Drug	Type/Classification	Mechanism	Indications	Contraindications
Acetazolamide	Diuretic	Block carbonic anhydrase (CA) = bicarbonate remains in the tubular lumen and excreted = Na+ remains in lumen and excreted (*water will follow*)	*Limited usefulness as a diuretic* - More for treatment of glaucoma To correct for **metabolic alkalosis** (increased pH) - Blocking CA and losing bicarbonate will decrease pH Altitude illness/acute mountain sickness (prophylaxis; symptomatic relief)	**Severe COPD**
Mannitol	Diuretic (Osmotic)	Increases osmolarity of plasma = increases osmolarity of tubular fluid (Mannitol freely-filtered in the glomerulus and non-reabsorbable) = more water in tubule = more Na+ remains in tubule due to concentration gradient decrease (*water will follow*)	Restore osmotic equilibrium after hemodialysis Decrease intraocular pressure (acute glaucoma) Reduce cerebral edema (neurosurgery)	**Pulmonary edema** (heart failure, pulmonary congestion) - Mannitol would further increase the water in extracellular space = compromise gas diffusion
Furosemide	Diuretic (Loop)	Inhibition of Na/K symporter in thick ascending LoH (critical mechanism of Na transport) *High* efficacy (high-ceiling) due to significant contribution of TALoH to reabsorption of Na+ and nephron segments past TALoH not capable of rescuing the solutes	*Widely-used* class of diuretics Beneficial for **pulmonary edema** - Increased venous capacitance -> decreased left ventricular filling pressure **CHF** = decreased preload, BP *Less useful for HTN treatment* compared to thiazide-type diuretics	

Hydrochlorothiazide	Diuretic (Thiazide)	Inhibition of Na/Cl symporter in distal convoluted tubule = increased NaCl excretion	**HTN** = best initial therapy in uncomplicated cases, most frequently used class of anti-hypertensive agents Chronic (longer half-life) and conditions associated with **edema**: CHF (decreased preload, BP), hepatic cirrhosis, kidney diseases with preserved GFR	**NSAIDs** blunt the hypotensive effects of thiazide diuretics **TRIPLE THERAPY** (NSAID + diuretic + ACE inhibitor/AT1 receptor blocker) = acute renal failure
Amiloride	Diuretic (K+ sparing)	Inhibit Na+ channels in the late distal tubule and collecting duct - Direct effect = decreased Na+ reabsorption - Indirect effect = inhibition of K+ secretion driven by increased Na+ delivery to late distal tubule/collective duct by result of other diuretics	Rarely used alone (modest natriuretic effect, can cause hyperkalemia); Usually applied for their K+ sparing actions with other diuretics (loop and thiazide diuretics = hypokalemia) - Amilozide = **Amiloride + Hydrochlorothiazide** = prevent hypokalemia	*Do not use with **ACE inhibitors or AT1 blockers (RAA inhibitors)*** - Increased risk of hyperkalemia
Spironolactone	Diuretic (Aldosterone antagonist)	Block aldosterone receptors in late distal tubule and collecting duct = decreased Na+ channel expression Major = Inhibit K+ secretion (similar to *Amiloride*, except blocking the expression of channels, not the channels themselves)	Only diuretic effective for **kidney failure** (decreased GFR) - Acts through capillary side, not tubular lumen - Don't need to be filtered through glomerulus in order to act Co-applied with loop or thiazide diuretics due to K+ sparing characteristic **CHF** = decreased preload, BP	*Do not use with **ACE inhibitors or AT1 blockers (RAA inhibitors)*** - Increased risk of hyperkalemia

			Resistant HTN due to primary aldosteronism	
Vasopressin	Anti-Diuretic	Activate V1 receptors in smooth muscle of GI tract and BVs = vasoconstriction	Vasodilatory shock Visceral bleeding Ileus	
Desmopressin	Anti-Diuretic	Preferential activation of V2 receptors in collecting duct = increases permeability of water due to increased aquaporin channels	Polyuria/polydipsia in central diabetes insipidus (insufficient ADH supply to pituitary) Primary nocturnal enuresis	**NSAIDs** and **morphine** = *potentiate* anti-diuretic effects = risk of water intoxication **HTN, heart failure**
Clonidine **Methyldopa**	Centrally-acting Adrenergic neuron inhibitor	Activate a2-adrenergic in brainstem vasomotor center = vasodilation = decreased vascular resistance	Methyldopa = **pregnancy-induced HTN** (not teratogenic)	*Dental Note*: Centrally-acting sympatholytic *causes* **xerostomia**
Reserpine	NE-depleting agent (*all you need to know)*			
Prazosin **Phentolamine**	Selective a1 inhibitor Non-selective	Inhibit a-adrenergic receptors in smooth muscle of arteriolar resistance vessels and veins = vasodilation = decreased vascular resistance	In conjunction with other anti-hypertensive agents (diuretics) *Dental Note*: **Phentolamine** used to remove LA by reversing vasoconstricting effect of sympathomimetic (EPI)	
Propranolol **Bisoprolol** **Labetalol**	B-blocker B-blocker B-blocker + a1 antagonist Class II Anti-Arrhythmic Drugs	Inhibit B-adrenergic receptor signaling = decreased renin secretion, cardiac output (HR, contractility)	**HTN**, **exertional angina**, **CHF** (decreased mortality) **Arrhythmias**	**Asthma** – can cause life-threatening bronchoconstriction **Diabetes** – altered sensitivity to insulin (increased risk of hypoglycemia) Abrupt discontinuation = sudden death, exacerbate

