Diuretics

Revised by
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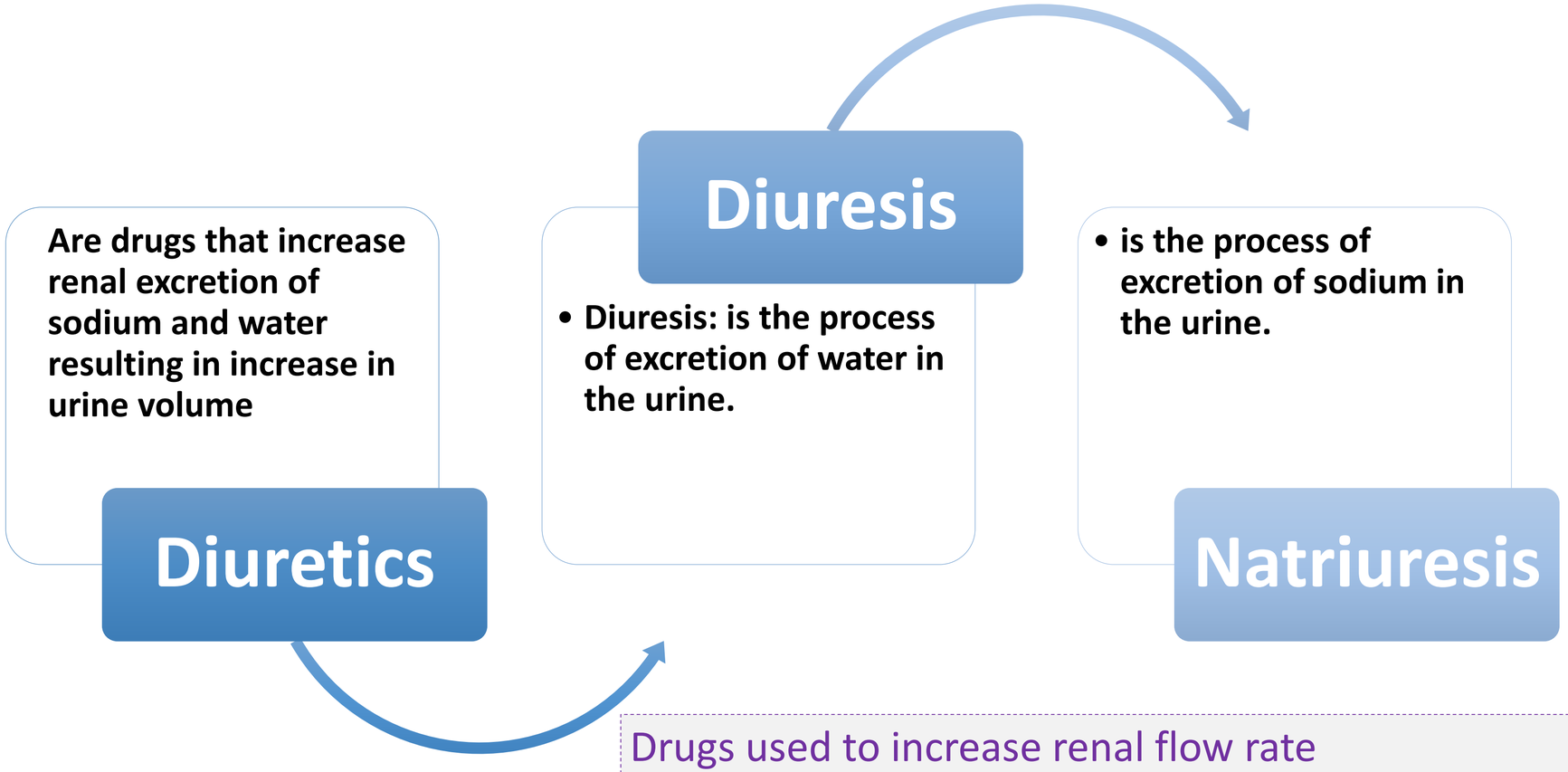
- Titles
- Boys' Slides
- Very important
- Extra information
- Doctor's notes

OBJECTIVES:

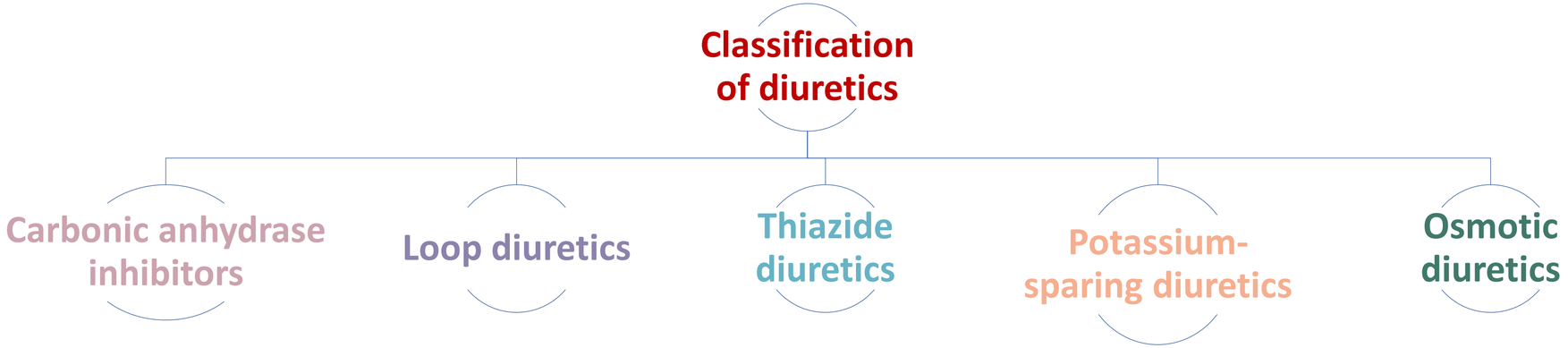
- Define and classify diuretics.
- Describe the mechanisms of action of diuretics.
- Identify the site of action of each class of diuretics in the nephron.
- Detail on the pharmacodynamic actions and pharmacokinetic aspects of diuretics.
- List ADRS, therapeutic uses, contraindications and drug-drug interactions of diuretics.

Diuretics



Drugs used to increase renal flow rate
Water diuresis
They act by ↑the quantity of sodium in urine (**natriuretic diuretics)**
Used to remove excess extracellular fluid (oedema)

Nephron Segment	Diuretics	Transporter mostly Na^+	Function	Filtered Na^+ re-absorbed
Proximal convoluted tubules	Carbonic anhydrase inhibitors	Na^+/H^+ transporter Carbonic anhydrase enzyme	Re-absorption of 100% glucose and amino acids, 66% Na, K, Ca, Mg; 85% $NaHCO_3$	85 % Na , HCO_3 65 % As $NaHCO_3$
Proximal Straight Tubules	-	Acid & base transporter	Secretion and re-absorption of organic acids and bases	-
Ascending Loop of Henle	Loop diuretics	$Na^+/K^+/2Cl^-$ co-transporter	Active reabsorption 25% Na, K, Cl Secondary re-absorption Ca, Mg	20-30% Active reabsorption Na, K, Cl Ca and Mag
Distal convoluted tubules	Thiazide diuretics ذا جاء **بزيد** (Thiazide) وهو أصلا جاي **متأخر** (**Distal** convoluted tubule)	Na^+/Cl^- co-transporter	Active tubular reabsorption of 5%Na, Cl, Ca	5-10% Active reabsorption Na, Cl
Cortical Collecting Tubules	K-sparing diuretics	Na^+ channel K & H transporter Aldosterone Antidiuretic hormone	Na reabsorption K & H secretion	5% Na reabsorption K & H secretion



Mechanism of actions of diuretics

Most diuretics act by interfering with the normal sodium reabsorption by the renal tubules resulting into sodium and water excretion.

