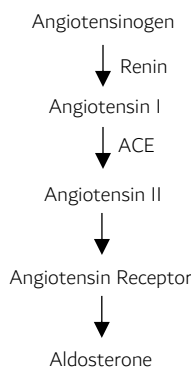


Renin-Angiotensin-Aldosterone System



ACE Inhibitors

I. Drugs

- A. Benazepril
- B. Captopril
- C. Enalapril
- D. Fosinopril
- E. Lisinopril
- F. Moexipril
- G. Quinapril
- H. Perindopril
- I. Ramipril
- J. Trandolapril

II. Mechanism: Inhibits angiotensin converting enzyme

- A. ↓Angiotensin II (↓Preload/Afterload)
- B. ↓Angiotensin II (↓Na/H₂O Retention)
- C. ↑Bradykinin (Vasodilation)

III. Applications

- A. Hypertension
- B. Heart Failure
- C. Diabetic Nephropathy
- D. Post-MI

IV. Adverse Effects

- A. Angioedema, cough
- B. Teratogenic
- C. Hyperkalemia

Angiotensin II Receptor Blockers

I. Drugs

- A. Azilsartan
- B. Candesartan
- C. Irbesartan
- D. Losartan
- E. Olmesartan
- F. Telmisartan
- G. Valsartan

II. MOA/Activity: Blocks angiotensin II receptors

III. Applications

- A. Hypertension
- B. Heart Failure

IV. Adverse Effects:

- A. Less angioedema
- B. Teratogenic
- C. Hyperkalemia

Aldosterone Antagonists

I. Drugs

- A. Eplerenone
- B. Spironolactone

II. MOA/Activity: Inactivates spironolactone receptor complex, decreases Na⁺ reabsorption.

III. Applications

- A. Diuretic
- B. Hypertension
- C. Heart Failure
- D. PCOS
- E. Secondary Hyperaldosteronism (cirrhosis/nephrotic syndrome)

IV. Adverse Effects:

- A. Gynecomastia
- B. Menstrual Irregularities
- C. Hyperkalemia

Renin Inhibitors

I. Drugs

- A. Aliskiren

II. MOA/Activity: Inhibits Renin

III. Applications

- A. Hypertension

IV. Adverse Effects:

- A. Decreased angioedema
- B. Teratogenic
- C. Hyperkalemia

Dihydropyridine Calcium Channel Blockers

I. Drugs

- A. Amlodipine
- B. Felodipine
- C. Isradipine
- D. Nicardipine
- E. Nifedipine
- F. Nisoldipine

II. MOA/Activity: Blocks L-type calcium channels

- A. Relaxation of smooth muscle of arterioles

III. Applications

- A. Hypertension
- B. Effort & vasospastic induced angina

IV. Adverse Effects:

- A. AV Node Block
- B. Peripheral Edema

Non-dihydropyridine Calcium Channel Blockers

I. Drugs

- A. Diltiazem
- B. Verapamil

II. MOA/Activity: Binds/blocks L-type calcium channels

- A. More negative inotropic/chronotropic effects than DHP CCBs

III. Applications

- A. Hypertension
- B. Effort & Vasospastic Angina

IV. Adverse Effects

- A. AV Block
- B. Peripheral Edema

Thiazide Diuretics

I. Drugs

- A. Chlorothiazide
- B. Chlorthalidone
- C. Hydrochlorothiazide
- D. Indapamide
- E. Metolazone

II. MOA/Activity:

- A. Inhibits Na/Cl Cotransporter
- B. Location: LOH and DCT

III. Applications:

- A. Hypertension
- B. Heart Failure
- C. Hypercalciuria
- D. Diabetes Insipidus

IV. Adverse Effects:

- A. Hypokalemia & Hyponatremia
- B. Hyperuricemia & Hypercalcemia
- C. Derived from sulfites.

