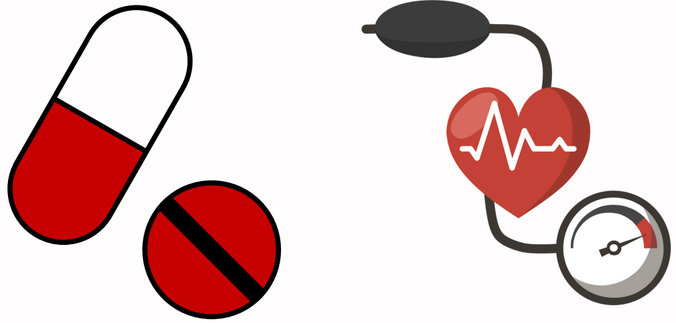
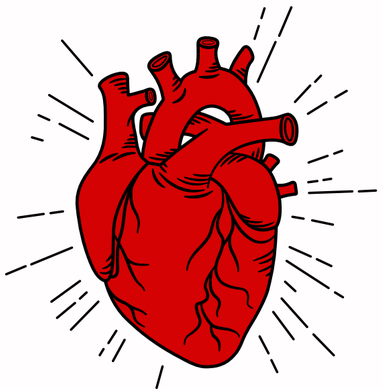


ANTI HYPERTENSIVES

DONE BY : ZEINA YASSIN & NOOR MARZOOQ

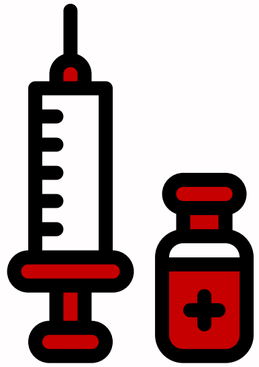


Before we start:

This file **doesn’t** include all the study material !
It’s just a simple summary with the drug names, their main mechanisms, side effects, and a few important notes. It’s meant to make the information easier to memorize or review, and to help you organize the topic more clearly.
You’ll find a QR code that you can scan , or simply click on , and it will take you to helpful short videos(The required ones are marked in red).

Good luck!❤️

Anti Hypertisives Classes :



- Diuretics
- Calcium channel blockers
- Beta blockers
- Angiotensin converting enzyme (ACE) inhibitors (ACEI)
- Angiotensin Receptor Blockers (ARBs)
- Peripheral a1-adrenergic antagonists
- Adrenergic neuron blocking agents (Sympatholytics)
- Central a2-adrenergic receptor agonists
- Vasodilators

DIURETICS

Drug Name	MOA	Side effects	Notes
Thiazide diuretics	inhibit sodium-chloride reabsorption	-adversely affect serum lipids and can reduce insulin sensitivity -Requires 2 weeks to become fully effective -PVR may increase at first hyperuricemia(trigger or worsen gout attacks)	-Adequate treatment of BP control in patients with mild or moderate primary hypertension -LD: Most efficacious in “low renin” or volume-expanded forms of hypertension -Efficacy of diuretics may be compromised during kidney failure
Loop diuretics	blocking the sodium-potassium-chloride (NKCC2) cotransporter.		
potassium-sparing	____		
Carbonic anhydrase inhibitors	____		

CALCIUM CHANNEL BLOCKERS

Category	Drug Name	MOA	Side effects	Notes
Dihydropyridines	Amlodipine Nifedipine	-Block Calcium in vascular smooth muscle -Decrease PVR - No effect on AV node conduction	-Reflex tachycardia -Hypotension -Headache -Flushing -Constipation -Edema	Amlodipine combination with ACE inhibitor reduced CV events in hypertensive patients
Non-dihydropyridines	Verapamil	Direct negative inotropic and chronotropic action (cardiodepressive)	-Cardiac depression -Hypotension -Headache -Flushing - Constipation	-Verapamil may causes heart failure in patients with borderline cardiac reserve - should not be given with Beta Blockers (bradycardia and cardiac conduction blockade) -Digoxin should not be given with them (risk of AV block and arrhythmia)
	Diltiazem	-Decreases AV conduction and heart rate -Weaker negative inotrope than verapamil		

