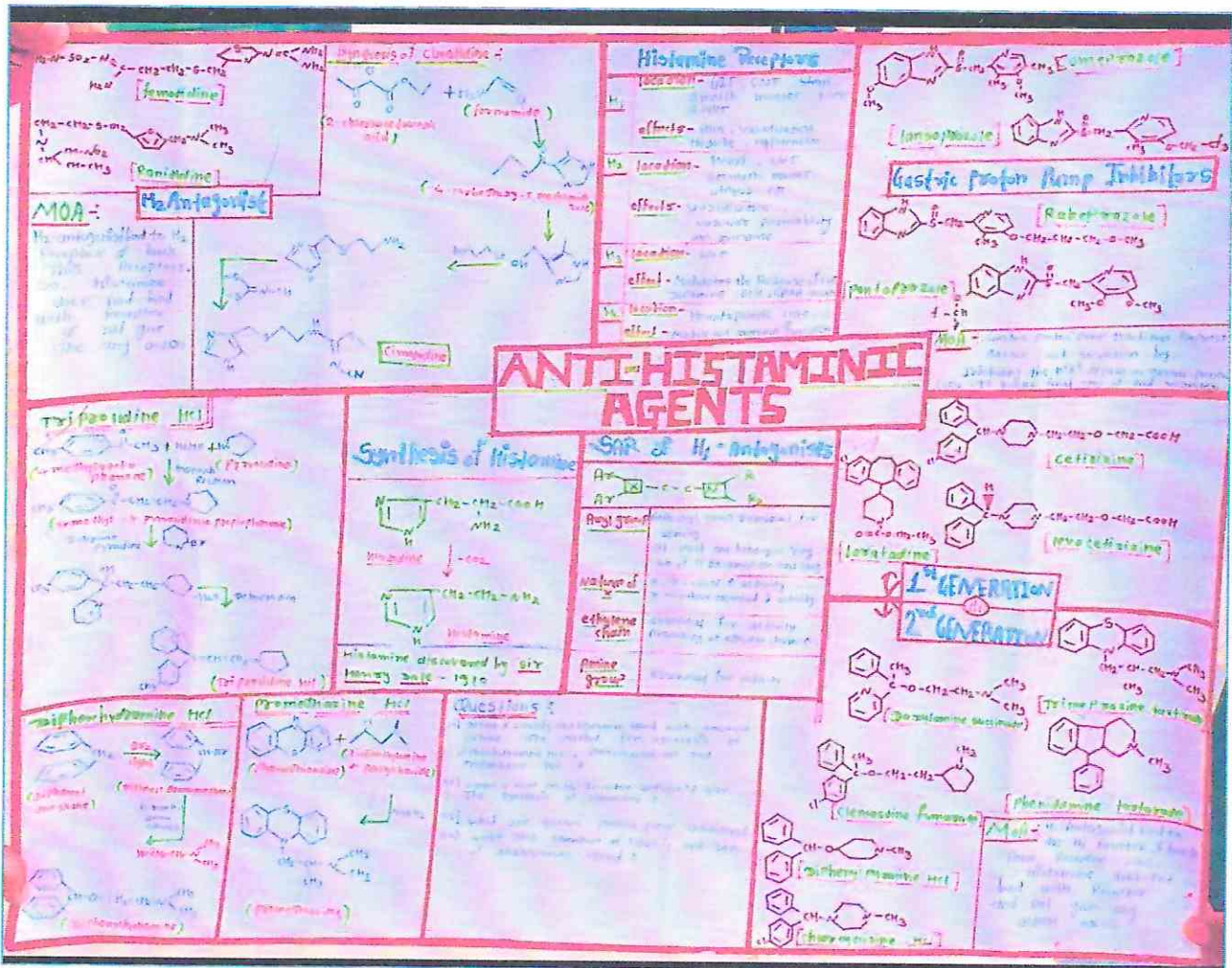


Teaching with special method like Mind Maps etc

In the second and third years of the B. Pharmacy, Medicinal Chemistry proves to be a challenging subject. The intricate structures of medicinal drugs make it difficult for students to memorize them effectively. To tackle this issue, we employ innovative teaching methods, utilizing Mind Maps on white sheets, to facilitate easier memorization of these complex structures. Instead of relying solely on rote memorization, students actively participate in the creation of visual representations, reinforcing their comprehension of medicinal chemistry concepts.



ANTI-HISTAMINIC AGENTS

Histamine Receptors

- H₁ Receptor**
 - Location - CNS
 - Effectors - smooth muscle contraction, vasodilation
- H₂ Receptor**
 - Location - GI tract
 - Effectors - gastric acid secretion
- H₃ Receptor**
 - Location - CNS
 - Effectors - Modulates the histaminergic system
- H₄ Receptor**
 - Location - Thymus gland, bone marrow
 - Effectors - Modulates immune response

Synthesis of Histamine

Histamine discovered by Sir Henry Dale - 1910

MOA of H₁ Antagonists

MOA of H₂ Antagonists

1st Generation

- Triprolidine HCl**
- Chlorpheniramine HCl**
- Pheniramine HCl**
- Proprietary HCl**
- Phenothiazine HCl**

2nd Generation

- Loratadine**
- Desloratadine**
- Cetirizine**
- Fexofenadine HCl**
- Levocetirizine HCl**
- Chlorpheniramine HCl**
- Pheniramine HCl**