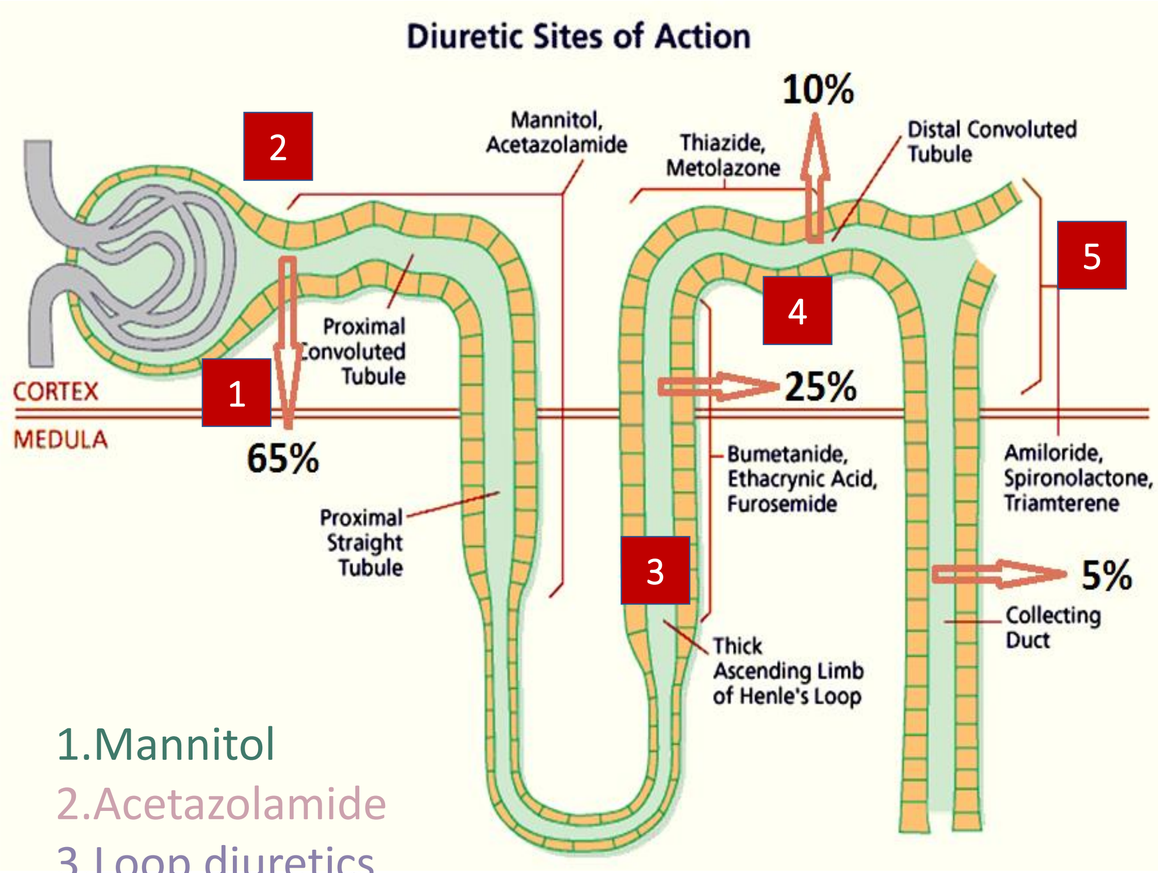
Sites of action for diuretics

How diuretics produce their effects?

Diuretics affect **carriers or transporters** in luminal membrane of renal tubular cells required for tubular reabsorption of sodium from filtrate back into blood.

https://www.youtube.com/watch?v=_7B8zIm8RBM

(7:23) Check this video اذا مالك خلق تحفظ



1.Mannitol
2.Acetazolamide
3.Loop diuretics
4.Thiazide
5.K sparing diuretics

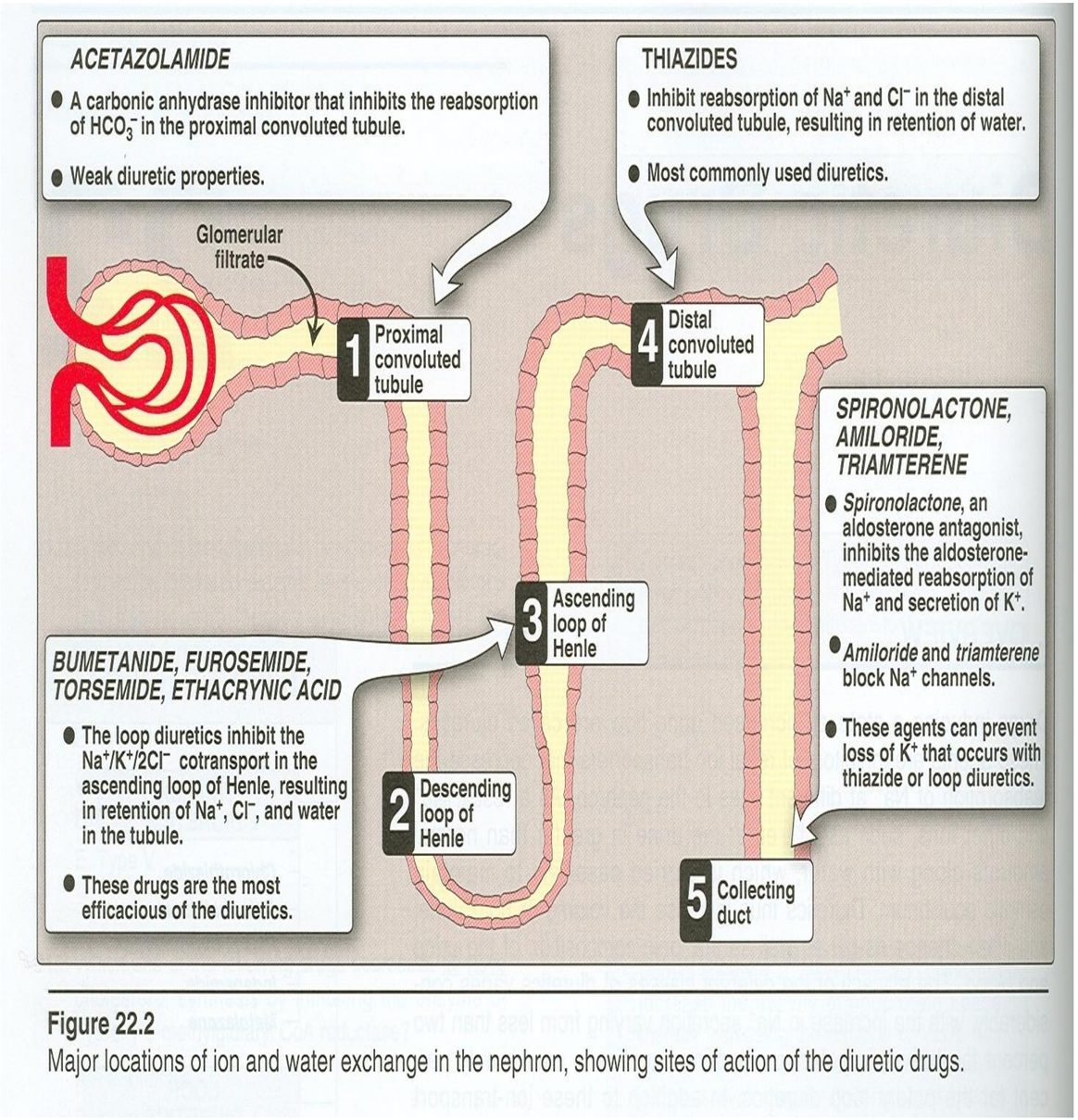
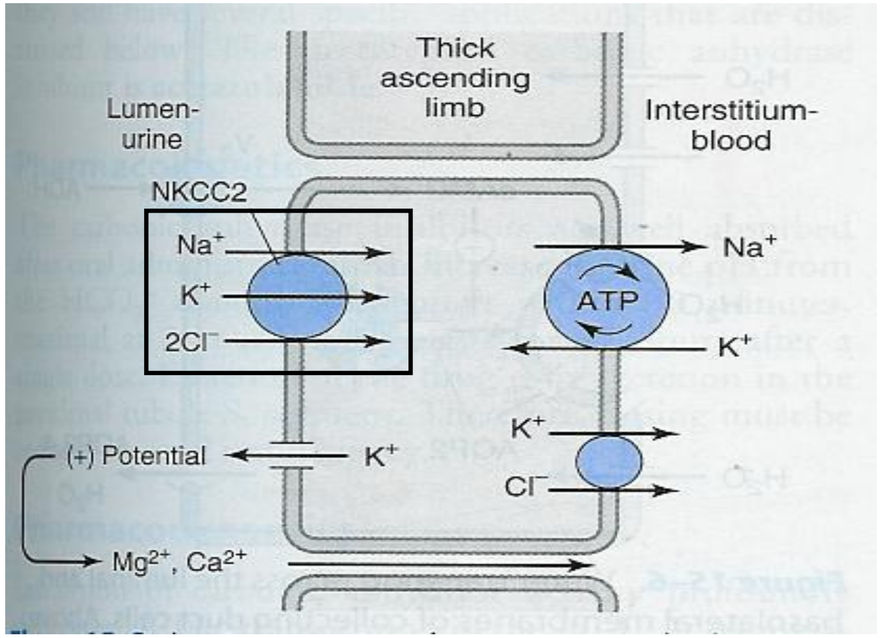
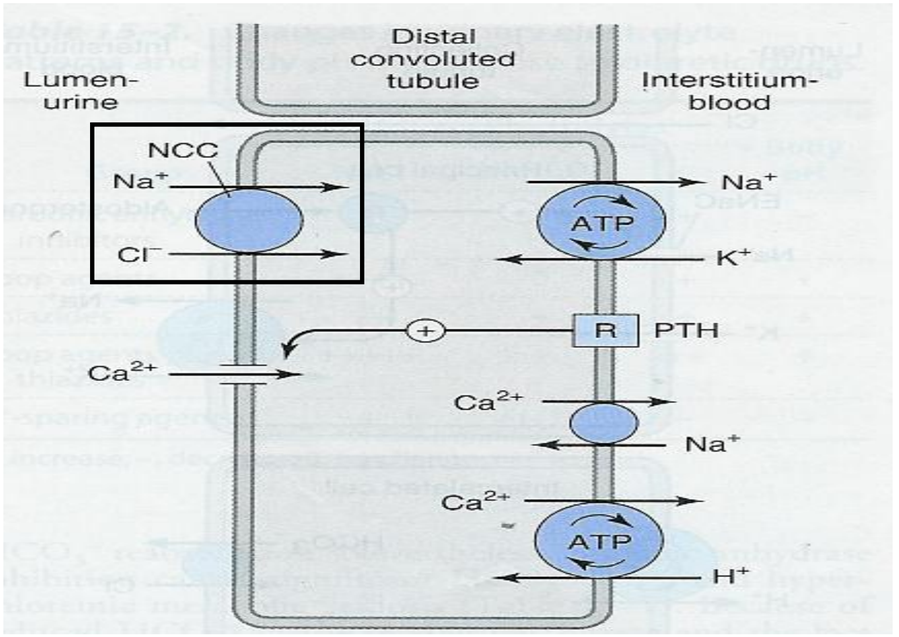


