Hyptension			
Diuretics:Thiazides	Initial ↓blood volume, Chronic ↓TPR	Effective in AA, >55, obese	
Sympatholytics: A1 B1 antagonists + a2 agonists			
Peripheral α1 Blockers Doxazosin, Terazosin **Prazosin**	Competitively block α1 receptors on vascular smooth muscle→ ↓TPR	Mild to moderate hypertension Can be used in **BPH***	**Postural hypotension, syncope,** nasal congestion ↓LDL, ↑HDL
Central α2 agonists	α2 autoreceptor agonist in the brain stem: **Gi** *K channel→ Hyperpolarization*: ✖CNS sympathetic outflow ✖NE release →↓TPR+ ↓Renin release		**Sedation, orthostatic hypotension**
Clonidine			Rebound hypertension
αMethyldopa		Mild Hypertension in Pregnancy	Rare: Autoimmune hemolytic anemia: +coombs
Reserpine	Irreversibly block VMAT: net depletion of neurotransmitters + ↓sympathetic outflow		Depression like syndrome GI
Guanethidine	False neurotransmitter Taken up by NET, displaces NE as it is concentrated into vesicles→ ↓Sympathetic outflow		≠TCAs: *block reuptake*
B -Blockers	Competitively block βreceptors ↓**Myocardial O2 Demand** • ↓CO: SV*HR: *Chronotropic* • ↓Force of contraction: *Ionotropic* ↓Renin→↓AngII→↓TPR: *Eventual decrease in LV mass* ↑Myocardial perfusion due to an augmented diastolic perfusion time ↓cAMP→ ↓SA+AV Nodal activity	***Younger white patients*** **Angina Pectoris,** MI, **CHF** Thyrotoxicosis Pheochromocytoma Migraine prophylaxis **Protective**: Post MI Chronic Prophylaxis: stable angina Acute treatment: unstable angina **Anti-Arrhythmic (EMP)**	**Impotence, Bradycardia, AV Block** Treat overdose: Saline, Atropine, Glucagon **≠Asthma/ COPD** **Diabetes**, *Additive: cardiac depressants* ***Non selective BBlocker*** *Vasoconstriction + paradoxical ↑blood pressure* **≠Vasospastic: Prinzmental/Invariant angina**
Atenolol, Esmolol Metoprolol	B1 selective; ↑Bioavailability Esmolol: Short acting	Prefered in diabetics + Asthma ***Block reflex tachycardia with Vasodilators***	 Metoprolol→ Dyslipidemia
Nadolol Propranolol	Non selective B1, B2; t½: 4-5 hours ↓Bioavailability	HTN + Coronary Artery disease	Rebound hypertension Can precipitate CHF
Timolol		*Glaucoma eyedrops*	
Acebutolol, Pindolol	B partial agonists		
Labetalol Carvedilol	Block β1,2 + α1: ↓Systolic + Diastolic BP	B1, B2 + α1 *Cocaine/ Epi overdose*	Postural hypotension

Calcium Channel Blockers	**✖L-type calcium channels** ***Block Ca influx into cytosol***: → ***block contraction*** ↓Cardiac O2 Demand via ↓Afterload + ↓Cardiac inotropy + Chronotropy ↑Cardiac O2 delivery First pass metabolism		Simple Hypertension Coronary Vasospasm: ***Prinzmental Angina*** Raynaud phenomenon	**SA Node Depression:** ***Bradycardia***, *SA nodal arrest* **Negative ionotropic effect**: **CHF** Gingival Hyperplasia, Peripheral edema, flushing, dizziness, constipation ≠βblockers: augments effects **Do not combine***
Dihydropyridines	Potent Vasodilation Block **L-type Ca** channels in **Vessels**: ↓TPR —> decrease AV node ERP —> increased Ventricular rate		Hypertension Angina: Excertional + Variant	**Gingival Hyperplasia** **Tachycardia** ≠Cardiogenic shock, systolic HF, Ven. Tachycardia, SA + AV block, Wolf Parkinson White AFIB
Nifedipine	Most Effective Anti-Hypertensive		Angina, hypertension, Raynauds	
Amlodipine			Angina Hypertension	
Clevidipine			Hypertensive emergency	
Nisoldipine	Selective for coronary vasculature		*Angina*	
Nimodipine	Selective for cerebral vasculature		*Transient Ischemic attacks*	
Phenylalkylamines **Verapamil**	Block **L-type Ca** channels: **Heart**	**Block SA + AV Node conduction** **↑ERP ↑PR interval** ↓Contractility, HR ***↓Firing rate of aberrant pacemaker sites →*** ***Block reentry mechanisms***	**Systemic hypertension** **Angina** Diastolic Heart Failure **Anti-Arrhythmic:** ***Supraventricular Tachyarrhythmias*** AFib, Aflutter	AV Block, Edema, Hyperprolactinemia **Constipation + GE-Reflux:** *Affect other smooth muscles*
Benzothiazepines **Diltiazem**	**Vessels + Heart**			**AV block** Ven Fib

