycardia, bradycardia and ST-T wave changes
		Cisplatin	thus alters the mitochondrial function and decreases the energy produced which activates apoptosis.	
Cell cycle specific				
G2 phase	**Topoisomerase inhibitors**	Tropotecan (topoiseomerase I inhibitor)	Topoisomerase is an enzyme that helps with the seperation od DNA strands.	
		Irinotecan (topoiseomerase II inhibitor)		
	Anti-tumor antibiotics	Bleomycin	Creates DNA stand breaks.	Cause dose related cardiomyocye injury and death thus leading to left ventricular dysfiunction.
		Dactinomycin	- Interfere with enzymes involved in copying DNA during the cell cycle.	Mechanism of action includes inhibition of topoisomerase 2 beta resulting in activation of cell death pathways and inhibition of mitochodrial biogenesis.
		Doxorubicin	- Bind to DNA therefore cannot replicate. - Blocks topoimerase function	
S phase	**Antimetabolites**	Methotrexate	Inhibits dihydrofolate reduction, blocks thymidylate and purine synthesis.	
		Gemcitabine	Inhibits DNA synthesis	
		5-Florouracil	Inhibits thymidylate synthesis	
			They all work by acting as a subtitube for normal bulding blocks of RNA and DNA, therefore replication cannot occur.	**Cytarabine- can cause pericardial effusion and cardiac temponade.**
G1 phase	**Hormonal agents**	Tamoxifen	Binds to estrogen receptos and blocks the proliferative action of estrogen on the tissue.	
		Megestrol acetate	supression of LH by inhibiting pituitary funcion.	
M phase	**Taxanes**	Paclitaxel	Inhibit function of microtubles.	
		Docetaxel		
	Vinca alkaloids	Vinblastine		
		Vincristine		
				Over-rall side effects include:
				- hari loss- chemo can damage hair follicles, causing hair to weaken, become brittle and fall out. H air almost always regrows after chemo. Main psychological complication.
				- nausea and vomiting: different types acute onset, delayed onsent and anticipatory.
				- myelosuppression
				- cardiotoxicity
				- pulmonary toxicity
				- anaemia

DISEASE	CATEGORY	DRUG NAME	MOA	ADVANTAGES	SIDE EFFECTS	LIMITATIONS
TYPE II DIABETES	**Insulin sensitiser e.g biguanide**	Metformin	- Aims to improve sensitivity to insulin - Increases anaerobic glucose metabolism in small intestine - Decreased gluconeogenesis and glycogenolysis in liver - Increased glucose uptake and glycogenesis in muscle	- No weight gain -No hypoglycaemia - anti hyperinsulinaemic effect (used in PCOS)	- GI intolerance; bloating, diarrhoea, abdominal discomfort - Vit B12 deficiency - Renal contraindications if GFR <30 ml/min - Rare risk of lactic acidosis	
	Sulfonylureas	Glimepiride Gliclazide **Glipizide** Glibenclamide	- Stimulates insulin secretion; effective when sufficient number of beta cells are present. - Act to close ATP dependent K+ channels to induce depolarisation; depolarisation opens Ca+2 channels. - Opening of Ca+2 channels will induce exocytosis of insulin containing vesicles out of beta cells.	- Rapid correction of hyperglycaemia - Prandial glucose control (glucose control while eating).	- Weight gain - Risk of hypoglycaemia	- Drugs only effective in the beginning of diagnosis when there are still sufficient levels of beta cells.
	PRARγ agonist & Insulin sensitiser	Thiazolidinediones	- Increases glucose uptake by muscle cells. - Decreases gluconeogenesis in liver. - Increases adipogenesis, lipogenesis, fatty acid uptake, glucose uptake and adiponectin in adipose tissue. - Decreases TNF a in adipose tissue.	- No hypoglycaemia - No inflammation - No fatty liver	- Weight gain - Risk of heart failure - Fluid retention and oedema - Risk of bone fractures - Bladder cancer	
	Incretin based therapies					
	1) DPP-4 inhibitors	**Sitagliptin** Vildagliptin Saxagliptin Linagliptin Alogliptin	- Enhances incretin effect - Increases glucose induced insulin secretion - Decreases glucagon secretion(?)	- Weight neutral - No hypoglycaemia	- Acute pancreatitis - Pancreatic cancer - Affects renal function; corrected by dose adjustments (except Linagliptin).	- DPP-4 inhibitors only work to increase half life of GLP-1 hormones that are present but the levels are downregulated in type II diabetes so not fully effective.
	2) GLP-1 receptor agonists	Exenatide **Liraglutide** Lixisenatide Dulaglutide Semaglutide	- Enhance incretin effect - Increases glucose induced insulin secretion - Decreases glucagon secretion - Satiety effect - Delayed gastric emptying	- Weight loss - No hypoglycaemia - Emerging CV benefits - Decreases blood pressure - Potential neural benefits	- GI effects; nausea, vomiting, diarrhoea - Risk of thyroid C-cell tumours - Acute pancreatitis	- GLP-1 agonists and DPP-4 inhibitors are contraindicated if given together.
	Alpha glucosidase inhibitors	Acarbose	- Slows down rate of carbohydrate digestion. - Binds to alpha glucosidase enzyme with greater affinity than the natural substrate (sucrose, maltose, maltotriose and dextrins.) - Alpha glucosidase is needed to digest carbohydrates so once slowed down rise in plasma glucose levels is slowed down too.	- No weight gain - No hypoglycaemia - May decrease triglycerides	- GI disease - Flatulence	
	SGLT-2 inhibitors	**Canagliflozin** Dapagliflozin Empagliflozin Ertugliflozin	- Inhibits renal glucose reabsorption by inhibiting SGLT-2 in proximal convoluted tubule. - Increases glucosuria	- Weight loss - No hypoglycaemia - Reduced blood pressure (osmotic diuresis) - CV benefits - Potential renal benefits	- Hypotension and volume depletion due to osmotic diuresis - Genitourinary infections - DKA - Risk of bone fractures or amputations	
TYPE I DIABETES	**Insulin**		-Replaces insulin -Leads to reduction in hepatic glucose -Increases peripheral glucose utilisation		- Injection site reactions -Weight gain -High risk of hypoglycaemia	

