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PHARMACOLOGY - II

UNIT 2

TOPIC :

- **Pharmacology of drugs acting on urinary system**
  a. Diuretics
  b. Anti-diuretics.

Drugs Acting on the Urinary System

→ The urinary system (kidneys, ureters, bladder, urethra) maintains fluid and electrolyte balance, acid-base homeostasis, blood pressure, and excretion of metabolic wastes. Drugs acting on the urinary system can modify urine volume, composition, and bladder function.

Diuretics

→ Diuretics are drugs that increase urine output by acting on the kidneys. They are used to remove excess salt and water, treat edema, hypertension, and certain renal and cardiac disorders.

→ Diuretics are agents that increase excretion of water and electrolytes (Na^+, Cl^-, K^+) from the body by acting on the renal tubules.

Functions of Diuretics

- Increase urine volume (diuresis)
- Reduce blood volume → lower blood pressure
- Reduce edema associated with heart, liver, and kidney disease
- Alter electrolyte balance (Na^+, K^+, Ca^{2+}, Mg^{2+})

Classification of Diuretics

Diuretics are classified based on site of action in nephron:

Class	Site of Action	Examples	Major Effects
1. Carbonic anhydrase inhibitors (CAIs)	Proximal convoluted tubule	Acetazolamide, Methazolamide	↑ HCO_3^- excretion, mild diuresis
2. Loop diuretics (High ceiling)	Thick ascending loop of Henle	Furosemide, Bumetanide, Torsemide	↑ Na^+, K^+, Cl^- excretion, potent diuresis
3. Thiazide diuretics	Distal convoluted tubule	Hydrochlorothiazide, Chlorthalidone	↑ Na^+, Cl^- excretion, mild diuresis
4. Potassium-sparing diuretics	Collecting ducts / Distal tubule	Spironolactone, Amiloride, Triamterene	↑ Na^+ excretion, ↓ K^+ loss
5. Osmotic diuretics	Proximal tubule & descending loop	Mannitol, Urea	↑ water excretion, ↑ urine osmolarity

A. Carbonic Anhydrase Inhibitors (CAIs)

Examples: Acetazolamide, Methazolamide

Mechanism of Action:

- Inhibit carbonic anhydrase → ↓ H^+ secretion → ↓ $NaHCO_3$ reabsorption → ↑ bicarbonate, Na^+, water excretion.

Uses:

- Glaucoma (↓ aqueous humor)
- Metabolic alkalosis
- Altitude sickness (prevention of acute mountain sickness)

Adverse Effects:

- Metabolic acidosis
- Hypokalemia
- Paresthesia
- Kidney stones (calcium phosphate precipitation)

B. Loop Diuretics

Examples: Furosemide, Bumetanide, Torsemide

Mechanism of Action:

- Inhibit Na^+-K^+-2Cl^- symporter in thick ascending loop of Henle → ↑ Na^+, Cl^-, K^+, Ca^{2+} excretion.

Effects:

- Potent diuresis
- ↓ blood pressure
- ↓ edema

Uses:

- Pulmonary edema (heart failure)
- Ascites (liver cirrhosis)
- Hypertension (resistant cases)
- Hypercalcemia

Adverse Effects:

- Hypokalemia, hyponatremia, hypomagnesemia
- Ototoxicity (hearing loss)
- Hyperuricemia (gout risk)
- Dehydration, hypotension

C. Thiazide Diuretics

Examples: Hydrochlorothiazide, Chlorthalidone, Indapamide

Mechanism of Action:

- Inhibit Na^+-Cl^- symporter in distal convoluted tubule → mild Na^+ and water excretion.

Effects:

- Moderate diuresis
- ↑ Ca^{2+} reabsorption → may prevent kidney stones

Uses:

- Hypertension (first-line therapy)
- Mild edema
- Nephrolithiasis (prevent calcium stones)

Adverse Effects:

- Hypokalemia, hyponatremia
- Hyperglycemia, hyperuricemia
- Hyperlipidemia
- Erectile dysfunction (rare)

D. Potassium-Sparing Diuretics

Examples:

1. Aldosterone antagonists: Spironolactone, Eplerenone
2. Epithelial sodium channel blockers: Amiloride, Triamterene

Mechanism of Action:

- Spironolactone: Blocks aldosterone → ↓ Na^+ reabsorption, ↑ K^+ retention
- Amiloride/Triamterene: Directly block Na^+ channels in collecting ducts → ↑ Na^+ excretion, ↓ K^+ loss

Uses:

- Prevent hypokalemia caused by other diuretics
- Heart failure (Spironolactone)
- Hyperaldosteronism

Adverse Effects:

- Hyperkalemia (risk in renal failure)
- Gynecomastia (Spironolactone)

- GI disturbances

E. Osmotic Diuretics

Examples: Mannitol, Urea

Mechanism of Action:

- Freely filtered at glomerulus → ↑ osmotic pressure in tubular lumen → water retention in tubule → ↑ urine volume

Effects:

- Rapid diuresis
- ↓ intracranial pressure (ICP)
- ↓ intraocular pressure (IOP)

Uses:

- Cerebral edema
- Acute glaucoma
- Oliguric renal failure (prevention)

Adverse Effects:

- Dehydration, electrolyte imbalance
- Pulmonary edema (fluid overload)
- Headache, nausea

ANTI-DIURETICS (ADH Agonists / Water Retention Agents)

→ Anti-diuretics are drugs that reduce urine output by increasing water reabsorption in the kidneys.

→ They are mainly used to treat conditions associated with excessive urination (polyuria) and dehydration, such as diabetes insipidus or hypotension due to volume depletion.

→ Anti-diuretics are agents that decrease urine volume by enhancing water reabsorption in the renal collecting ducts, often by mimicking or potentiating the action of antidiuretic hormone (ADH, vasopressin).

Physiology of Water Reabsorption

- ADH (Vasopressin): Hormone secreted by posterior pituitary.
- Acts on V_2 receptors in the collecting ducts → ↑ aquaporin-2 channels → ↑ water reabsorption → ↓ urine output.
- Also acts on V_1 receptors → vasoconstriction → ↑ blood pressure (at high doses).

Classification of Anti-Diuretics

Class	Examples	Mechanism / Site of Action	Uses
1. ADH (Vasopressin) and analogues	Vasopressin, Desmopressin (DDAVP), Terlipressin	V_2 receptor agonist → ↑ water reabsorption in collecting ducts; V_1 → vasoconstriction	Diabetes insipidus, bleeding esophageal varices, nocturnal enuresis
2. Drugs that potentiate endogenous ADH	Carbamazepine, Chlorpropamide	↑ ADH release or enhance renal response to ADH	Central diabetes insipidus, polyuria
3. Osmotic anti-diuretics (water retention via solute)	Glycerol (rare), Glucose (in specific conditions)	Indirectly ↑ water retention by creating osmotic gradient	Rare clinical use

A. Vasopressin (ADH)

Source: Posterior pituitary (natural) or synthetic.

Mechanism of Action:

- V_2 receptor agonist: ↑ water reabsorption in collecting ducts → ↓ urine output
- V_1 receptor agonist: ↑ vascular smooth muscle contraction → ↑ blood pressure

Uses:

- Central diabetes insipidus (deficient ADH)
- Management of shock (vasoconstrictive effect in vasodilatory shock)
- Esophageal varices (↓ portal pressure)
- Nocturnal enuresis

Adverse Effects:

- Water intoxication → hyponatremia
- Headache, nausea
- Vasoconstriction → ischemia (high dose)

B. Desmopressin (DDAVP)

Synthetic analogue of vasopressin with longer duration.

Mechanism:

- Selective V_2 receptor agonist → ↑ water reabsorption
- Minimal V_1 effects → less vasoconstriction

Uses:

- Central diabetes insipidus
- Nocturnal enuresis (children)
- Hemophilia A & von Willebrand disease (induces release of factor VIII and vWF)

Advantages over vasopressin:

- Oral, nasal, or parenteral administration
- Fewer cardiovascular side effects

C. Terlipressin

Synthetic vasopressin analogue

Mechanism:

- Prodrug → converted to lysine-vasopressin
- Acts mainly on V_1 receptors → vasoconstriction
- Mild V_2 effect

Uses:

- Variceal bleeding (reduces portal venous pressure)
- Septic shock (as vasopressor)

Adverse Effects:

- Hypertension, bradycardia, abdominal cramps