Anti- Angiotensin II drugs: ↓**CO**, AHD + Aldosterone + ↓AngII effects→ ↓**TPR**		✓**Diabetes** ✓**Renal insufficiency**	≠**Pregnancy, Bilateral Renal Stenosis**: *Precipitate renal failure*
ACEi Benazepril Fosinopril **Captopril** **Enalapril:** IV hypertensive emergency	✖ACE: ↓**Angiotensin II** • ↓Vasoconstriction: ↓Afterload • Kidney: Efferent arteriolar dilatation→ ↓GFR: → ↑*Renin* • ↑Bradykinin release: Vasodilation • ✖Aldosterone release→ prevent Na + H20 retention *Beneficial in patients: LV systolic dysfunction, HF, stroke, diabetic/hypertensive nephropathy*	**1° Hypertension** **CHF**, proteinuria, diabetic nephropathy *Prevent heart remodeling from chronic HTN* **Black: coadminster with Thiazide**	*African Americans do not respond* • **Cough (Bradykinin)** • **Angioedema** • Teratogen: *renal malformations* • ↑Creatinine: **(↓GFR)** • Hypotension • **Hyperkalemia** ≠NSAIDS
Ang.II R blockers:ARBS Losartan Candesartan Valsartan	Selectively **block AT-1** Receptor • ↓Vasoconstriction: ↓Afterload • ✖ AngII kidney effects + aldosterone stimulation: ↑Renal excretion of Na + H20 • Less of ↓GFR compared to ACEi *No effect on bradykinin*	Hypertension, HF, proteinuria, diabetic nephropathy *Intolerent to ACEi*	• Teratogen: *renal malformations* • ↑Creatinine: **(↓GFR)** • Hypotension • **Hyperkalemia** ≠NSAIDS
Aliskiren	**Direct renin inhibitor** Block conversion: *Angiotensinogen* → *Ang.I*	Hypertension	• **Stroke**, Angioedema • Hyperkalemia, Hypotension • Renal toxicity ≠ACEi or ARBs
Direct Vasodilators: Augment NO: Orally active			
Hydralazine	NO prodrug: **Arteriole dilation: ↓Afterload** Direct vasodilator via NO release: ↑cGMP	Severe HTN, HF Safe in pregnancy	**Tachycardia:** *give BBlocker* **Edema:** *give diuretic* **SLE-like syndrome** ≠Angina/ CAD
Minoxidil *Rogaine*	Open ATP-dependent K channels→ Hyperpolarization: ↓TPR;Arteriole vasodilation	**Male pattern baldness**	**Hypertrichosis**, Tachycardia, Edema ↓Insuline release: Diabetogenic
Diazoxide	Open ATP-dependent K channels	Insulinoma	Diabetogenic
NO Inducers:			
Nitrates	*See Angina*		
PDE5 inhibitors: **Sildenafil**- *Viagra*	Indirectly ↑NO effects *Not anti-hypertensives*	Erectile Dysfunction	*≠with Nitrates:* → *Life threatening Hypotension*
Nitroprusside	Short acting Venous + Arterial Vasodilation: Direct NO oxide release ↑cGMP pathways Metabolized→ CN CN+ Methemoglobin→ Cyanomethemoglobin	IV: **Hypertension Emergency**	**Cyanide toxicity**: *Treat:* ***1. NaNitrite 2. Na Thiosulfate****: cyanometHb* → *Thiocyanate: ↑kidney excretion* Tachycardia, postural hypotension

Nitrite: Induce methemoglobinemia: oxidize iron to +3
which has higher affinity for CN: binds CN
Give Thiosulfate: binds to CN and converts to inactive to be excreted