Figure 22.2
Major locations of ion and water exchange in the nephron, showing sites of action of the diuretic drugs.

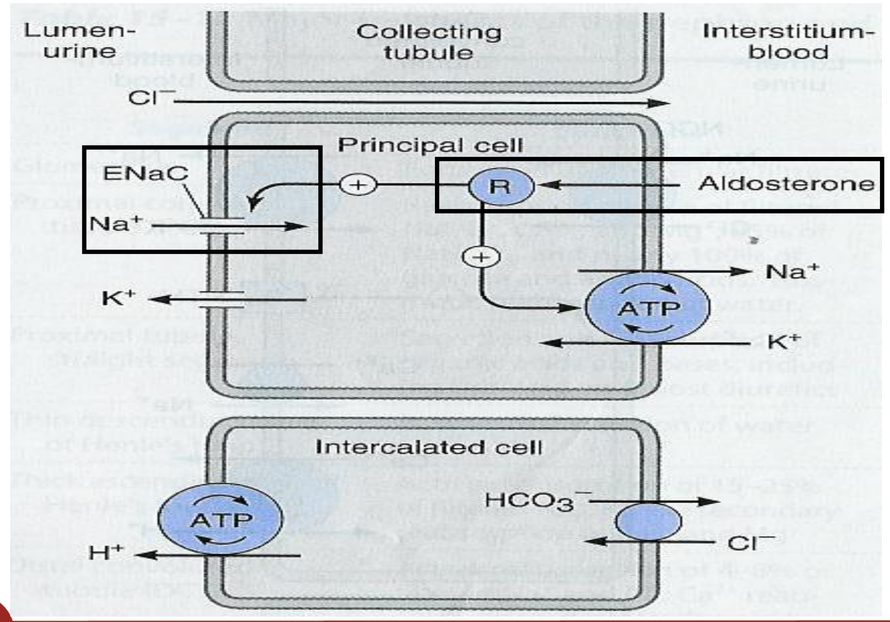
ASCENDING LOOP OF HENLE



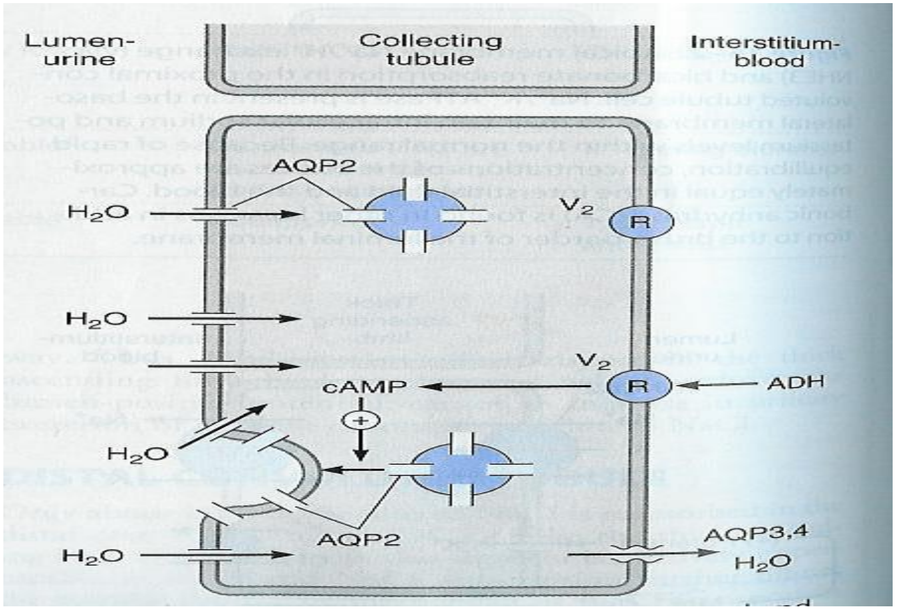
Distal convoluted tubules (DCT)



COLLECTING TUBULES (CT)



COLLECTING TUBULES (CT)



Carbonic Anhydrase Inhibitors

Inhibitors = Zola

Acetazolamide "مدي الزوليه يا ستي" أو "يا ستي الزوليه مدي" ممكن نقرأ اسم الدرق بالمقلوب

Dorzolamide "درسوا للميد" ممكن نقرأ اسم الدرق

Drug

Acetazolamide – dorzolamide

Acetazolamide is a potent specific inhibitor of carbonic anhydrase, enzyme inhibition is non competitive

"زوليه" proximal Convoluted = Zola "ملفوفه من الأول"

Mechanism of action

Site of action: proximal convoluted tubules.

Inhibits **carbonic anhydrase (CA) enzyme** in proximal convoluted tubules thus interferes with **NaHCO3 re-absorption** and causes diuresis.

Pharmacokinetics

given orally once a day.

Onset of action is rapid (30 min). t½ 6-9h

Duration of action (12 h).

Excreted by active secretion in proximal convoluted tubules.