CATEGORY	EXAMPLE	MOA	USE	ADRs	DDIs
Osmotic Diuretics	Mannitol	-Act on the PCT and loop of Henle to increase the solute conc. within the tubules	Reduce high intracerebral pressure	Allergies	
		- This leads to more movement of water into the tubles and limits reabsorption of solutes			
		-Leads to decrease in plasma volume causing BP to decrease			
Loop Diuretics	Furosemide	-Acts on the Na+/K+/Cl- symporter in the loop of henle	Oedema (+/- HTN in advanced CKD)	Alkalosis	Aminoglycosides - Ototoxicity and nephrotoxicity
		-Inhibits Cl- reabsorption		Increased urate (gout)	Digoxin - Hypokalaemia due to ↑ digoxin binding & toxicity
		-This prevents reabsorption of Na+ and K+		Increased lipids	
		-Causes more water to be excreted therefore plasma volume decreases		Ototoxicity	Steroids - Increased risk of hypokalaemia
		-Causes BP to decrease		Hypokalaemia	Lithium - Reduced lithium levels
Thiazides	Bendroflumethiazide	- Acts on the Na+-Cl- transporter in the DCT to inhibit Na+ reabsorption	Hypertension	Gout due to ↑ urate	Digoxin - Hypokalaemia due to ↑ digoxin binding & toxicity
		-Promotes Ca2+ reabsorption		Hyperglycaemia	
				Hypercalcaemia	β-blockers - Hyperglycaemia, hyperlipidaemia, hyperuricaemia
				↑ LDL and TG	
Thiazide-like	Indapamide			Erectile dysfunction	Steroids - Increased risk of hypokalaemia
				Hypokalaemia	Carbamazepine - Increased risk of hyponatraemia
					Lithium toxicity
Potassium-sparing Diuretics	Amiloride	-Act on the principal cells of the late DCT and collecting duct	Patients with low potassium where a diuretic is required	Hyperkalaemia	ACEi - Increased hyperkalaemia
		-Inhibits the expression of ENaC therefore inhibiting Na+ reabsorption			
		-Also prevents K+ secretion into the tubule therefore less is excreted			
		-Usually used with other diuretics to prevent K+ excretion			
Carbonic Anhydrase Inhibitors	Acetazolamide	-Act at the PCT to inhibit reabsorption of sodium bicarbonate	Glaucoma	Metabolic acidosis	
		-Leads to the excretion of Na+, K+ and PO3	Altitude sickness	Hypokalaemia	
		-Weakest diuretic		Renal stenosis	
Aldosterone Antagonists	Spironolactone	-Acts on the intercalated cells of the DCT and collecting duct	Heart failure	Hyperkalaemia	
		-Inhibits the expression of ENaC and Na+/K+-ATPase transporter	Ascites	Gynaecomastia	
		Prevents K+ secretion and K+ reabsorption = K+ - sparing diuretic	Hypertension		
			Hyperadrenalism		
ADH Antagonists	Lithium	-Acts on the DCT and collecting ducts to inhibit water reabsorption	Hyponatraemia	Hypernatraemia	
				Deranged LFTs	