Loop Diuretics

I. Drugs

- A. Bumetanide
- B. Ethacrynic Acid
- C. Furosemide
- D. Torsemide

II. MOA/Activity:

- A. Inhibits Na/K/2Cl cotransporter.
- B. Location: Ascending limb of LOH

III. Applications:

- A. Hypertension
- B. Heart Failure/Pulmonary Edema
- C. Hypercalcemia
- D. Hyperkalemia

IV. Adverse Effects:

- A. Ototoxicity
- B. Hyperuricemia
- C. Hypokalemia
- D. Sulfa (except ethacrynic acid)

Vasodilators

I. Drugs

- A. Hydralazine
- B. Minoxidil

II. MOA/Activity: Relaxes vascular smooth muscle.

III. Applications:

- A. Hypertension

IV. Adverse Effects:

- A. Reflex tachycardia

Sodium Transport Channel Blockers

I. Drugs

- A. Amiloride
- B. Triamterene

II. MOA/Activity:

- A. Blocks Na transport channel
- B. Location: DCT & Cortical collecting ducts

III. Applications:

- A. Hypertension
- B. Heart Failure

IV. Adverse Effects:

- A. Hyperuricemia
- B. Kidney Stones
- C. Hyperkalemia

Carbonic Anhydrase Inhibitors

I. Drugs

- A. Acetazolamide

II. MOA/Activity:

- A. Inhibits carbonic anhydrase.
 1. Loss of bicarbonate in urine
 2. Hyperchloremic metabolic acidosis
- B. Decreases aqueous humor production.

III. Applications:

- A. Altitude sickness
- B. Glaucoma

IV. Adverse Effects:

- A. Metabolic Acidosis
- B. Hypokalemia
- C. Nephrolithiasis
- D. Avoid in hepatic failure (decreased ammonia excretion)

Osmotic Diuretics

I. Drugs

- A. Mannitol

II. MOA/Activity:

- A. Hydrophilic structure attracts water intravascularly and into renal tubules.

III. Applications

- A. Maintains urine flow in toxic ingestions.
- B. Intracranial hypertension

IV. Adverse Effects

- A. Hypo/Hyponatremia

Digoxin

I. Drugs

- A. Digoxin

II. MOA/Activity:

- A. Inhibits Na/K-adenosine ATPase.
- B. Increases vagal tone.
- C. Neurohormonal inhibition

III. Applications:

- A. Reduced Ejection Fraction Heart Failure
- B. Symptomatic Afib/Aflutter

IV. Adverse Effects:

- A. Narrow therapeutic index
- B. Yellow/Blurred Vision
- C. Cardiac arrhythmias
- D. Heart block in combination with AV Blockers
- E. Toxicity Tx: Digoxin Fab & K⁺ repletion

Phosphodiesterase Inhibitors

I. Drugs

- A. Milrinone

II. MOA/Activity:

- A. Inhibits phosphodiesterase (↑cAMP)
- B. ↑cAMP causes increased contractility

III. Class Applications

- A. Heart Failure

IV. Adverse Effects:

- A. Long-term use increases mortality
- B. Thrombocytopenia

Class IA Antiarrhythmics

I. Drugs

- A. Quinidine
- B. Procainamide
- C. Disopyramide

II. MOA/Activity

- A. Blocks voltage sensitive sodium channels
 - 1. Slows Phase 0
- B. Potassium Channel Inhibition
 - 1. Slows phase 3 & ↑ERP.
- C. Calcium Channel Blocker
- D. Anticholinergic

III. Application:

- A. Treatment of tachy-arrhythmias (atrial, AV junctional, Ventricular)

IV. Adverse Effects:

- A. Anticholinergic

Class IB Antiarrhythmics

I. Drugs

- A. Lidocaine
- B. Mexiletine

II. MOA/Activity

- A. Sodium Channel Blocker
 - 1. Associates/Dissociates rapidly.
 - 2. Slows Phase 0 (less than Class IA)
- B. Late Sodium Channel Blockade
 - 1. Shortens Phase 3 Repolarization
- C. Decreases Action Potential Duration