Produces alkaline urine Because of the aldosterone effect there will be an exchange between K and Na (K out Na in)

"رحت بيت الخال يوريني الزوليه" alkaline urine (Zola)

Pharmacodynamics

-↑ urine volume mildly acts on HCO_3

It ↓ reabsorption of bicarbonate in the proximal tubule & prevent the acidification of urine in the distal tubule

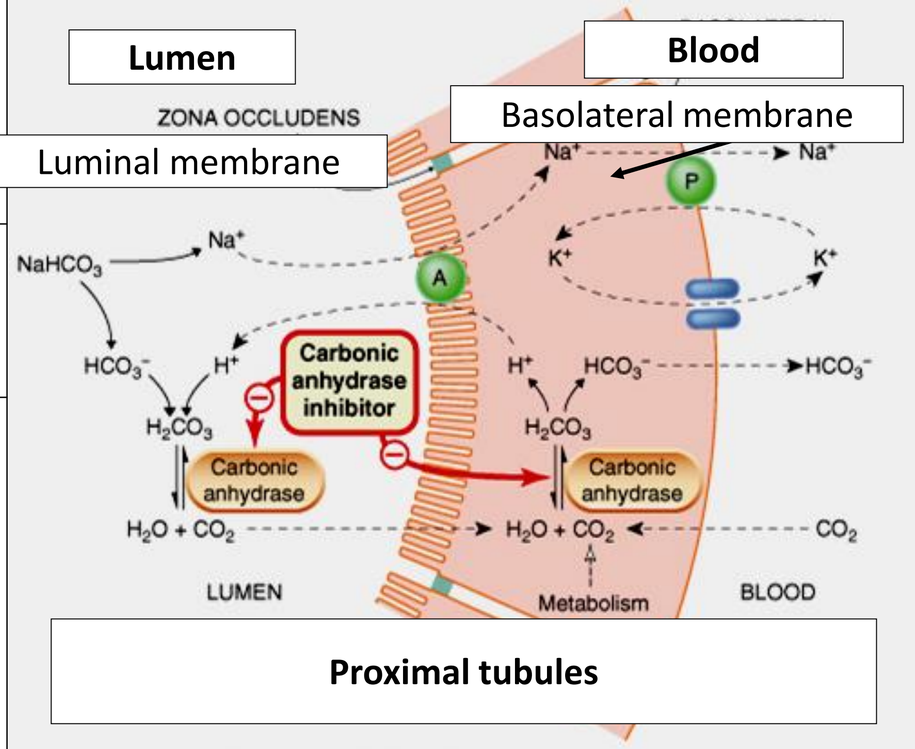
-↑ urinary excretion of sodium, potassium , bicarbonate (alkaline urine).

-With repeated dosage the diuretic a "متى أسدد لك دين الزوليه" **Metabolic acidosis**(Zola) development of **Metabolic acidosis.**

Self- limiting action of acetazolamide restrict its use to mild edema.

-↑ Urinary phosphate excretion. Because phosphate prefer alkaline urine

-Promotes K+ excretion by ↑the load of Na+ delivered to the distal tubules.



Proximal tubules

CA is required for reversible reaction,

in which CO2 +H2O ⇌ H2CO3

H^+ $HCO3^-$

Carbonic Anhydrase Inhibitors

Therapeutic uses

Open angle glaucoma. carbonic anhydrase inhibitors cause ↓ Intraocular pressure (IOP) by reducing aqueous humor formation in ciliary body of eye. (tolerance does not develop to this effect) which drug? The one taken as topically which is Dorzolamide

↓ Cerebrospinal fluid (CSF) of brain. ↓of carbonic anhydrase in the choroid plexus →↓formation of CSF. Useful in management of benign intracranial hypertension.

As prophylactic therapy, in acute mountain sickness. given nightly 5 days before the ascent ↓ weakness, breathlessness , dizziness, nausea, cerebral & pulmonary edema.

- Epilepsy (decrease cerebrospinal fluid, CSF). glial cells contain carbonic anhydrase. Nerves are highly responsive to rise in pH. ↑7.4→ 7.8 causes convulsions ↓of neuronal carbonic anhydrase →↓ pH in the vicinity of neurons→↓ convulsions .
- Metabolic alkalosis. especially an alkalosis caused by diuretic-induced increases in H^+ excretion & metabolic alkalosis of heart failure
- Urinary alkalinization to enhance renal excretion of acidic substances (cysteine in cystinuria).
- Hyperphosphatemia

Adverse effects

ACOD azolamide causes ACIDosis

- Hypokalemia (potassium loss).
- Metabolic acidosis.
- Renal stone formation (calcium phosphate stones).
- Hypersensitivity reaction.

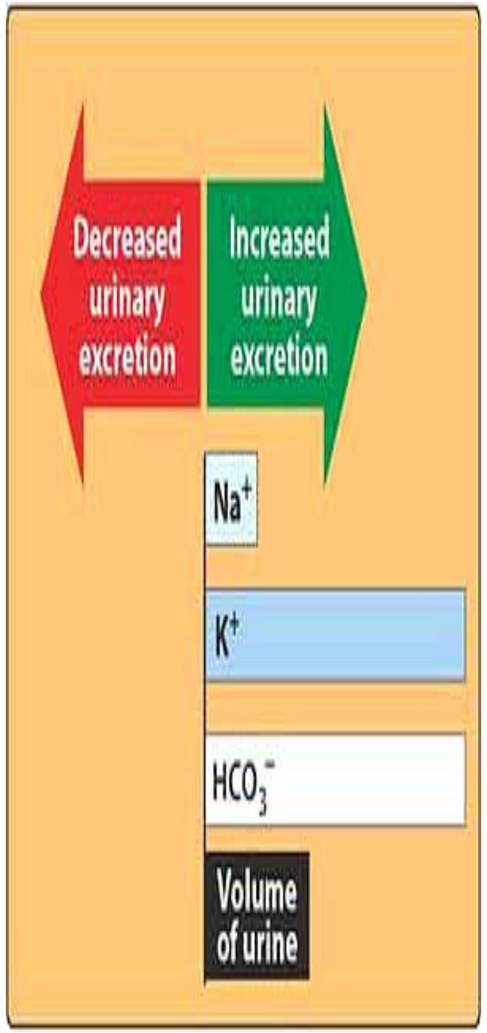
- Drowsiness
- Numbness
- Tingling sensation of the face & extremities

Disturbance of vision

Contraindicated in patients with liver cirrhosis (alkaline urine ↓excretion of NH_4→ hyperammonemia & hepatic encephalopathy)

Dorzolamide

- Is a carbonic anhydrase inhibitor
- Used topically for treatment of open-angle glaucoma. "درسوا للميد زين وفتحوا عيونكم"
- no diuretic or systemic side effects (Why?) it's given as eye drops



Why do CA inhibitors have weak diuretic properties?

Diuretic properties decreases after several days as the blood bicarbonate falls.

