

Cardiovascular Drugs: OPRA Review

1. Antihypertensives

Mechanism of Action (MOA):

- **ACE Inhibitors** (e.g., Enalapril, Lisinopril): Inhibit angiotensin-converting enzyme (ACE), reducing angiotensin II formation, leading to vasodilation and decreased aldosterone secretion.
- **ARBs** (e.g., Losartan, Valsartan): Block angiotensin II receptors, preventing vasoconstriction and aldosterone effects.
- **Calcium Channel Blockers** (e.g., Amlodipine, Diltiazem): Block calcium entry into vascular smooth muscle and myocardium, causing vasodilation and reduced contractility.
- **Beta-Blockers** (e.g., Metoprolol, Propranolol): Block beta-adrenergic receptors, reducing heart rate and myocardial contractility.
- **Diuretics:**
 - **Thiazide Diuretics** (e.g., Hydrochlorothiazide, Chlorthalidone): Inhibit sodium reabsorption in the distal tubules, leading to increased sodium and water excretion.
 - **Loop Diuretics** (e.g., Furosemide, Bumetanide): Inhibit sodium-potassium-chloride cotransporter in the ascending loop of Henle, leading to potent diuresis.
 - **Potassium-Sparing Diuretics** (e.g., Spironolactone, Eplerenone): Antagonize aldosterone in the distal tubules, reducing sodium retention and potassium excretion.

- **Carbonic Anhydrase Inhibitors (e.g., Acetazolamide):**
Inhibit carbonic anhydrase, reducing bicarbonate reabsorption and causing mild diuresis.
- **Osmotic Diuretics (e.g., Mannitol):** Increase osmotic pressure in the renal tubules, preventing water reabsorption.

Important Side Effects (SE):

- **ACE Inhibitors:** Dry cough, angioedema, hyperkalemia, hypotension.
- **ARBs:** Hyperkalemia, hypotension, dizziness.
- **CCBs:** Peripheral edema, headache, flushing, constipation (with non-DHP CCBs like Verapamil).
- **Beta-Blockers:** Bradycardia, fatigue, bronchospasm (in non-selective agents), masking of hypoglycemia.
- **Diuretics:**
 - **Thiazide Diuretics:** Hypokalemia, hyperuricemia, hyperglycemia, dehydration.
 - **Loop Diuretics:** Severe hypokalemia, ototoxicity, dehydration, metabolic alkalosis.
 - **Potassium-Sparing Diuretics:** Hyperkalemia, gynecomastia (spironolactone), metabolic acidosis.
 - **Carbonic Anhydrase Inhibitors:** Metabolic acidosis, hypokalemia, paresthesia.
 - **Osmotic Diuretics:** Fluid imbalance, hypernatremia, risk of pulmonary edema.

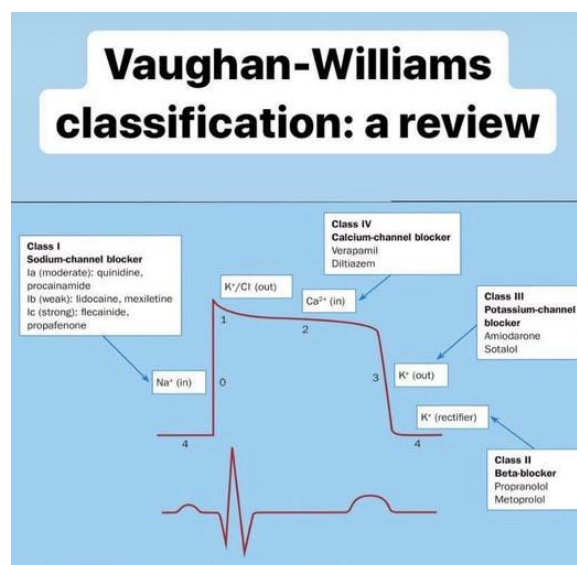
Key Notes:

- ACE inhibitors and ARBs should not be used together.
 - Beta-blockers are contraindicated in acute heart failure and severe asthma.
 - Thiazide diuretics are first-line for hypertension but can cause electrolyte imbalances.
 - Loop diuretics are preferred for heart failure with fluid overload.
 - Potassium-sparing diuretics should be avoided in patients with renal impairment due to the risk of hyperkalemia.
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2. Antiarrhythmics (Vaughan-Williams Classification)

Mechanism of Action (MOA):

- **Class I (Na⁺ Channel Blockers):**
 - IA (e.g., Quinidine, Procainamide): Moderate Na⁺ blockade, prolongs repolarization.
 - IB (e.g., Lidocaine, Mexiletine): Weak Na⁺ blockade, shortens repolarization.
 - IC (e.g., Flecainide, Propafenone): Strong Na⁺ blockade, no effect on repolarization.
- **Class II (Beta-Blockers) (e.g., Metoprolol, Esmolol):** Reduce sympathetic stimulation, decrease heart rate and conduction velocity.
- **Class III (K⁺ Channel Blockers) (e.g., Amiodarone, Sotalol):** Prolong repolarization, increase action potential duration.
- **Class IV (CCBs) (e.g., Verapamil, Diltiazem):** Inhibit calcium channels, reducing AV node conduction.



Important Side Effects (SE):

- **Class I:** Proarrhythmic effects, QT prolongation (IA), CNS effects (IB), severe arrhythmias (IC).
- **Class II:** Bradycardia, hypotension, fatigue.
- **Class III:** Torsades de pointes (especially with Sotalol), pulmonary fibrosis, thyroid dysfunction (Amiodarone).
- **Class IV:** Bradycardia, hypotension, constipation.

Key Notes:

- Amiodarone requires monitoring of thyroid, liver, and pulmonary function.
 - Beta-blockers and CCBs should be used cautiously in heart failure.
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3. Anticoagulants & Antiplatelets

Mechanism of Action (MOA):

- **Anticoagulants:**

- **Heparin & LMWH (e.g., Enoxaparin):** Enhance antithrombin III, inhibiting thrombin and factor Xa.
- **Warfarin:** Inhibits vitamin K-dependent clotting factors (II, VII, IX, X).
- **DOACs (e.g., Apixaban, Rivaroxaban):** Directly inhibit factor Xa or thrombin (Dabigatran).

- **Antiplatelets:**

- **Aspirin:** Inhibits COX-1, reducing thromboxane A2 production.
- **P2Y12 Inhibitors (e.g., Clopidogrel, Ticagrelor):** Block ADP receptors on platelets, preventing aggregation.

Important Side Effects (SE):

- **Heparin:** Bleeding, heparin-induced thrombocytopenia (HIT).
- **Warfarin:** Bleeding, teratogenicity, requires INR monitoring.
- **DOACs:** Lower risk of bleeding than warfarin but still significant.
- **Aspirin:** GI ulcers, bleeding risk.
- **P2Y12 Inhibitors:** Bleeding, rare TTP (Clopidogrel).

Key Notes:

- Warfarin has many drug interactions; INR monitoring is essential.
 - DOACs have a more predictable effect but are costly.
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