CLASSIFICATION	DRUG NAME(S)	MOA	USE	PREGNANCY	ADRs
Drugs Acting at Glutamatergic Synapse					
1) Na+ channel blockers	Valproate	-Blocks Na+ from binding	First line for generalised seizures	Unsafe	P450 enzyme inhibitor
		-Also increases GABA activity	Second line for focal seizures		Increased appetite and weight gain
		- Can not be used below the age of puberty			Hepatitis
					Ataxia
	Carbamezipin	-Binds to Na+ channels increasing their refractory	First line for focal seizures	Unsafe	P450 enzyme inducer
		period	**NOTE: can exacerbate absence and**		Visual disturbances especially diplopia
			myoclonic seizures		Dizziness and ataxia
					Syndrome of Inappropriate ADH secretion
	Phenytoin	-Binds to Na+ channels increasing their refractory	Second line for focal seizures	Unsafe	P450 enzyme inducer
		period			Dizziness and ataxia
					Osteomalacia
					Hirsutism
					Gingival hyperplasia
2) Ca2+ channel blockers	Lamotrigine	-Binds to Na+and Ca2+ channels to prevent	Second line for generalised tonic-clonic	Unsafe	Stevens-Johnson Syndrome
		depolarisation	seizures	(but safest)	
Drugs Acting at GABA Synapses	Benzodiazepine e.g	-Binds to a separate site on GABAa receptor to	First line for status epilepticus		
	Lorazepam and	increase the movement of Cl- into the post-synaptic			
	Diazepam	neuron			
	Vigabatrin	-GABA Transaminase inhibitor	Third line for focal seizures		Risk of severe, symptomatic , persistent visual field
		-Increases the levels of GABA in the synaptic cleft	**NOTE: can exacerbate absence and**		constriction
			myoclonic seizures		
	Cannabinoidiol	-May be effective in preventing refractory status			
		epilepticus			

NOTE ON PREGNANCY:
-All AEDs carry teratogenic risk
-Women and girls MUST be treated with 5mg folic acid before any possibility of pregnancy
-Adjust combination pill dosage when prescribing P450 inducers
-Progesterone-based contraception is not recommended with P450 inducers
-Oestrogen-based contraception reduces lamotrigine levels and may cause seizures

CATEGORY	EXAMPLE	MOA	USE	ADRs	DDIs
H2 Receptor Antagonist	Ranitidine	Blocks the action of histamine at H2 recptor on parietal cells.	Heals peptic ulcers.	diarrohea	
	Cimetidine	It is a reversible, competitive antagonist.		dizziness	Inhibit some CYP450 enzyme and can affect drugs
				rashes (self limitng)	such as warfrin and profolin
	Tamotidine			hepatitis & pancreatitis	
Proton Pump Inhibitors	Omeprazole	Irreversible inhibition of th H+/K+ ATPase.	Control acid secretion	Inc. risk of GI infection	Affects CYP219 which turn propragil to active form.
	Lansoprazole	The produrg is actuvated in low pH and when it enters acidic environment, it gets		eg C.difficile	Decreases efficacy of lansipropril- omeprazole
	Esomeprazole	pronated and cannot leave. This causes accumulation in canniculus and triggers		Inc. risk of fractures	
		the actvation of the drug. When actuvated irreversible binds to exposed cystine		hypomagnesia&natreamia	
		residues of the proton pump. Prevents furter relase of H ions to make HCL acid.		Intestinal nephritis	
Antacids	Magnesium based	Insolube in water but reacts with HCL acid, makes MgCl and H2O. The salt is not		less risk of alkalosis	*Aluminium calcium and magnesium antacids can
		readily absorbed intestine			collate with some drusg eg. tetracycline and
	Aluminum based	Not well absorbed from inestine		Constipation, abdo.	vilothroxine to produce an insoluble complex.
				cramps and dirrhorea	
	Sodium bicarbonate	Water soluble with acd. Absorbed by intestine.		Systemic alklosis	
				*not rec. low NA diet	
		Bases that neutraise gastric acid in the stomach.			
Alginates		Mucus viscosity! It helps to protect the stomach from acidic environment.	Acid reflux		
		It is a gel that flotas on stomach and stops acid reflux.			
		*combine with antacids			
Misoprostol		Stable analogue of PGE1 which have protective GI protective function.		Miscarriage as it	
		Increases protective mucus secretion and decreses gastric acid secretion.		causes uterine	
				contractions.	
Sucralfate		In acid it polymerises to form a sticky gel that stongly coats stomach ulcers.	Peptic ulcers		*use on empty stomach as it can react
		Protective physical barrier on the surfacre of ulcer.			with dietary proteins
Bismuth salts		Collates to form a protective layer by having a high affinity for the exposed mucosa	Peptic ulcers		*use on empty stomach as it can react
		glycoprotein and the necrotic tissue found in ulcers.			with dietary proteins