LOOP DIURETICS
High Ceiling diuretics

The most potent diuretic , termed **“high ceiling diuretic”**

Efficacy: High natriuresis as 25-30% Na^+ is reabsorbed

Induce expression of COX, PGE↓ salt transport in TAL

↓Renal vascular resistance &↑renal blood flow→ PGs

- Potency 40 ,t½ 0.8 h

Bumetanide

B ume tanide ”بأمي تنادي“ ممكن نقرأ اسم الدرق

- Potency 0.7, t½ 1h

Ethacrynic Acid

Etha cry nic Acid ” إذا بكى نسى“ ممكن نقرأ اسم الدرق

Tor semide ”ثور صامد“ ممكن نقرأ اسم الدرق

Torsemide

- Potency 3, t½ 3.5h

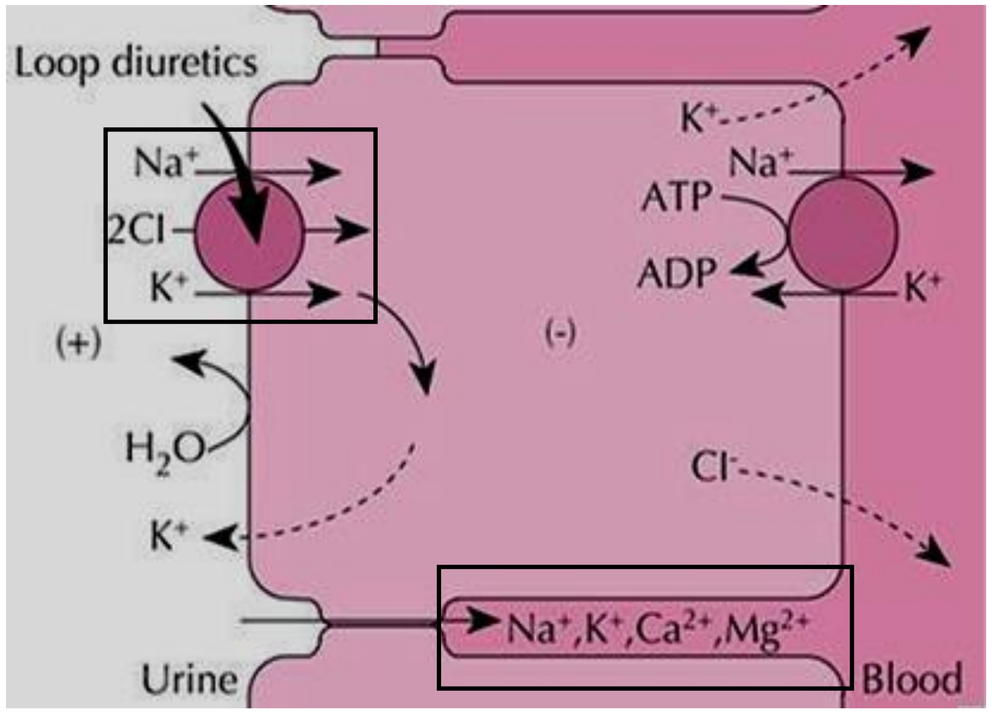
For Semide ”لأجل صامد“ ممكن نقرأ اسم الدرق

Furosemide

- Potency 1, t½ 1.5h

Ascending loop of Henle

- Is impermeable to water
- **In thick ascending loop of Henle (TAL)** is responsible for active re-absorption of Na, K and Cl (25-30% Na^+ is reabsorbed) via transport system in luminal membrane called $Na^+/ K^+ / 2Cl^-$ co-transporter
- Ca and Mg are reabsorbed and enter the interstitial fluid via paracellular pathway



LOOP DIURETICS

Mechanism	Pharmacokinetics	Pharmacological effects	Uses	Adverse effects
inhibit **Na^+ / K^+ / 2 Cl^- co-transporter** in the luminal membrane of the thick ascending loop of Henle (TAL). inhibit Ca^{++} and Mg^{++} re-absorption.	Given orally or I. V. Have fast onset of action (suitable for emergency) Have short duration of action. Excreted by active tubular secretion of weak acids into urine. By acid carrier (avidly bound to plasma proteins). Interfere with uric acid secretion (hyperuricemia). #gout Bumetanide is the most potent	↑urinary excretion of Na^+ and K^+. ↑urinary excretion Ca^{++} and Mg^{++}. ↑urine volume. ↑renal blood flow. Furosemide and ethacrynic acid reduce pulmonary congestion and left ventricular filling pressures in heart failure →↑ venous capacitance	because of their rapid onset of action they are drugs of choice for emergency situations as: Severe edema associated with congestive heart failure, nephrotic syndrome. Treatment for Oliguric ARF. Treatment of hypercalcemia 3 Acutes: Acute pulmonary edema. Acute hyperkalaemia. Acute hypercalcemia. Toxicity of Br, F & I	• Hypovolemia. • Hyponatraemia (↓ blood Na^+). • Hypokalemia (↓ blood K^+). • Hypomagnesaemia (↓ blood Mg^{2+}). • Hypocalcaemia (↓ blood Ca^{2+}). • Metabolic alkalosis. Alkalosis because H goes in urine. • Postural hypotension, first dose causes severe drop in BP. • **Dietary K supplementation or K-sparing diuretics should be used to avoid hypokalemia.** • Hyperuricemia **(increase blood uric acid and gouty attack).** Oto toxicity = Loop Diuretics • Ototoxicity **(risk increased if combined with aminoglycosides).** • Allergic reactions, Hypersensitivity • To sulphonamides. • Hyperglycemia. • Anurea unresponsive to a trial dose of loop diuretic.

Thiazide Diuretics

Low Ceiling diuretics

Drugs as:

Metolazone
- **Potency 5, t½ 5h**

Chlorothiazide
- **Potency 0.1, t½ 2h**

Hydrochlorothiazide
- **Potency 1 , t½ 3h**

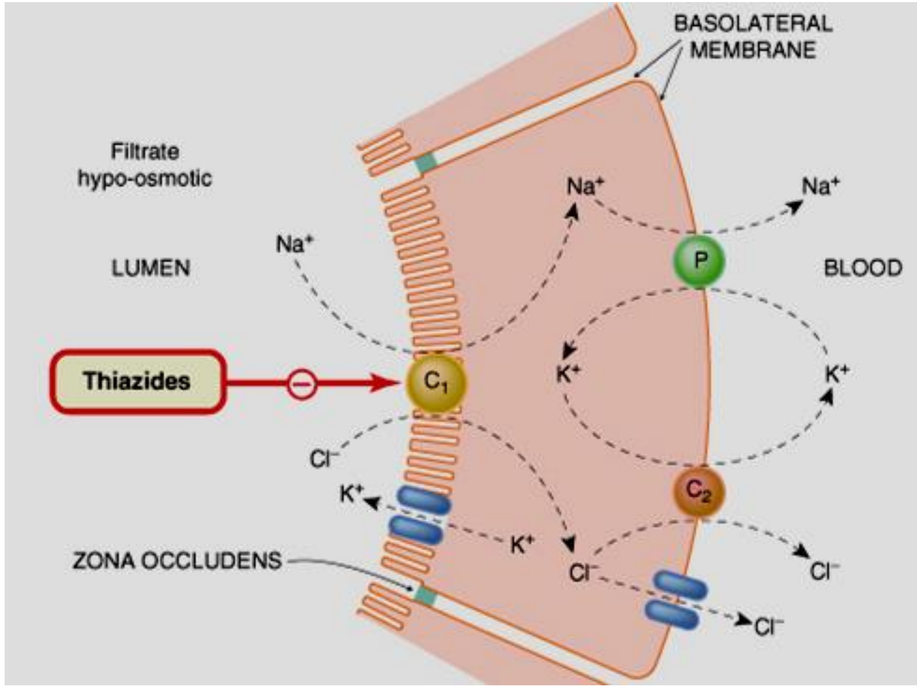
most commonly used thiazide diuretic

Chlorthalidone

Potency 10, t½ 26h

(thiazide) كل ما له ذا يزيد (dap) الدب

Indapamide
- **Potency 20, t½ 16h**



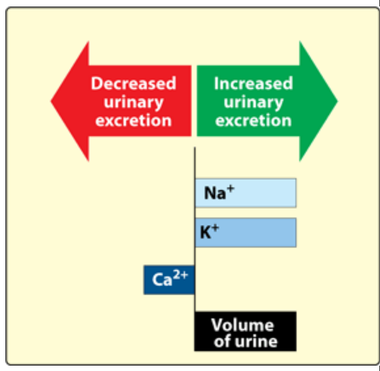
Mechanism of action:

acts via inhibition of Na/Cl co-transporter on the luminal membrane of **distal convoluted tubules.**

Efficacy: Moderate natriuresis (5-10% of filtered load of sodium is reabsorbed).