				angina (increased density of receptors due to sensitization) **NSAIDs** blunt the hypotensive effects of thiazide diuretics **EPI** (in LA) can *severely* raise BP (**reflex bradycardia**) when also taking non-selective B-antagonist
Captopril **Enalapril** **Ramipril**	ACE inhibitors	Inhibit ACE (decreased conversion of angiotensin I to II) = decreased BP Potentiated by decreased Na+ and diuretics = increased renin release	**HTN** (except primary aldosteronism) **Acute MI**, coronary artery disease **Diabetes Mellitus** (renoprotective) **CHF**	**Pregnancy** – teratogenic **Angioedema** – nose, mouth, throat, larynx, glottis **NSAIDs** blunt the hypotensive effects of thiazide diuretics **TRIPLE THERAPY** (NSAID + diuretic + ACE inhibitor/AT1 receptor blocker) = acute renal failure
Losartan **Valsartan**	AT1 antagonist	Inhibit angiotensin II receptor type 1 (AT1) = decreased systemic vascular resistance (BP), aldosterone release, catecholamine release (adrenal medulla and sympathetic neurons) Potentiated by decreased Na+ and diuretics = increased renin release	**HTN** (except primary aldosteronism) **Heart failure** **Acute MI,** coronary artery disease **CHF**	**Pregnancy** – teratogenic Acute renal failure **NSAIDs** blunt the hypotensive effects of thiazide diuretics **TRIPLE THERAPY** (NSAID + diuretic + ACE inhibitor/AT1 receptor blocker) = acute renal failure

Aliskiren	Direct renin inhibitor (*all you need to know*)			
Nifedipine **Amlodipine** **Verapamil** **Diltiazem**	Ca2+ Ch antagonist (Dihydropyridines – first two only) Class IV Anti-Arrhythmic	Inhibit L-type Ca2+ channels - Arterial vasodilation = decreased vascular resistance - Coronary vasodilation = increased coronary blood flow	HTN, **exertional and variant angina** **Arrhythmias**	*Dental Note*: Can cause **gingival hyperplasia** *Note*: **Verapamil** inhibits **CYP3A4** *Side effect*: Increased plasma concentration of **Digoxin** by Verapamil
Hydralazine	Direct vasodilator	Unknown but powerful vasodilator and secondary sympathetic activation (baroreceptor unloading = increased HR)	Severe HTN HTN emergencies of pregnancy (preeclampsia) **CHF**	Dangerous = can lead to MI (*powerful*)
Sodium Nitroprusside	Direct vasodilator	Release NO = smooth muscle relaxation = vasodilation Compared to hydralazine = safer (less effect of baroreceptor unloading)	HTN emergencies Surgeries (short-term reduction in BP to reduce bleeding) **CHF**	
Minoxidil	Direct vasodilator	**Prodrug** = metabolized by liver to active metabolite = smooth muscle relaxation = vasodilation = decreased peripheral vascular resistance	HTN, severe and poorly-responding to other medications (but not for emergencies)	*Side effect*: hypertrichosis (excessive hair growth) Liver cirrhosis = less effective conversion
Glyceryl trinitrate (Nitroglycerin) **Isosorbide dinitrate**	Organic nitrates	Decrease oxygen demand by vasodilation *Direct absorption = fast = avoid first-pass metabolism*	**Exertional angina** **Acute MI** (except patients with hypotension)	Severe hypotension in patients with autonomic dysfunction, treated with erectile dysfunction with