BETA-BLOCKERS

Drug Name	MOA	Side effects	Notes	Contraindications
Propranolol	Nonselective (competitive antagonist of β1 and β2 adrenergic receptors)	-Hypotension, AV block, severe bradycardia,HF. - Bronchial constriction (spasm). -Acute withdrawal syndrome. - Increase triglyceride & decrease HDL levels . -Induce glucose intolerance -cross BBB (Nightmares/depression)	-Delays AV node conduction (useful in the management of cardiac arrhythmia). - Neutralize reflex tachycardia induced by vasodilators. -Lipid soluble. -Effective in patients with high renin activity	-Bronchial asthma - Peripheral vascular disease - AV (heart) block - **Not contraindicated:** in patients with ischemic heart disease secondary to dyslipidemia
Atenolol	β1 selective antagonist	-----	-Administered once daily -Less lipid soluble than. other β antagonists.	----
Metoprolol	Selective inhibitor to β1	Useful in asthmatic patients	-----	----

BETA-BLOCKERS

Drug Name	MOA	Side effects	Notes
Nadolol	Non-selective β antagonist	------	-Administered once daily.

ACE INHIBITORS

Drug Name	MOA	Side effects	Notes	Contraindications
Enalapril	-Inhibit conversion of inactive angiotensin I to angiotensin II which: 1. Reduces vessel tone 2. Reduces Na^+ retention via aldosterone 3. Blocks degradation of bradykinin, a vasodilator	-Severe hypotension. -Hyperkalemia. -Angioedema. -Cough. - Skin rash . -Taste alterations.	-Very useful in diabetic patients -Slows progression - Enalapril : Excretion primarily renal. -Lisinopril : Slowly absorbed. -Captopril: Sulfhydryl containing moiety causes some taste changes.	-Pregnancy(should not be administered in second or third trimester) - African American population, because of low renin. -Hyperkalemia (exacerbated with potassium sparing diuretic).
Ramipril				
Lisinopril				
Captopril				

ANGIOTENSIN I RECEPTOR BLOCKERS (ARB'S)

Drug Name	MOA	Side effects	Notes
Losartan	They block Angiotensin II receptor type 1 . -Decreases TPR . -Inhibits Aldosterone release. - Block Na+ reabsorption.	-Angioedema (Subcutaneous swelling of eyes and lips). - Dizziness	-Not to be administered during pregnacy (first trimester also in 2 nd & 3 rd trimesters) -AT receptors important in embryonic renal development.

PERIPHERAL A_1 BLOCKERS

Drug Name	MOA	Side effects	Notes
Prazosin	-Blocks a1 -AR on resistance vessels from binding NE released from nerve terminals -Decreases vascular tone(vasodilates) -Thereby decreases PVR and BP	-Postural dizziness -Headaches -Drowsiness - 'first dose phenomenon' -- Syncopal reaction-orthostatic hypotension (upon standing) - After first dose, tolerance to this reaction	-Relaxes prostate and bladder neck muscles in older patients to improve urination. Blood pressure should be monitored, especially with other medications.
Doxazosin Terazosin		----	-longer t1/2 than prazosin -Used for treatment of benign prostate hypertrophy

-α-blockers are less effective than diuretics in preventing cardiovascular events, mainly heart failure
-NIH recommends NOT to use a-blocker as the first drug of choice in hypertension (it is safe, just not effective in preventing heart failure)
-A reasonable addition, to facilitate blood pressure control

SYMPATHOLYTICS

Drug Name	MOA	Side effects	Notes	Drug Interactions
Guanethidine	-Enters peripheral nerve terminals via same transporter as NE - Depletes NE stores in vesicles -False neurotransmitter	-Orthostatic hypotension (Guanethidine) - Depression - Nasal Congestion -Bradycardia -Impotence (Guanethidine) -Diarrhea (Guanethidine) - Salt and water retention	-Effective orally (takes 72 hours to reach maximum effect) - Plasma t1/2 – approximately 5 days. - Guanethidine is indicated only for moderate to severe hypertension.	- Drugs that alter function of the amine pump can block uptake to site of action: tricyclic antidepressants, monoamine oxidase inhibitors, ephedrine, amphetamines, phenothiazines . - After chronic use of guanethidine, the above agents could cause hypertension due to development of receptor supersensitivity.
Reserpine	-Blocks transport of dopamine into storage granules in nerve terminals - Depletes stores of catecholamines and serotonin in CNS and PNS -Decreases sympathetic tone, total peripheral resistance and cardiac output		-Absorbed from GI tract (2-6 wks to achieve maximal effect) - Plasma t1/2 – 11.5-16 days -Largely hepatic metabolism	