Thiazide Diuretics

Pharmacokinetics	Given orally, efficiently absorbed from the GIT. slow of onset, Lipid soluble. long duration of action (40 h) are secreted by active tubular secretory system of the kidney. Eliminated by Glomerular filtration & tubular secretion , some is reabsorbed may interfere with uric acid secretion and cause hyperuricemia. thiazide should be taken with caution in patients with gout or high levels of uric acid.
Pharmacological effects	↑ urinary NaCl excretion. Which can result in the excretion of very hyperosmolar (concentrated) urine. ↑ urinary K excretion (Hypokalemia) ↑ urinary magnesium excretion ↓ urinary calcium excretion ↑ calcium re-absorption (Hypercalcemia) ↓ uric acid excretion Causes vasodilatation , diazoxide, non diuretic thiazide is a potent vasodilator of urine volume in case of diabetes insipidus
uses	Treatment of essential hypertension (cheap-well tolerated). Ineffective when the GFR is less than 30 to 40 ml/min, except metolazone & indapamide. Treatment of mild heart failure (to reduce extracellular volume). Calcium nephrolithiasis due to hypercalciuria (to increase calcium re-absorption and decrease renal calcium stones) Treatment for Osteoporosis Calcium Re-absorption (Hypercalcemia & osteoporosis) الطول (Thiazide) ذا يزيد Nephrogenic diabetes insipidus polyuria (decrease blood volume and GFR)

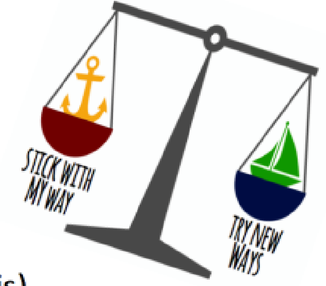


Thiazide Diuretics

Adverse Effects	**Fluid and electrolyte imbalance** **Metabolic alkalosis.**	
	4 HYPOs:	4 HYPERs:
	Hyponatremia	**HyperUricaemia (gout)**
	Hypovolemia (volume depletion)	**HyperCalcemia**
	Hypokalemia	**HyperGlycaemia**
	Hypomagnesaemia	**HyperLipidemia** ↑LDL
		HyperGLUC

MEMORIZING STATION

PHARMACOLOGY 435



Thiazides diuretics Hyper / Hypo effects:

HYPER effects in serum: (Ugly Girls Like **Cars**)
- **HYPER**uricemia (precipitate acute gouty arthritis)
- **HYPER**glycemia
- **HYPER**lipidemia (increase cholesterol and LDL)
- **HYPER**calcemia (renal calcium resorption, decrease calcium in urine)

HYPO effects in serum: (Miss Nora Volunteered in Kuwait)
- **Hypo**magnesaemia
- **Hypo**natremia
- **Hypo**volemia and thus **HYPO**tension (decreases blood volume and peripheral vascular resistance)
- **HYPO**kalemia

Mechanism of antidiuretic effect of thiazide in diabetes insipidus

This is special for abnormal conditions.

Thiazide → **↓ Distal tubular Na+ reabsorption** → **↑Urinary excretion** → **↓ Extracellular volume** → **↑Proximal Na+ & Water reabsorption** → **↓ Distal delivery of Na+ & water** → **↓ Urine volume**

K+ sparing diuretics make the K+ stay (not go down in the urine) STAy
Spironolactone
Triamterene
Amiloride

PHARMACOLOGY 435

Potassium-sparing diuretics

- Steroids
  - Aldosteron Antagonists
    - Spironolactone
      - The K STAys
    - Eplerenone
- nonsteroids
  - Na^+ channels inhibitors
    - Amiloride
      - Amil or ride "أمل أو رايد" أو "أمل أو عايدة" ممكن نقرأ اسم الدرق
      - Amal ride the car(amiloride) and said (trai metrene) , ok give me the soare key (K Sparing diuretic)
    - Triamterene
      - Tria mterene "تراي مترين = يعني ترى طولي مترين" ممكن نقرأ اسم الدرق

Mechanism of action

Act in **collecting tubules** and ducts by inhibiting Na re-absorption and K & H excretion (K-sparing effect) by either:

Inhibition of Na influx through Na channels in the luminal membrane

(triamterene – amiloride).

By antagonizing cytoplasmic aldosterone receptors

(Spironolactone)

Potassium-sparing diuretics

Pharmacodynamics	↑ urinary Na^+ excretion ↓ urinary K^+ excretion Hyperkalemia ↓ H^+ excretion (acidosis)
Therapeutic uses	• Drug of choice for patients with hepatic cirrhosis • Secondary hyperaldosteronism. hyperaldosteronism is divided into primary and secondary secondary is a result of a disease like hepatic cirrhosis due to hyperalbunimia which decreases blood volume which stimulates RAAS and results in hyperaldosternism. **(CHF, hepatic cirrhosis, nephrotic syndrome).** • Treatment of hypertension **(combined with thiazide or loop diuretics to correct for hypokalemia).** to prevent loss if supplements didn't work
Adverse Effects	Hyperkalaemia. Metabolic acidosis. Gynaecomastia. only in spironolactone because it blocks testostrone and aldosterone leading to a decrease in sexaul desire GIT upset and peptic ulcer
Contraindications	**Hyperkalaemia:** as in chronic renal failure, K+ supplementation, β-blockers or ACE inhibitors. (Hyper) كبير (kalaemia) كلام (no) مان بلا (Spiro) سوبر **liver disease** (dose adjustment is needed).

Only in males slides

Aldosterone Antagonists

Aldosterone antagonists are competitive antagonist at the collecting duct→↑ Excretion of Na+,Cl-&↓Excretion of K+,H+,NH4

Actions depend on renal PGs production

	spironolactone	Eplerenone 100x more specific than spironolactone at clinical dose
pharmacokinetics	Well absorbed from the GIT ,t½=1.6h. Highly protein- bound Undergoes enterohepatic recycling. Delayed onset of action (nuclear receptor), maximum diuretic action 4 days. Converted in gut & liver to canrenone [active metabolite, t½=16h].	Eliminated by metabolism(CYP3A4),t½ 5h. Low affinity for progesterone and androgen receptors
	Both ineffective in adrenalectomized patients	
Therapeutic uses	• Enhances Natriuresis caused by Other Diuretics. • Prevents Hypokalemia. • Thus, Used in Combination with Loop & Thiazide Diuretics • Primary Hyper-aldosteronism called Conn's syndrome. • Secondary hyperaldosteronism caused by nephrotic syndrome, heart failure and liver cirrhosis.	• Edema of Liver Cirrhosis. • resistant Hypertension. • Nephrotic syndrome. • Heart Failure, Improve survival.
ADRS	• Hyperkalemia • Metabolic Acidosis in cirrhotic patients • CNS Side Effects • Impotence • Gynecomastia	• Menstrual Irregularities • Hirsutism • Deepening of Voice • Peptic Ulcers • Gastritis
contraindications	• Hyperkalemia • Renal failure • Other K+ sparing diuretics	• ACE-I • K+ suplement

Only in males slides

	Sodium channel inhibitors	
	Triamterene 0.1, t½ 4.2 h, elimination by metabolism	Amiloride Potency 1, t½ 21h, Renal elimination
Therapeutic uses	Enhances Natriuresis caused by Other Diuretics Prevents Hypokalemia Thus, Used in Combination with Loop & Thiazide Diuretics Liddle's Syndrome genetic disorder characterized by hyperactivity of Na channels in collecting ducts Lithium-Induced Diabetes Insipidus	
ADRS	Hyperkalemia Renal Stones Interstitial Nephritis Megaloblastosis in cirrhotic patients caused by block to folate "folic acid"	Hyperkalemia
contraindications	Hyperkalemia Renal failure Other K+ sparing diuretics ACE-I & ARBs K+ suplement Aliskiren	

Osmotic diuretics

Drug

Mannitol

ممكن نقرأ اسم الدرق ماني طويل

Osmotic diuretics =Mannitol (Suger) اسمها د.منان تجنن مثل السكر

Pharmacokinetics

- Poorly absorbed, If given orally ⟶ osmotic diarrhea
- Given intravenously
- Not metabolized
- Excreted by glomerular filtration without being re-absorbed or secreted within 30-60 min .
- t½0.25-1.7h, prolonged in renal failure to 36h

منان (Mannitol) ما تأخذ وتعطي(not Reabsorbed or secreted) مع أي أحد

ماني (Mannitol) ماخذ ومعطي(not Reabsorbed or secreted) معك

Pharmacological actions

Mannitol increases urine output by osmosis, drawing water out of cells and into the bloodstream

Acts in proximal tubules & descending loop of Henle by **osmotic effect.** IV administration of any solute filtered by glomeruli may produce osmotic diuresis when the amount delivered to tubules exceeds their absorptive capacity

↑water excretion with relatively less effect on Na+ **(water diuresis).**

د.منان تعرف تجذب البنات بشرحها (drag water out of the cell) وتخلي المحاضرة زي المويه سهله(only drag the water)

Expand the extracellular fluid volume (initially), decrease blood viscosity, and inhibit renin release, ↑renal blood flow.