			CHF **GERD** – relieve esophageal spasms (smooth muscle relaxation)	phosphodiesterase 5 inhibitors Headaches (often severe)
Digoxin	Cardiac glycosides	Inhibit sacrolemmal Na/K ATPase = increase cytosolic Na+ = decrease Ca2+ efflux = increase cardiac contractility due to Ca2+ accumulation in SR	**CHF** – limited to pts who don't improve on ACE inhibitors and B-blockers at maximal doses; very low therapeutic range	*Side effect*: **Ventricular arrhythmias (overdose)**, elevated extracellular K+ reduces digoxin binding to Na/K ATPase = decreased effectiveness
Lidocaine **Quinidine**	Class I Anti-Arrhythmic (Na+ blockers)	Block Na+ channels = decrease excitability threshold = decrease automaticity Block K+ channels = increase AP duration (only Quinidine)	**Arrhythmias**	*Note*: **Quinidine** inhibits **CYP2D6** (decreased codeine-morphine metabolism) = decreased opioid analgesia Reversal of local Lidocaine by **Phentolamine** can result in dangerous cardiac depression, esp patients with liver or kidney disease = **increased elimination half-life of Lidocaine**
Amiodarone	Class III Anti-Arrhythmic (K+ blockers)	Block K+ Ch = increase AP duration = increase refractoriness = decrease abnormal automaticity	**Arrhythmias**	*Note*: **Amiodarone** inhibits **CYP450** *Side effect*: Long-term treatment may lead to blue-grey discoloration of sun-exposed areas, oral hyperpigmentation
Aspirin **Ibuprofen**	COX inhibitor (anti-platelet agent) NSAIDs	Inhibit synthesis of thromboxane A2 (involved in platelet activation and vasoconstriction); irreversibly binds COX1	*Look below for more detail*	

Clopidogrel	ADP receptor inhibitor (anti-platelet agent)	**Prodrug** Target P2Y1/P2Y12 = critical for platelet aggregation		Liver cirrhosis = less effective conversion
Abciximab **Eptifibatide**	GP IIb/IIIa inhibitor (anti-platelet agent)	Complex plays a crucial role in the final step of platelet aggregation		
Heparin **Enoxaparin** **Dalteparin**	Directly acting anticoagulant Low MW heparin	Decrease thrombin and Xa Decrease Xa (not thrombin)		***Antidote = Protamine Sulfate***
Hirudin **Bivalirudin** **Dabigatran (Pradaxa)**	Direct thrombin inhibitor (direct oral anticoagulant)			***Antidote = Idarucizumab (Praxbind)***
Rivaroxaban (Xarelto) **Apixaban (Eliquis)**	Direct Factor Xa inhibitor			***Antidote = Andexanet Alfa (Andexxa)***
Warfarin	Indirect oral anticoagulant	Competitive inhibitor of VitK epoxide reductase - VitK critical for Factor II, VII, IX, X = no longer serve as cofactor in synthesis = block activation of clotting factors *Note*: **CYP2C9**-mediated metabolism - Induced? Warfarin decrease = less anticoagulation - Inhibited? Warfarin decrease = more anticoagulation		***Antidote = Vit K*** Risk of uncontrolled bleeding with anti-platelet treatment (**Aspirin, Ibuprofen, Clopidogrel**)
Alteplase	Fibrinolytic	Stimulate plasminogen -> plasmin	Relieving thromboses (e.g., **acute MI, pulmonary embolism, ischemic stroke, DVT**)	
Aminocaproic Acid **Tranexamic Acid**	Anti-fibrinolytic	Competitive inhibition of plasminogen and plasminogen	Oozing sockets after dental extractions	

		activators from binding to fibrin = limited fibrinolysis	Post-surgery in hemophilics	
Articaine	Amide + Ester = metabolized in plasma and liver	Concentration (potency) = 4% (moderate) pKa (onset of action) = 7.8 (fast) Protein binding (duration) = 66% (moderate)		
Bupivacaine	Amide = metabolized in liver	Concentration (potency) = 0.5% (high; more lipophilic); most potent pKa (onset of action) = 8.1 (moderate); least effective Protein binding (duration) = 95% (long)	<u>Vasodilatory potential</u>: Procaine Bupivacaine Lidocaine Articaine Prilocaine Mepivacaine Cocaine (vasoconstrictor)	
Lidocaine	Amide = metabolized in liver	Concentration (potency) = 2% (moderate) pKa (onset of action) = 7.8 (fast) Protein binding (duration) = 70% (moderate)		
Aspirin **Ibuprofen**	Non-steroidal anti-inflammatory drugs (NSAIDs)	Target = cyclooxygenase inhibition - COX1 = GI tract, kidney - COX2 = induced by inflammation (*main target*) Decreased prostaglandins = decreased vasodilation, thermal set point,	**Anti-inflammatory** **Anti-pyretic** **Analgesics** **Anti-platelet** Acute pain, mild to moderate post-procedural pain, TMJ disorders	***Side effect:*** **GI bleeding** (dose-dependent) *Look at interaction with B-blockers, diuretics, ACE inhibitors/AT1 antagonist, Warfarin*

		neuroinflammation/ sensitization of nociceptive endings, thromboxane A2 (decreased platelet aggregation)		
Acetaminophen	*Not* an NSAID	Unknown	**Antipyretic analgesic** of choice when NSAIDs cannot be used due to contraindications Children Post-operative dental pain (less likelihood of inflammation)	Few side effects compared to NSAIDs, but **toxic effects** (occurs when recommended dose is exceeded) = - Overdose = **liver damage** - Alcohol = increased toxic metabolite due to CYP2E1 induction by alcohol Weak/no anti-inflammatory or anti-platelet properties