Disease	Investigation/ Confirmation	Treatment	
Dyspepsia	Uninvestigated dydpepsia		
	Test H.Pylori	If thats the cause give full does PPI for 4 weeks	
	Reoccurence	Low does PPI possible to relive sympoms	Consider H2 antagonist
	Further invest. if needed.		
	Functional dyspepsia		
	Ensure H.pylori is eradicated	If they had a positive test.	
	Persistent symptoms	Low dose PPI 4 weeks	H2 antagonist 4 weks
	Reoccurence	Low does PPI possible to relive sympoms	
H.Pylori	Tipple therapy for 1 week	2 antibiotics: amoxicilin, metronidazole and clarithromycin	
		1 PPI: 2x a day	Continue after for ulcer healing, for 4 wks
NSAID Induced Ulcer		Stop NSAID	
		Full does PPI or H2 antagonist for 8 weeks.	
	If H.Pylori is poitive	Give eradication treatment after ulcer healing	
	If they have to go back pon NSAID	Low does for small period COX-2 selective NSAID and PPI	
GORD	Endosopy confirmed	PPI or H2 antagonist (if not tolerated)	High dose then step down approach 4-8 wks
Oseophagitis		Needs to heal therefore PPI	
Barret's Oesophagus		PPI high dose and cancer surveillance	

DISEASE	CATEGORY	DRUG NAME	MOA	ADRs	DDIs
Hyperlipidaemia	**DRUGS**	**Statins** e.g Atorvastatin	-Competitive inhibtion of HMG-CoA reductase	-myalgia	-CYP3A4 metabolises statins causing
			- Leads to upregulation of LDL receptors by the liver causing increased	-rhabdomyolysis	a reduced effect of the drug
				-GI disruption	-Grapefruit increases the blood levels
					of statins causing an increased risk of
					1
				-pruritius	
		Fibrates e.g Fenofibrate	-PPARα agonists	-myositis	- can increase the effects of warfarin
			- Increase the production of lipoprotein lipase	-cholelithiaisis	causing the patient to bleed more easily
			-Leads to increased catabolism of VLDLs and increased clearance of	-GI upset	
			triglycerides from plasma lipoproteins	-abnormal LFTs	
			- inhibits hormone-sensitive lipase in adipose tissues	-flushing (give with low	
		Nicotinic acid (Niacin)	-Inhibits the formation of VLDL therefore, decreasing LDLs	dose aspirin to prevent it)	
				- hepatotoxicity	
				- headaches	
				- itching	
				- GI disturbance	
		Omega 3 ethyl esters	- used in combination with statins for type IIb/III hypertriglyceridaemia	- GI discomfort	- may increase prothrombin time when
			- used as monotherapy for type IV hypertriglyceridaemia		used with anticoagulants
		Ezetemibe	- Acts on the brush border of the small intestinal mucosa to inhibit NPC1L1	-headache	
			- Leads to reduced absorption of cholesterol by the gut	-abdominal pain	
				-diarrhoea	
		PCSK9 inhibitors e.g	- monoclonal antibody against PCSK9		
		Alirocumab	- inhibits the degradation of LDL receptors		
		Inclisiran	- siRNA that blocks the synthesis of PCSK9		
			-inhibits the degradation of LDL receptors		
	DIETARY/LIFESTYLE	**Red rice yeast**	-contains monacolin-K		
			- similar to lovastatin		
		Plant sterols	- lower LDL cholesrerol		
			-work with statins but not ezetemibe		
		Fish oils	- similar to omega 3 ethyl esters		
		Alcohol	-increase HDLs	- also increase TGLs and	
				blood pressure	
		Endurance exercise	- increases HDLs		