Therapeutic uses

Acute renal failure due to shock or trauma (maintain urine flow- preserve kidney function). severe injury , haemorrhage, hypovolaemia, →↓ GFR, absorption of water & salts is complete , distal part dries up→ irreversible damage

In acute drug poisoning: To eliminate drugs that are reabsorbed from the renal tubules e.g. salicylates, barbiturates, bromides .

To ↓ intracranial & intraocular pressure before ophthalmic or brain procedures **(cerebral edema).**

To maintain urine volume & to prevent anuria resulting from large pigmentation load to the kidney e.g. haemolysis, rhabdomyolysis. But contraindicated in Anuric patients or patients not responding to a test dose of mannitol

Adverse effects

Headache, nausea, vomiting → hyponatremia

Extracellular volume expansion, complicates heart failure & pulmonary oedema. Contraindicated in chronic heart failure

Excessive use→ dehydration & *hypernatraemia (Adequate water replacement is required). The only drug with this effect

Only in males slides

Drug – Drug interactions

Tbiazide diuretics

Thiazides Diminish effect → Uricosurics Sulphonylurea

Thiazides Increase effect → Digitalis Diazoxide

NSAIDs → Reduce Thiazide efficacy

Loop diuretics

NSAIDS Probenecid → ↓Diuretic Response

Digitalis → Arrhythmias

Aminoglycosides → ↑ Ototoxicity of Loop Diuretic

K-Sparing diuretics

A) Sodium Channel blockers:

ACE Inhibitors
Beta-Blockers
K Supplements
K-Sparing Diuretics
Aliskiren

→ **↑Hyperkalemia induced by K-Sparing diuretics**

B) Aldosterone antagonists

Aldosterone antagonist depends on PG so NSAIDS block their effect

Salicylates → ↓Secretion of canrenone
↓Efficacy of Spironolactone

Spironolactone alters clearance → Digitalis

Treatment of hypertension:

Thiazide diuretics

used alone or in combination with beta-blockers at low-dose (fewer side effects)

In presence of renal failure, loop diuretic is used.

Edema States

Thiazide diuretic is used in mild edema with normal renal function

Loop diuretics are used in cases with impaired renal function.

Congestive Heart failure

Thiazides may be used in only mild cases with well-preserved renal function

Loop diuretics are much preferred in severe cases especially when GF is lowered

In life-threatening acute pulmonary edema, furosemide is given IV.

Renal failure

Thiazides are used till GFR ≥ 40-50 ml/min

Loop diuretic are used below given values.

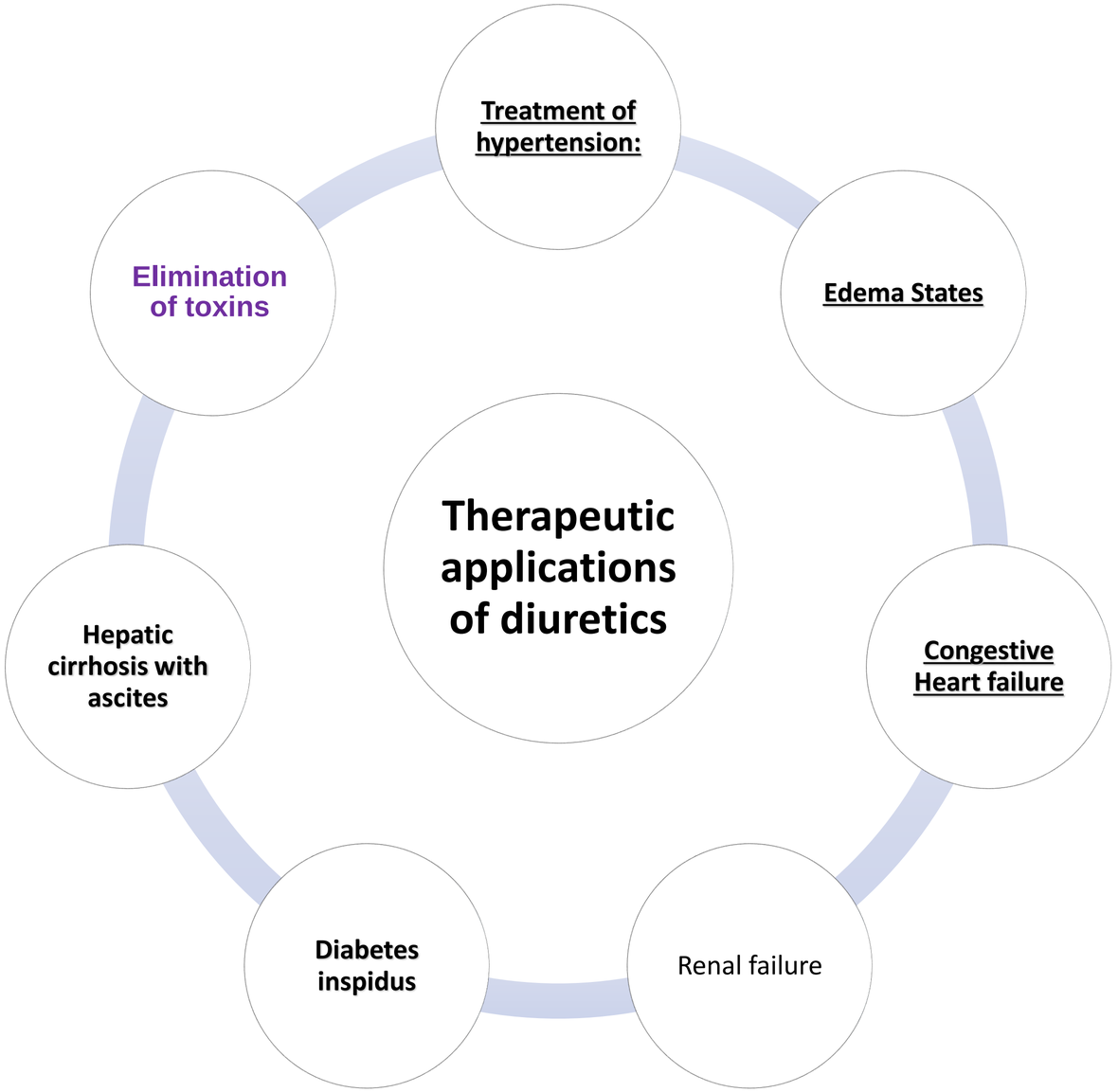
Diabetes inspidus

Large volume(>10 L/day) of dilute urine

thiazide diuretics reduces urine volume

Hepatic cirrhosis with ascites

Spironolactone is of choice.