III. Applications:

- A. Ventricular Arrhythmias
- B. Lidocaine: Local Anesthesia

IV. Adverse Effects:

- A. Lidocaine: Nystagmus, slurred speech, paresthesias, agitation, convulsions

Class IC Antiarrhythmics

I. Drugs

- A. Flecainide
- B. Propafenone

II. MOA/Activity

- A. Sodium Channel Blockade
 - 1. Slows Phase 0 – Strong Effect

III. Application:

- A. Conversion/Maintenance of Sinus Rhythm in AFib/Flutter
- B. Ventricular Arrhythmias

IV. Adverse Effects:

- A. Contraindicated in structural heart disease.
- B. Blurry Vision

Class III Antiarrhythmics

I. Drugs

- A. Amiodarone
- B. Dronedarone
- C. Sotalol
- D. Dofetilide
- E. Ibutilide

II. MOA/Activity

- A. Potassium Channel Blocker
 - 1. Prolongs Phase III Repolarization
 - 2. Increases Refractory Period
- B. Amiodarone/Dronedarone: Also Class I, II, and IV Effects
- C. Sotalol: Non-selective Beta-Blocker
- D. Ibutilide: Class IA Effects

III. Class Applications

- A. Arrhythmias

IV. Adverse Effects:

- A. Amiodarone
 - 1. Pulmonary Fibrosis
 - 2. Hepatotoxicity
 - 3. Thyroid Toxicity
 - 4. Optic Neuritis/Corneal Deposits
 - 5. Blue-Gray Skin Discoloration
- B. Sotalol
 - 1. Prolonged QT
- C. Sotalol/Dofetilide/Ibutilide: Initiated in hospital due to pro-arrhythmic properties.

Adenosine

I. Drugs:

- A. Adenosine

II. MOA/Activity: Activates adenosine 1 receptor (A1)

- A. Decreases conduction velocity.
- B. Prolongs refractory period.
- C. Slows AV node.

III. Class Application:

- A. Acute supraventricular tachycardia
- B. Cardiac Stress Testing

IV. Adverse Effects:

- A. Hypotension
- B. Flushing
- C. Chest Pain

Magnesium Sulfate

I. Drugs

- A. Magnesium Sulfate

II. MOA/Activity

- A. Slows SA node.
- B. Decreases conduction velocity.
- C. Cerebral vasodilation (eclampsia)

III. Class Application:

- A. Torsades de pointes
- B. Digoxin induced arrhythmias.
- C. Pre-eclampsia/Eclampsia

IV. Adverse Effects:

- A. Loss of reflexes
- B. Respiratory depression

Organic Nitrates

I. Drugs

- A. Isosorbide dinitrate
- B. Isosorbide mononitrate
- C. Nitroglycerin

II. MOA/Activity:

- A. Relaxes vascular smooth muscle.
- B. NO → cGMP
- C. Reduces preload.
- D. Increases oxygen supply to cardiac tissue.

III. Applications:

- A. Angina

IV. Adverse Effects:

- A. Headache
- B. Hypotension (caution with phosphodiesterase inhibitors)

Ranolazine

I. Drugs

- A. Ranolazine

II. MOA/Activity:

- A. Sodium channel blocker
- B. Decreases intracellular Na & Ca, improving relaxation in diastole.

III. Class Applications

- A. Angina

IV. Adverse Effects:

- A. QT prolongation
- B. Drug interactions

Aspirin

I. Drugs

- A. Aspirin

II. MOA/Activity:

- A. Irreversible COX-1 Inhibitor
- B. Reduces thromboxane A2 production.

III. Class Application:

- A. Treatment/Prophylaxis of ACS
- B. Stroke prophylaxis

IV. Adverse Effects:

- A. Bleeding
- B. Reye's Syndrome

P2Y₁₂ ADP Receptor Inhibitors

I. Drugs

- A. Ticlopidine
- B. Clopidogrel
- C. Prasugrel
- D. Ticagrelor

II. MOA/Activity:

- A. Inhibits binding of ADP to P2Y₁₂ ADP receptors on platelets

III. Applications

- A. Treatment/Prevention of stroke
- B. Treatment/Prevention of ACS

IV. Adverse Effects

- A. Bleeding
- B. Clopidogrel:
 1. Neutropenia/TTP
 2. Genetic variances prevent activation.
- C. Prasugrel
 1. TTP
- D. Ticlopidine
 1. TTP/Agranulocytosis/Aplastic Anemia

Glycoprotein IIa/IIIb Receptor Inhibitors

I. Drugs

- A. Apiximab
- B. Eptifibatide
- C. Tirofiban

II. MOA/Effects:

- A. Blocks GIIb/IIIa receptors
- B. Inhibits binding to fibrinogen and von Willebrand factor.