DISEASE	CATEGORY	DRUG NAME	MOA	DOSING	ADRs	DDIs	CLINICAL MONITORING	PREGNANCY
Rheumatoid Arthritis	**Synthetic DMARDs**	Methotrexate	-Blocks AICAR which leads to blockage of adenosine deaminase	once a week	Pneumonitis	Trimethoprim - increased risk of marrow aplasia	- initial FBC, Renal and LFTs before starting therapy	Unsafe
			-Leads to inhibition of adenosine metabolism	with folic acid	Teratogenic	Co-trimaxazole - increased risk of marrow aplasia	- check every 2 weeks until dose is stable	
			In cancer:		Increased risk of infections	High-dose aspirin - increased risk of Mtx toxicity	- then check every 2-3 months	
			-Acts as an anti-folate by inhibting DHFR. Leads to inhibition of DNA and RNA synthesis		Hepatotoxicity			
		Sulfasalazine	- Sulfapyridine component acts to supress IL-1 and TNF		Insomnia		- FBC, renal and LFTs	Safe with 5mg Folic acid
			- 5-ASA component acts in gut to treat IBD		Anaemia		- Monthly blood tests initially for 6 months then reduce	
					Oligospermia (reversible)			
					Hepatotoxicity			
					Pancreatitis			
		Azathioprine	- Attaches to 6-MP to inhibit purine synthesis		Myelosuppression	Allopurinol - increases the risk of toxicity	-TPMT test	Safe as long as dose is <2mg/kg
					Hepatotoxicity			
		Cyclophosphamide	- binds to DNA and cross-links its strands with RNA		Leucopoenia		- watch renal function, weight and blood counts	Unsafe
					Increased risk of malignancy			
					Haemorrhagic cystitis			
		Mycophenolate	- Depletes guanosine nucleotides in B and T cells		Risk of PML			Unsafe
					Hepatic disorders			
		Ciclosporin and Tacrolimus	- Calcineurin inhibitor (prevents T cell activation)		Acute hepatitis	CYP450 induers reduce the effect of ciclosporin		Safe but should be avoided
					Hyperlipidaemia			
					Cytopaenia	CYP450 inhibitors increase the toxicity		
	Biologic DMARDs	Anti TNF inhibitors	- monoclonal antibody against TNF		Risk of TB reactivation			Safe
	NOTE: two biologic	(e.g infliximab)			Risk of Hep B and C reactivation			
	DMARDs should never				Increased risk of infections			
	be used together				Increased risk of malignancy			
	Corticosteroids	Prednisolone	- Bind to glucocorticoid receptor then the compex enters the nucleus		**Glucocorticoids:**			Safe
			-Complex binds to GRE and inhibits synthesis of inflammatory mediators		Diabetes, osteoporosis, myopathy			
			- Also inhibits COX-2 enzyme		**Mineralocorticoids:**			
					Hypertension, fluid retention			
					Note: risk of adrenal crisis if it is			
					stopped quickly			

DISEASE	CATEGORY	EXAMPLES	MOA	ADRs	DDIs
Parkinson's	Anti-muscarinics	Benztropine	-Competitively inhibits muscarinic receptors	Memory problems	
		Biperiden	-Leads to inhibition of involuntary muscle movements	Drowsiness	
				Constipation	
				Blurred vision	
				Tachycardia	
	Levodopa		-Crosses the BBB to enter the brain where it is converted to dopamine	Dyskinesia	
			-Replaces dopamine lost in the substantia nigra	Wearing off effects	
			-Restores functional movement	Progressive on-off periods	
			-First-line treatment	Schizophrenia	
			-Usually combined with other drugs	Nausea and vomiting	
				Anorexia	
				NOTE: L-DOPA on its own is	
				responsible for most of its side	
				effects therefore it is usually	
				combined with other drugs	
	MOA-B Inhibitors	Selegiline	-Inhibits MOA-B enzyme	Dizziness	
		Rasagiline	-Prevents the oxidation of Dopamine in the brain	Headache	
			-Can be used alone or combined with L-DOPA	GI distress	
			-Decreases motor fluctuations when used with L-DOPA	Sedation	
	Dopamine Agonists	Bromocriptine	-Stimulate the Dopamine receptors in the brain to produce more	Withdrawal	
		Pramiprexole	dopamine	Psychiatric disorders	
			-Rescue treatment for sudden on/off periods	Hallucinations	
				Confusion	
	COMT Inhibitors	Entacapone	-Inhibits metabolism of L-DOPA	Diarrhoea	
			-Allows more dopamine to enter the brain	Discoloured urine	
Myasthenia Gravis	Acetylcholinesterase	Pyridostigmine	- Inhibits acetylcholinesterase enzyme from breaking down Ach at the	Abdominal cramps	
	Inhibitors	Neostigmine	NMJ	Excessive tearing	
			-More Ach is available for muscle contraction	Hypersalivation	
	Immunosuppressants	Corticosteroids	(See 'immunosuppresants' drug profile)		
		Azathioprine			
			-Act to destroy and neutralise the autoantibodies in the bloodstream		
	Immunoglobulins	IVIg	and blocks the production of new autoantibodies		