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SUMMARY

Diuretics	Mechanism of action	Effects	Uses	Side effects
CA inhibitors Acetohexamide Dorzolamide	Inhibition of NaHCO3 reabsorption in PCT	↑ Urinary Na HCO3, K Urinary alkalosis Metabolic acidosis	Glaucoma, epilepsy Mountain sickness Alkalosis Phosphatemia	Metabolic acidosis , Urinary alkalosis Hypokalemia
Loop diuretics Furosemide	Na/K/2Cl transporter in TAL the most effective	↑Urinary Na, K, Ca, Mg	Acute pulmonary edema (Drug of choice) Heart failure Hyperkalemia, Hypercalcemia	Hypokalemia, hypovolemia, hyponatremia, hypomagnesemia, **hypocalcemia** Precipitate gout, alkalosis
Thiazide diuretics hydrochlorothiazide	Na and Cl cotransporter in DCT	↑Urinary Na, K, Mg BUT↓ urinary Ca (hypercalcemia) Metabolic alkalosis	Commonly used Hypertension, mild heart failure, nephrolithiasis, diabetes inspidus	Hypokalemia, hyponatremia, hypovolemia,hypomagnesemia, **hypercalcemia** Metabolic Alkalosis, precipitate gout Hyperlipidemia, hyperglycemia
K-sparing diuretic Spironolactone.	competitive antagonist of aldosterone in CCT	↑ Urinary Na ↓ K, H secretion Metabolic acidosis	Hepatic cirrhosis (Drug of choice)	Gynaecomastia **Hyperkalaemia**, Metabolic acidosis. GIT upset and peptic ulcer
Osmotic diuretic Mannitol	Osmotic effect in PCT	↑Urine excretion ↑ Little Na	• Cerebral edema, glaucoma • Acute renal failure, drug toxicities	Extracellular water expansion Dehydration Hypernatremia

MCQ

1. A patient taking aminoglycosides was prescribed one of these diuretics, then the patient experienced hearing loss and damage to the ear. Which drug did he take?

A) Chlorothiazide . B) Furosemide. C) Acetozolamide. D) Spironolactone.

2. A patient came to the emergency room suffering from severe pulmonary edema, which diuretic is the best choice for his case?

A) Spironolactone. B) Dorzolamide. C) furosemide. D) Hydrochlorothiazide.

3. How do thiazide diuretics affect these ions?

A) Inhibits Ca, Mg, Na, K. B) Reabsorbs Ca, Mg, Na, K. C) Inhibits Ca, Mg, but reabsorbs Na, K. D) Inhibits Mg, Na, K, but reabsorbs Ca.

4. A diabetic patient who urinates frequently was given a thiazide diuretic, how will it fix his problem as a compensatory mechanism?

A) Decrease GFR and blood volume by reabsorbing sodium. B) Increasing GFR and blood volume by excreting more sodium.

C) Decreasing GFR and increasing blood volume. D) Increasing GFR and decreasing blood volume

5. A patient had been taken (hydrochlorothiazide) , he came to see his doctor and on investigation the doctor noticed a decrease of K level , what is the drug that the doctor might use it in combination in this case?

A) Mannitol. B) Indapamide. C) Metalazone. D) Amiloride.

6. Which one of these diuretics is known to be (water diuresis) ?

A) Amiloride. B) Metalazone. C) Mannitol. D) hydrochlorothiazide.

7. Spironolactone can be characterized by which of the following properties ?

A) It binds to a transmembrane receptor. B) It inhibits aldosterone synthesis. C) it is bio transformed to an active metabolite. D) It is more potent than hydrochlorothiazide .

8. Which of the following is the mechanism of action of spironolactone?

A) Though osmotic effects. B) Through enzyme inhibition. C) Through interaction with hormonal receptors. D) Through inhibition of a co-transporter.

9. Which of the following diuretics act on specific membrane transport proteins?

A) A) Mannitol. B) Indapamide. C) Amiloride. D) Spironolactone.

Answers:
1. B
2. C
3. D
4. A
5. D
6. C
7. A
8. C
9. B

MCQ

10. A 54-year old male develops congestive heart failure after suffering his second myocardial infarction. His physician put him on a regimen of several medications, including frusemide. On follow-up, the patient is found to have hypokalemia. The addition of which medication would likely resolve the problem of hypokalemia, while helping to treat the underling condition?

A) Hydrochlorothiazide. B) Spironolactone. C) Acetazolamide. D) Ethacrynic Acid.

11. A 50-year- old-male with pitting edema of the ankles developed gynecomastia and erectile dysfunction while being treated with which of the following drugs?

A) Hydrochlorothiazide. B) Metolazone. C) Spironolactone. D) triamterine.

12. A 45-year- old female with a long history of alcohol abuse is being treated for cirrhosis- associated ascites. Her physician decided to give her amiloride, a diuretic helpful in edema caused by cirrhosis. What common side effect should be monitored in this patient?

A) Hyponatremia. B) Hypercalcemia. C) Hypermagnesemia. D) Hyperkalemia.

13. A 45-year- old male with history of medication- controlled hypertension presented to you with complaints of a painful swollen big toe. You suspected gout and checked his uric acid levels, which were elevated. Upon looking at the list of his medication you realized that one of them might be the cause. Which medication might that be?

A) Dorzolamide. B) Spironolactone. C) Hydrochlorothiazide. D) Mannitol.

14. Which of the following statements about diuretics is false?

A) All potassium-sparing diuretics may be taken orally.
B) Osmotic diuretics cause an expansion of the extracellular fluid volume.
C) Spironolactone and amiloride produce potassium loss by the same mechanism.
D) Both a & b.

15. A 50-year- old-man with mild hypertension treated with spironolactone complains of discomfort in his chest, he has slightly enlarged fat deposits in his breasts with prominent nipples. His physician decides to switch this patient to a drug that has the same mechanism of action but will avoid this adverse effect. Which of the following drugs would he use?

A) Amiloride. B) Torsemide. C) Chlorthalidone. D) Aldosterone.

Answers:
10. B
11. C
12. D
13. C
14. C
15. A

MCQ

16. Which drug falls under the carbonic anhydrase inhibitors class?

A) Acetazolamide. B) Bumetanide. C) Chlorothiazide. D) amiloride.

17. A 75-year- old woman with hypertension is being treated with a thiazide. Her blood pressure responds well and reads at 120/76mm hg. After several months on the medication, she complains of being tired and weak. an analysis of the blood indicates low values for which of the following?

A) Calcium. B) Potassium. C) Sodium. D) Uric acid.

18. A 57-year- old man with a history of heavy alcohol use is being admitted for a first episode of congestive heart failure, which likely resulted from untreated alcoholic cardiomyopathy. the cardiologist decides to start the patient on diuretic therapy. which class of diuretics is preferred in this case?

A. loop diuretics because they exert their action at the distal convoluted tubule
B. loop diuretics because the thick ascending limb is an area of high capacity for NaCl reabsorption
C. thiazide diuretics because they exert their action at the thick ascending limb of the loop of Henle
D. thiazide diuretics because they increase cardiac output

19. A 35-year- old woman presents to your office for a regular checkup. she has no complains. On examination, her blood pressure is slightly elevated at 145/85. she is physically fit and follows al heathy diet. you decide to start her on antihypertensive therapy and prescribe hydrochlorothiazide. how does this agent work?

A) inhibits reabsorption of sodium chloride in the early DCT. B) decrease net excretion of chloride, sodium and potassium

C) increases excretion of calcium. D) inhibits reabsorption of sodium chloride in the thick ascending limb of the loop of Henle.

20. which of the following is an action of loop diuretics on ionic excretion?

A) increased sodium excretion. B) decrease magnesium loss. C) decreased calcium loss. D) decreased potassium loss.

21. when furosemide is administrated to a patient with pulmonary edema, there is often symptomatic relief within 5 minutes of starting treatment. this relief is primarily due to:

A) a rapid diuretic effect. B) an increase in venous capacitance. C) a direct effect on myocardial contractility D. psychological effects.

Answers:
16. A
17. B
18. B
19. A
20. A
21. B

MCQ

22. A new diuretic is being studied in human volunteers. compared with placebo, the new drug increase urine volume, increases urinary Ca2+, increase plasma pH and decrease serum K+. if this new drug has a similar mechanism of action to an established diuretic, it probably :

A) block the NaCl cotransporter in the DCT. **B)blocks aldosterone receptors in the CT**

C) inhibits carbonic anhydrase in the PCT. **D) inhibits the Na+/K+/2Cl- cotransporter in the TAL.**

23. Which drug of the following diuretics is used in treatment of glaucoma?

A) Acetazolamide

B) Dorzolamide

C) Furosemide

D) Indapamide

24. Which class of diuretics work by acting on proximal tubules?

A) Loop diuretics

B) Thiazide diuretics

C) Potassium-sparing diuretics

D) Carbonic Anhydrase Inhibitors

Answers:
22. A
23. B
24. D

Pharmacology
team 436

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