III. Applications:

- A. Prevents ischemic complications in PCI.

IV. Adverse Effects

- A. Bleeding

Cyclic Nucleotide Phosphodiesterase Inhibitors

I. Drugs

- A. Dipyridamole

II. MOA/Activity:

- A. Inhibits cyclic nucleotide phosphodiesterase.
- B. Increases cAMP levels, decreases thromboxane A2, lowering platelet adhesion.

III. Application

- A. Stroke prophylaxis

IV. Adverse Effects

- A. Bleeding
- B. Coronary steal phenomenon

Phosphodiesterase Type III Inhibitors

I. Drugs

- A. Cilostazol

II. MOA/Effects:

- A. Inhibits phosphodiesterase type III.
- B. Increases cAMP levels, decreases thromboxane A2, lowering platelet adhesion.

III. Applications:

- A. Intermittent claudication

IV. Adverse Effects

- A. Bleeding
- B. Increased mortality in heart failure

Heparin & Low Molecular Weight Heparins

I. Drugs

- A. Heparin
- B. LMWH:
 1. Enoxaparin
 2. Dalteparin
 3. Tinzaparin

II. MOA/Activity

- A. All: Binds antithrombin III, potentiates inactivation of factor Xa
- B. Heparin only: Potentiates inhibition of thrombin.

III. Class Application:

- A. DVT/PE Treatment & Prophylaxis
- B. Acute MI
- C. Drug of choice in pregnancy (doesn't cross placenta)

IV. Adverse Effects:

- A. Bleeding
 1. Heparin Reversal Agent: Protamine
- B. Heparin-induced thrombocytopenia (HIT)
- C. Hypersensitivity

Fondaparinux

I. Drugs

- A. Fondaparinux

II. MOA/Activity

- A. Binds antithrombin III, potentiates inactivation of factor Xa.

III. Class Application:

- A. DVT/PE Treatment & Prophylaxis

IV. Adverse Effects:

- A. Bleeding, no reversal agent
- B. HIT unlikely.
- C. Avoid in renal failure.

Parenteral Direct Thrombin Inhibitors

I. Drugs

- A. Argatroban
- B. Bivalirudin
- C. Desirudin

II. MOA/Activity

- A. Direct Thrombin Inhibitor

III. Class Application:

- A. DVT/PE Treatment & Prophylaxis in HIT
- B. PCI at risk for HIT

IV. Adverse Effects:

- A. Bleeding, no reversal agent

Oral Direct Thrombin Inhibitors

I. Drugs

- A. Dabigatran

II. MOA/Activity:

- A. Direct Thrombin Inhibitor

III. Class Application:

- A. Stroke prophylaxis in atrial fibrillation

IV. Adverse Effects:

- A. Increased clotting with abrupt discontinuation
- B. Idarucizumab, reversal agent
- C. Esophagitis

Factor Xa Inhibitors

I. Drugs

- A. Rivaroxaban
- B. Apixaban

II. MOA/Activity:

- A. Inhibits factor Xa
- B. Decreases conversion of prothrombin to thrombin

III. Applications

- A. Stroke prophylaxis in afib
- B. DVT/PE Treatment/Prophylaxis

IV. Adverse Effects:

- A. Bleeding, no reversal agent
- B. Abrupt discontinuation can lead to thrombosis.