NSAIDs	DRUG CLASS	DRUG	USES	MOA	PHARMACOKINETICS	ADRs	DDIs	RISK FACTORS	INDICATIONS/ USES
									- Topical use and paracetamol in OA.
- Anaelgesic **- Anti-inflamatory** **- Anti-pyretic**	**NON-SELECTIVE** **- Inhibition of both COX-1 and COX-2**	ASPIRIN IBUPROFEN NAPROXEN DICLOFENAC	- Moderate dose is used as an NSAID (but primarily as antiplatelet). - Low dose is an irreversible COX inhibitor. - Anti inflammatory at high doses.	- Aspirin IRREVERSIBLY inactivates COX whereas other NSAIDs are reversible inhibitors. - Inhibition of COX; decreased prostaglandin, prostacyclin and thromboxane synthesis. - Compete with arachidonic acid for binding site on COX.	- Most NSAIDs are weak acids so fully absorbed along GI tract. - Typically do not undergo first pass elimination. - Highly protein bound. - Hepatic metabolism to inactive products - First order metabolism in moderate doses while zero order in high doses.	COX-1 inhibition means loss of homeostasis; acid imbalance means gastric mucosa damage, renal blood flow regulation lost so kidney injury, nephritis etc. **GI ADRs** - Dyspepsia - Nausea - Peptic ulcers - Bleeding and perforation - Enhanced cytotoxicity and hypoxia due to decreased blood flow - Decreased hydrophobicity of mucus layer due to acidic nature of NSAIDs. - IBD exacerbation. - Rectal irritation and bleeding. **Renal ADRs;** - CKD - Congestive heart failure - Cirrhosis - Hypertension - Oedema - Hyperkalaemia (decreased renin secretion)	- Another NSAID and low dose aspirin use together; decreased CV protection. - Use with Sulfonylurea; hypoglycaemia - Use with Methotrexate; accummulation and hepatotoxicity, leukopenia RA - Warfarin; increased risk of bleeding. - Diuretics - ACEi and ARBs.	- Age - Prolonged use Glucocorticoid steroids - Anticoagulants - Smoking - Alcohol - H. pylori infection - History of peptic ulcers.	- Inflammatory conditions; OA and RA - Post-operative pain - Topical use on cornea - Menorrhagea - (Low dose aspirin) Platelet aggregation inhibition. - To close patent ductus arteriosus - Cancer reduction.
	SELECTIVE **- Inhibition of COX-2 only**	CELECOXIB ETORICOXIB PARECOXIB	- Monitoring can be useful in severe OA and RA.	- Some evidence of less analgesic effect. - Same mode of action as non-selective.		- Less inhibitory action on COX-1 so reduction in ADRs. - Less GI adverse effects but RENAL ADRs SIMILAR to non-selective patients. **CVS ADRs** - Exacerbation of HF - Increased BP due to increased salt and water retention - Prostaglandins act as ADH antagonists; vasoconstriction through reduced antagonism **- Efficacy of antihypertensives is reduced with NSAIDs.**		**- Patients with prothrombotic risk, coronary or cerebrovascular disease should not be prescribed NSAIDs.**	
	NON-NSAID, NON-OPIATE ANALGESIC with antipyretic action	PARACETAMOL		- MOA not clear; COX-2 selective inhibition in CNS (spinal cord); decreased pain signal along the spinothalamic tract. - Little anti-inflammatory action.	- Well absorbed from GI tract.	- Well tolerated at therapeutic doses; fever common ADRs and does not inhibit homeostatic prostaglandin action.			
	NAPQI				- Conjugation with glutathione makes it harmless. - Highly nucleophilic; necrosis and apoptosis. - 150 mg/ kg sufficient to cause severe irreversible hepatocellular and renal tubular damage.				

NSAID ACTION	ANALGESIC	- Decreased PGE-2 synthesis in dorsal horn - Decreased neurotransmitter release - Decreased excitability of the first order neuron of the spinothalamic tract (pain pathway) - Several days of dosing provides full analgesia.
	ANTI INFLAMMATORY	- PGE 2 and PGD 2 release after injury is reduced. - Reduced COX activity means vasodilation and swelling is no longer favoured. - Provides symptomatic relief but less effect on underlying chronic condition.
	ANTI PYRETIC	- Pyrexia is induced when there is IL2 release as an inflammatory mediator from site of injury; IL2 affects hypothalamic temperature control. - Inhibition of hypothalamic COX-2 results in anti-pyretic effect.
	ANTICOAGULANT	- Inhibition of COX-1 results in inhibiton of thromboxane A2 production.

PARACETAMOL OVERDOSE	- Can be asymptomatic for many hours.
	- Nausea, vomitting and abdominal pain for the first 24 hours.
	- Liver damage and UQ pain- 24 to 48 hours.
	- Maximal liver damage in 3-4 days.
	- Prothrombin time is a sensitive indicator of damage.
	- Management of paracetamol overdose is ACTIVATED CHARCOAL; if overdose was within the last few hours.
	- Activated charcoal was to reduce risk of absorption.
	- Next management option is N- acetylcysteine (NAC).