CENTRAL A2-ADRENERGIC RECEPTOR AGONISTS

Drug Name	MOA	Side effects	Notes	Indications	Drug Interactions
Methyldopa	After activation of the sympathetic nervous system and the release of norepinephrine (NE), the NE binds to presynaptic α_2-receptors to provide negative feedback, which reduces further NE release. **These drugs mimic this action**	-sedation -dry mouth - sodium retention -dizziness -hemolytic anemia (rare)	Prodrug taken up by central adrenergic neurons and converted to the a2 adrenergic receptor agonista - methylnorepinephrine	first choice for hypertension during pregnancy	-Tricyclic antidepressants may prevent the antihypertensive effect -Barbiturates may reduce the efficacy of through induction of hepatic microsomal enzymes -Monoamine oxidase inhibitors when coadministered may produce hypertension and CNS stimulation
Clonidine		-dry mouth - sodium retention -dizziness -drowsiness	Oral plasma t1/2 – 12-16 hrs -Transdermal administration of clonidine by patch (replaced once per week) useful in patients unable to take oral medication	useful in the diagnosis of pheochromocytoma (adrenal tumor	

GANGLIONIC BLOCKERS

Drug Name	MOA	Side effects	Organ effects
Trimethaphan	Blocks transmission in both sympathetic and parasympathetic systems by acting on autonomic ganglia	-potentiates tubocurarine -histamine release causing bronchoconstriction and increased respiratory secretions	Blood vessels: vasodilation Heart (SA node): tachycardia Heart (ventricles): decreased contractile force Iris: mydriasis Gastrointestinal tract: constipation Urinary bladder: urinary retention Salivary glands: dry mouth Sweat glands:anhidrosis
Pentolinium Mecamylamine		—	

VASODILATORS

Drug Name	MOA	Pharmacokinetics	Side effects
Hydralazine	- Direct arteriolar vasodilation - Alters smooth muscle Ca^{2+} by hyperpolarizing the cell -Decreases total peripheral resistance -Reflex sympathetic activation: increased heart rate, increased contractility, increased plasma renin activity	-plasma half-life 1 hour -Antihypertensive action lasts 12 hours (possibly due to storage in arterial wall)	- Reflex tachycardia - Can precipitate MI in elderly or CAD patients - Reflex response can be blocked by propranolol - Sodium and water retention (preventable with diuretic) - Headache, nausea, dizziness - Lupus syndrome

VASODILATORS

Drug Name	MOA	Pharmacokinetics	Side effects	Notes
Minoxidil	-Activates ATP-sensitive K^+ channels causing hyperpolarization and smooth muscle relaxation -Arteriolar vasodilation -Decrease in total peripheral resistance	-plasma half-life 4 hours - Hypotensive effect lasts 12–24 hours - Must be metabolized in liver to active metabolite (minoxidil N-O sulfate)	-Similar to hydralazine - Hypertrichosis (excessive hair growth) -Must be given with a diuretic and a sympatholytic agent (usually β-blocker)	-Reserved for severe hypertension
Fenoldopam	Dopamine D1 agonist causing vasodilation, renal vasodilation, and natriuresis	-Rapidly metabolized - Short acting	----	Given by continuous infusion in emergencies or postoperatively

VASODILATORS IN TREATMENT OF HYPERTENSIVE CRISIS

Drug Name	MOA	Pharmacokinetics	Side effects
Sodium Nitroprusside (SNP)	• Liberates nitric oxide causing vascular smooth muscle dilation • Decreases total peripheral resistance	-Given by IV infusion -Light sensitive, unstable in aqueous solution - Antihypertensive effect ceases when infusion stops - Metabolized to sodium thiocyanate (slow renal clearance) -Cyanide accumulation can cause lactic acidosis	- Rebound hypertension -Tolerance
Diazoxide	-Dilates arterial smooth muscle via activation of KATP channels -Little or no effect on venous smooth muscle -Decreases total peripheral resistance	- Administered IV -Onset within 2 minutes -Duration 6–24 hours	-Tachycardia -Angina

VASODILATORS IN TREATMENT OF HYPERTENSIVE CRISIS

Drug Name	MOA	Pharmacokinetics
Labetalol&Carvedilol	-Mixed α_1 and non-selective β adrenergic receptor antagonists -Block receptors in blood vessels and heart -Labetalol selectivity α_1:β = 1:3 -Carvedilol selectivity α_1:β = 1:10 -Decrease total peripheral resistance without reflex tachycardia	-Administered orally or IV (for hypertensive crisis) -Useful in pheochromocytoma (Labetalol) -Plasma half-life: 2 hours (oral), 5 hours (IV)

In the world full of anxiety and tachycardia; be someone's beta blocker 🫂

If this helped, remember us in your prayers <3

With our warmest regards and best wishes!❤️