Vitamin K Antagonists

I. Drugs

- A. Coumadin

II. MOA/Activity:

- A. Inhibits vitamin K epoxide reductase.
- B. ↓ production of factors II, VII, IX, and X

III. Applications

- A. Stroke prophylaxis in afib, protein C and S deficiency, prosthetic heart valves
- B. DVT/PE Treatment/Prophylaxis

IV. Adverse Effects

- A. Drug/Drug Interactions
- B. Bleeding
 - 1. Reversal: Vitamin K, FFP, Concentrated factor replacement
- C. Purple toe syndrome
- D. Skin necrosis/Skin lesions
- E. Hypercoagulation on initiation
- F. Teratogenic

Thrombolytics

I. Drugs

- A. Alteplase
- B. Reteplase
- C. Tenecteplase
- D. Streptokinase
- E. Urokinase

II. MOA/Activity:

- A. Converts plasminogen to plasmin.
- B. Plasmin cleaves thrombi.

III. Applications

- A. Acute MI
- B. Acute ischemic stroke
- C. Massive pulmonary embolism

IV. Adverse Effects

- A. Bleeding
- B. Contraindications
 - 1. History of intracranial hemorrhage
 - 2. Recent stroke
 - 3. Head trauma
 - 4. Brain tumor
 - 5. Metastatic Cancer

Procoagulants & Reversal Agents

I. Drugs

- A. Aminocaproic Acid

1. MOA: Inhibits plasminogen activation
2. Applications: Bleeding
3. Adverse Effects: Theoretical thrombosis

- B. Tranexamic Acid

1. MOA: Inhibits plasminogen activation
2. Applications: Bleeding
3. Adverse Effects: Theoretical thrombosis

- C. Protamine Sulfate

1. MOA: Forms complex with heparin
2. Applications: Heparin reversal
3. Adverse Effects:
 - a) Hypersensitivity
 - b) Bradycardia
 - c) Hypotension/Flushing
 - d) Bleeding in excess

- D. Vitamin K

1. MOA: Repletes Vit K, coumadin reversal
2. Applications: Bleeding associated with coumadin
3. Adverse Effects:
 - a) Hypersensitivity
 - b) Anaphylactoid reaction

- E. Idarucizumab

1. MOA: Binds to dabigatran
2. Applications: Bleeding related to dabigatran use

Statins

I. Drugs

- A. Atorvastatin
- B. Fluvastatin
- C. Lovastatin
- D. Pitavastatin
- E. Pravastatin
- F. Simvastatin

II. MOA/Activity:

- A. HMG CoA Reductase Inhibitor
- B. Reduces intracellular cholesterol (increasing uptake from plasma)

III. Applications

- A. Hyperlipidemia

IV. Adverse Effects

- A. Elevated liver enzymes
- B. Rhabdomyolysis
- C. Myalgias
- D. Contraindicated in pregnancy/breastfeeding.

Niacin

I. Drugs

- A. Niacin

II. MOA/Activity:

- A. Inhibits lipolysis.
- B. ↓ FFA levels, ↓ cholesterol production

III. Applications

- A. Hyperlipidemia

IV. Adverse Effects

- A. Flushing, reduced by ASA or slow release.
- B. Hepatotoxicity
- C. Hyperuricemia/Gout
- D. Pruritus

Fibrates

I. Drugs

- A. Fenofibrate
- B. Gemfibrozil

II. MOA/Activity:

- A. Activates PPARs
- 1. ↑HDL
- 2. ↓triglycerides, ↓LDL

III. Applications

- A. Hypertriglyceridemia

IV. Adverse Effects

- A. Gallstones
- B. Myositis/Rhabdomyolysis risk

Bile Acid Sequestrants

I. Drugs

- A. Cholestyramine
- B. Colesevelam

II. MOA/Activity:

- A. Binds bile acids/bile salts.
- B. Increases production of bile acids, which are formed from cholesterol.

III. Applications

- A. Hyperlipidemia

IV. Adverse Effects

- A. Reduced absorption of Vit D, E, A, K
- B. Drug absorption interference
- C. ↑Triglycerides

Selective Cholesterol Absorption Inhibitors

I. Drugs

- A. Ezetimibe

II. MOA/Activity:

- A. Decreases cholesterol absorption in gut.

III. Applications

- A. Hyperlipidemia

IV. Adverse Effects

- A. Do not use in hepatic insufficiency.