NAME	TYPE	ROUTE OF ADMINISTRATION	MECHANISM OF ACTION	USES	ADVERSE DRUG REACTIONS	DRUG-TO-DRUG INTERACTIONS
MORPHINE	strong agonist	PO, PR, IV, IM, & SC	μ > κ & δ receptors	post-operative pain, major trauma, patient controlled analgesia	sedation, respiratory depression, constipation, & addiction	anti-histamines, cough relievers, & sleep medication
DIAMORPHINE	strong agonist	IV, IM, & SC	μ > κ & δ receptors	post-MI pain & dyspnoea relief in acute pulmonary oedma	sedation, respiratory depression, constipation, & addiction	anti-histamines, cough relievers, & sleep medication
FENTANYL	strong agonist	PO, IV, IM, epidural, intrathecal, nasal, & transdermal	μ > κ & δ receptors	intra-operative, general anasthesia, concious sedation, chronic/cancer pain	sedation, respiratory depression, constipation, & very high addiction	CYP3A4 inducers &/ inhibitors
ALFENTANIL	strong agonist	IV	μ receptors	post-operative pain & general anasthesia	—	—
METHADONE	strong agonist & NMDRI	PO	μ & δ receptors; N-methyl-D-aspartate receptor inhibitor	opiod dependance treatement & chronic pain	addiction, sedation, & respiratory depression	CYP2B6 inducers &/ inhibitors
CODEINE	weak agonist	PO & IM	μ > κ & δ receptors	mild pain relief, anti-diarrhoeal, & cough depressant	respiratory depression (children), constipation, & addiction	CYP2D6 inducer &/ inhibitors
TRAMADOL	weak agonist & SNRI	PO, PR, & IM	μ > κ & δ receptors; serotonin/ norepinephrine reuptake inhibitor	mild/moderate pain	constipation, addiction, psychiatric distrubance	
BUPRENORPHINE	partial agonist	transdermal, buccal, & sublingual	μ receptor agonist & κ receptor antagonist	opiod dependance treatement & moderate-to-severe pain	respiratory depression, hypotension, nausea, syncope	CYP3A4 inducers &/ inhibitors
NALOXONE	antagonist	PO, IV, IM, SC, & intranasal	μ > κ & δ receptors	opioid overodse	arrhythmia, syncope, headache	—
β-ENDORPHIN	endogenous ligand	—	μ receptors; type 1 for pain in nervous system; type 2 & 3 for respiratory depression, reduced gastro-intestinal motility, vasodilation & pupillary constriction in brainstem	appetite, sexual behaviour, & exercise pain	—	—
MET-ENKEPHALIN	endogenous ligand	—	δ receptors; increased activation of μ receptors, in brainstem	fight or flight response	—	—
DYNORPHIN	endogenous ligand	—	κ receptors; cognitive effects such as dysphoria, hallucinations, & depressed conciousness within brain and brainstem	appetite, mood, & stress	—	—
NOCICEPTIN	endogenous ligand	—	nociceptin receptors: opposite effect of μ receptors	associated with pain & fear learning	—	—

Poising protocol	Treatment		
Immediate and supportive	Remove the poerson from contact wih poison		
Actions	Vtal signs and injury		
	History- from patient if you can, chaperone,		
	packaging, written otes, anything to get time period		
	*Tricyclic anti-depressants cause resp. distress and seizures.		
Prevention of absorption	Gatric levage = never!		
	Actuvated charcoal, large quantity needed		
	Timing of overdose makes efficay of charcoal unpredictable		
	Later= modified relase prep. and antimuscarinics		
	Not suitable for comatose/drowsy patients due to risk of		
	aspiration		
Enhance elimation	Continue activated charcoal - up to 36bhrs: phenobarbital		
	and benzodiazepine		
	Sodium bicarbonate- alkaline diuresis, high pH		
	eg salicylate poising (weak acid)		
	Forced diuresis is not reccomeneded		
	Haemodalysis for drugs with small vd		
	*lipophilic drug= in tissue therfore haemodialysis won't work		
Antidotes	Overdose	Drug	MOA
	Competitive Antagonist		
	Opiod	Naloxone	High affinity for opiod receptors that dispalces opiod quickly but short acting.
	Certain organophosphates/acetylcholinesterase inhbitors	Atropine	Competitive antagonist of muscarinic ACh receptor types M1-5.
	Chealating agents		
	Ironand aluminum chelating agent	Desferrioxamine	
	Cyanide poising	Soium nitrate and sodium thiosulfate in combo/hydroxocobalamin	Binds cyanide and forms non-toxic stable water soluble vit b that is removed.
	Manipulating drug use		
	Paracetamol	Acetylcystine	Precursour of gluthaione, inc. non-toxic metobolite of paracetamol
	Ethylene glycol (antifreeze poisin)	Fomepizole	Comptetitive inhibitor of alcohol dehydrogenase enzyme
	Antibodies		
	Digoxin	Digozin specific antibody (DigiFab)	
	Dabigatran	Idaruizumab (Parabind)	
	Factor Xa inhibitors	Recombinant modified human factor Xa protein	
		Andexanet alfa (ondexya)	Specific reversal agent for factor Xa inhibitors. Inc. normal factor Xa in body

*MENSA is given when prescibing cyclophosphamide to prevent hemorrhagic cystitis. This inhibits Acrolein which is a metabolite of cyclophosphamide. TOXBASE is used in UK ti find info on poisining.

