PHARMACOLOGY-II (VIth Semester)

Q.1.

a) Electrolytes

→ Salts and minerals that can conduct electrical impulses in the body. common human electrolytes are sodium chlorides, Potassium, calcium and sodium bicarbonate. Electrolytes control the fluid balance of the body and are important in muscle contraction energy generation and almost every major biochemical reaction in the body.

b) COPD

→ Chronic obstructive pulmonary disease

c) Naloxone

→ It is a non-selective and competitive opioid receptor antagonist. It works by reversing the depression of the central nervous system and respiratory system caused by opioids.

d) B.P formula

$$B.P = 4.4 \times \frac{\text{Blood volume}}{\text{Body weight}}$$

e) Hypovolemia

→ Hypovolemic shock is a life-threatening condition that results when you lose more than 20% of your body's blood or fluid supply.

Q.2. Write down the classification and MOA of antihypertensive drugs?

Ans. classification:-

① Diuretics

Thiazides :- Hydrochlorothiazides, chlorthalidone

High ceiling :- Furosemide

K+ sparing :- spironolactone, Amiloride

② ACE Inhibitor
→ Ceptopril, Enalapril, Lisinopril, Perindopril

③ Angiotensin (AT_1 receptor) blocker
→ Losartan, Candesartan, Irbesartan

④ Direct renin inhibitor
→ Aliskiren

⑤ Calcium channel blocker
→ Verapamil, Diltiazem, Nifedipine, Amlodipine

⑥ β-Adrenergic blocker
→ Propranolol, Metoprolol

⑦ β+α Adrenergic blocker
→ Lebetalol, Carvedilol

⑧ α-Adrenergic blocker
→ Prazosin, Doxazosin, Terazosin

⑨ Central Sympatholytic
→ Clonidine, Methyldopa

⑩ Vasodilator
- Arteriolar → Hydralazine, Minoxidil
- Arteriolar + Venous → Sodium nitroprusside

MOA of Diuretics
- ↑ urine output
- ↓ fluid overload
- ↓ B.P

MOA of ACE Inhibitor
- ↑ vasoconstrictor
- ↑ Sympathetic action
- ↑ Aldesteron
- ↑ B.P

MOA of Angiotensin
- Blocking the binding of angiotensin II to its receptor hence less activity of angiotensin II which les B.P

* MOA of β-blockers
→ β-blocker blocks sympathetic function on heart by blocking β-receptor.
→ ↓ HR
• ↓ B.P
• ↓ Myocardial contraction

* MOA of calcium channel blockers
• This Ca^{++} channel are found in different parts of body.
• These Ca^{++} channel blockers block: T-types and/or L-type Ca^{++} channel.

Q.8 Write down the classification of drug used in bronchial asthama?

Ans. Classification of Brochial asthama

1) Bronchodilators
A) β_2 - Sympathomimetics
→ Salbutamol, Terbutaline, Ephedrine
B) Methylxanthine
→ Theophylline, Aminophylline, Doxophylline
C) Anticholinergic
→ Ipratropium bromide, Tiotropium bromide
D) Leukotriene antagonists
→ Montelukast, Zafirlukast
3) Mast cell stabilizers
→ Sodium cromoglycate, Ketotifen
4) Corticosteroids
a) Systemic
→ Hydrocortisone, Prednicelone
b) Inhalational
→ Beclomethasone dipropionate, Ciclesonide
5) Anti-IgE antibody
→ Omalizumab

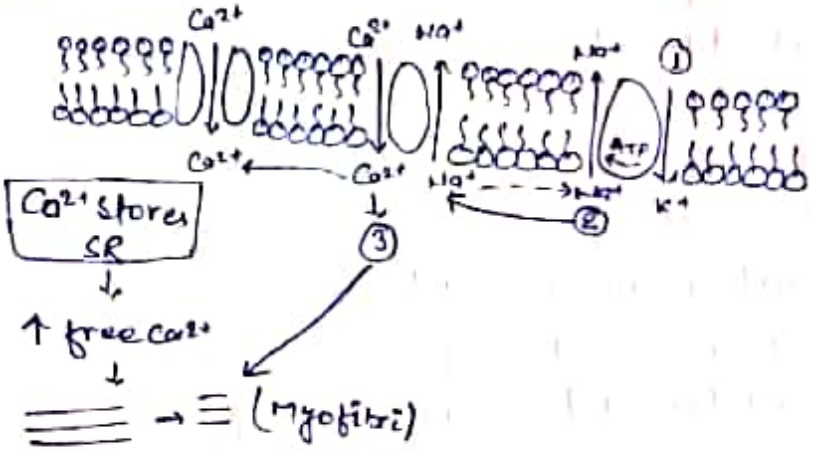
Q.4. Write short note on cardiac glycoside, their MOA and sources of digitalis?

Ans. The term digitalis is used to refer to entire group of cardiac glycoside.

All glycosides consists of an a glycon active part i.e steroid nucleus lactone ring to which are attached one or more sugar moiety. The Pharmacological activity resides in the aglycon but attached to sugar modify solubility & cell Permeability.

Sources	Glycosides
• Digitalis purpurea (leaf)	Digitoxin, Gitoxin
• Digitalis Lanata (leaf)	Digitoxin, Gitoxin
• Astropanthus gratus (seed)	Strophanthin-G

MOA



1) Digitalis inhibits Na^+-K^+ exchange by Na^+-K^+ ATPase.
2) Conc^n of intracellular Na^+ ↑es.
3) ↑ed Na^+ leads to a greater Ca^{2+} influx causing stronger systolic conc^n

⇒ Increased the contractility of cardiac cell by ↑ing free Ca^{2+} conc^n

↓

Inhibition of Na^+-K^+ ATPase causes as result ↑es in intracellular Ca^{2+} occurs

↓

↑ directly facilitate entry of Ca^{2+} cardiac cell

↑ Release of Ca^{2+} from SR as results myofibril contraction occurs.

5 a) Differentiate between classical & variant angina?

Ans.

Classical Angina	variant Angina
• Typical, classic, common angina	• prizmetal angina
• occurs during emotional stress, heavy exercise	• occurs in rest also
• Easily predictable	• Due to spasm of coronary artery.
• Treating more rest, nitroglycerin	
• Due to atherosclerosis	

b) Write down the MOA of Furosemide & Heparin?

- MOA of Furosemide

→ Furosemide, a loop diuretic, inhibits water reabsorption in the nephron by blocking the sodium-potassium-chloride contransporter in the thick ascending limb of the loop of Henle. This is achieved through competitive inhibition at the chloride binding site on the contransporter, thus preventing the transport of sodium from the lumen of the loop of Henle into the basolateral interstitium.
The lumen becomes more hypertonic while the interstitium becomes less hypertonic which in turn diminishes the osmotic gradient for H_2O reabsorption.

- MOA of Heparin

- Heparin catalyzes the inhibition of several coagulation proteases by antithrombin.
- AT- inhibits activated coagulation factors of the intrinsic and common pathways, including thrombin, Xa & IXa.
- Heparin Yes the rate of thrombin-antithrombin reaction at least a thousandfold by serving as a catalytic template to which both the inhibitor and Protease bind.