CATEGORY	EXAMPLE	MOA	USE	ADRs
Hormonal contracepion	COCP	Interruption of physiological control of the menstrual cycle.		For COCP:
	Progesterone depot	Primary action is to inhibit ovulation but has endometrial		Risk of thromboembolism
	Progesterone implant	and cervical mucus effects		is dose dependent.
	Low dose progestogen		*Used in older women, and risk of thrombosis	
Hormone Replacement Therapy	Estrogen	Oestradiol: Valerate	In women who has hysterectomy	Risk of breast cancer
				Venous thromboembolism
	Progesterone	Medroxyprogesterone acetate (Provera)		Risk of stroke
			Intact uterus give both so progesterone	Ototoxicity
	Combined	Prempack C	can protect against endometrial cancer.	Hypokalaemia
Inhibitors and Antagonists	Mifepristone (RU486)	Progesterone and glucocorticoid receptor antagonist.	Termination of pregnancy	
		Anti progesterone.		
		Sensitises the myometrium to prostaglanding-induced contractions.		
SERM	Tamoxifen	ER antagonist: binding of ER causes cells to arrest the cell cycle.	Hormone receptor positive breast cancer.	Endometrium- ER agonist
Selective Estrogen Receptor				Increase risk of endo. cancer.
Modulator	Raloxifene	ER agonist in bone!	Post menopause osteoporosis & breast canc.	
	Clomiphene	competes with oestrogen for ER binding. Leads to ovulation induction.	Treatment for anovulation	Significant ovulation eg sextuplets
Selective Progesterone	Ulipristal acetate	Delay or inhibit ovulation	Emergency contraception (within 60 hrs.)	
Receptor Modulator			Uterine fibroids	

RESPIRATORY DRUGS	CLASS	EXAMPLES	MOA	USES	SIDE EFFECTS
BETA AGONISTS	SHORT ACTING	SALBUTAMOL	- Primarily work on relaxing bronchial smooth	- To give an immediate relief of asthma	- Headache
			muscle; open up the airway and ease air flow.	attacks; only work for a while.	- Dizziness
			- Interacts with membrane bound Gs to stimulate		- Tremor
			cAMP production.		- Hypokalaemia
			- Calcium channels open and reduce phosphoryl.		- Heart palpitations
			of myosin light chains.		
			- Increase mucociliary clearance and reduced		
			mucus build up.		
		TERBUTALINE			
	LONG ACTING	FORMETEROL		- Most of them used in combination with	
		INDACTEROL		ICS; longer term relief.	
		SALMETEROL		- Asthma and COPD treatment.	
INHALED CORTICOSTEROIDS		BECLOMETHASONE	- Potent anti inflammatories	- Asthma treatment	- Throat irritation
(ICS)		BUDESONIDE	- Perfuse into cells, bind to specific receptor		- Oral thrush
		CICLESONIDE	proteins and stimulate synthesis of LIPOCORTIN.		- Hoarseness of voice
		FLUTICASONE	- Inhibit synthesis of prostaglandins and leukotriene		- Need for antifungal treatment
			mediators from macrophages, monocytes and		- Systemic side effects (unlikely); osteoporosis, derm
			mast cells.		
LEUKOTRIENE RECEPTOR		MONTEKULAST	- Both bronchodilator and anti-inflammatory	- New class of drug for asthma treatment;	- Diarrhoea, vomitting
ANTAGONISTS		ZAFIRLUKAST	- Production of leukotriene by WBCs cause	available in tablet form	- Fever
			bronchocontriction; antagonising means relaxation.		- GI discomfort
					- Headache
					- Dry mouth
METHYLXANTHINES		THEOPHYLLINE	- Noncompetitive inhibition of PDE 4 enzyme	- Commonly seen in later control of asthma.	- High risk of toxicity (narrow therapeutic window)
			- Intracellular rise in cAMP and cGMP.		- Arrhythmia
			- Inhibition of myosin light chain kinase; enzyme		- Headache
			responsible for phosphorylation of myosin (contraction)		- Nausea
					- Palpitations
					- Seizures
LONG ACTING ANTI-		ACLINIDIUM BROMIDE	- All of them in the form of inhalers except Tiatropium		- Arrhythmias
CHOLINERGICS		GLYCOPYRONNIUM	(tablet).		- Cough
		TIATROPIUM	- Competitive inhibition of ACh on muscarinic receptors.		- Dry mouth
		IPRATROPIUM	- Reduced muscle tone and dilation of airways.		- Nose bleeds
		UMECLIDINIUM			- Headache
					- Nausea
					- Contraindicated in patients with prostate problems a