MOA of 2nd Generation

MOA of 1st Generation



ANTI - HYPERLIPIDEMIC AGENTS		Coagulant & Anti-coagulant		
<p>HDL (Good Cholesterol) (High Density Lipoprotein)</p> <p>Good cholesterol carries extra cholesterol to your blood, into your liver where it is broken down and removed from the body. This means high levels of HDL help to maintain a healthy heart.</p> <p>Cholesterol and triglycerides with low level of high density lipoprotein cholesterol (HDL)</p> <p>Ezetimibe</p>	<p>CHOLESTEROL SYNTHESIS</p> <p>3 Acetyl CoA ↓ HMG CoA ↓ Mevalonate ↓ Isopentenyl Unit ↓ Squalene ↓ Lanosterol ↓ Cholesterol</p> <p>LDL (Bad cholesterol) (Low density lipoprotein)</p> <p>Bad cholesterol carries cholesterol to tissues throughout your body. When you have too much LDL, it can build up in the walls of your arteries, causing narrowing & reduced blood flow. LDL is bad for heart health.</p> <p>The incidence of the heart failure is correlated with elevated levels of low density lipoproteins (LDL)</p> <p>Fluvastatin <chem>CC(C)C(O)C1=CC=C(C=C1)C2=CC=CC=C2C3=C(C)C(=O)N(C)C3</chem></p> <p>Atorvastatin <chem>CC(C)C(O)C1=CC=C(C=C1)C2=CC=CC=C2C3=C(C)C(=O)N(C)C3</chem></p> <p>Simvastatin <chem>CC(C)C(O)C1=CC=C(C=C1)C2=CC=CC=C2C3=C(C)C(=O)N(C)C3</chem></p> <p>Statins</p>	<p>Synthesis of Warfarin</p> <p>4-Hydroxycoumarin + Benzoylacetone</p> <p>Acemic Warfarin</p> <p>(Anti-coagulant drug)</p>	<p>Broken Blood Vessel</p> <p>Clotting Factor</p> <p>Prothrombin → Thrombin</p> <p>Fibrinogen → Fibrin</p> <p>Fibrin Mesh</p> <p>Blood clot</p>	<p>Intrinsic Pathway</p> <p>Damaged Vessel Wall</p> <p>XII → XIIa → XI → XIa → X → Xa</p> <p>Extrinsic Pathway</p> <p>Tissue to extravascular space</p> <p>III → VII → VIIa → VIIa + III → VIIa-III complex</p> <p>VIIa-III complex → X → Xa</p>
<p>Serum Cholesterol ↔ Cellular Cholesterol</p> <p>LDL-R</p> <p>Conversion to xanthone within cells to storage as granules</p> <p>Diet → Exogenous → Serum Cholesterol</p> <p>Bile Acids → Intestine → Feces</p> <p>Re-absorption</p> <p>Bile Acid Sequestrants → BAS</p> <p>Lipoprotein Catabolism → Fibrates</p> <p>Clofibrate <chem>CC(C)C(O)C1=CC=C(C=C1)C2=CC=CC=C2C3=C(C)C(=O)N(C)C3</chem></p> <p>Gemfibrozil <chem>CC(C)C(O)C1=CC=C(C=C1)C2=CC=CC=C2C3=C(C)C(=O)N(C)C3</chem></p>	<p>MOA</p> <p>Warfarin → Vitamin K (antidote)</p> <p>Warfarin → Vitamin K (antidote)</p> <p>Warfarin → Vitamin K (antidote)</p>	<p>MOA</p> <p>Warfarin → Vitamin K (antidote)</p> <p>Warfarin → Vitamin K (antidote)</p> <p>Warfarin → Vitamin K (antidote)</p>	<p>MOA</p> <p>Warfarin → Vitamin K (antidote)</p> <p>Warfarin → Vitamin K (antidote)</p> <p>Warfarin → Vitamin K (antidote)</p>	



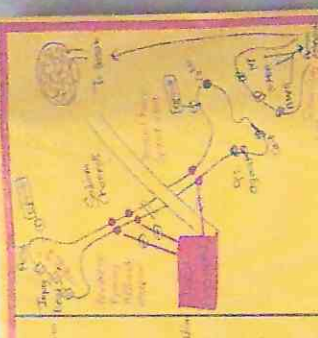
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 Gondur, Dhule

LOCAL ANESTHETICS:

Cause reversible loss of sense - loss of any particular site by neuronal conduction on that particular site.

Mechanism of Action:

- In normal condition, nerve fiber conduction depends on the flow of Na⁺ ions.
- Because the nerve sheath is impermeable to Na⁺ ions.
- Voltage gated Na⁺ channels are present in the nerve sheath.
- When the voltage reaches a certain level (threshold level), the channels open and Na⁺ ions enter the cell.
- This causes depolarization and the nerve impulse is conducted.
- Local anesthetics block the Na⁺ channels.
- This prevents the nerve impulse from being conducted.
- This results in reversible loss of sensation.



Benzoinic Acid Derivatives:

- Cocaine
- Procaine
- Chlorbutol
- Benzocaine
- Tetracaine
- Articaine
- Prilocaine
- Proparacaine
- Propylparacaine
- Propylchlorbutol
- Propylchlorbutol
- Propylchlorbutol

Amino Benzoic Acid Derivatives:

- Procaine
- Chlorbutol
- Benzocaine
- Tetracaine
- Articaine
- Prilocaine
- Propylparacaine
- Propylchlorbutol
- Propylchlorbutol
- Propylchlorbutol

Miscellaneous:

- Articaine
- Prilocaine
- Propylparacaine
- Propylchlorbutol
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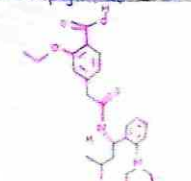

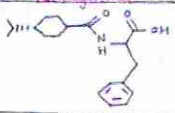
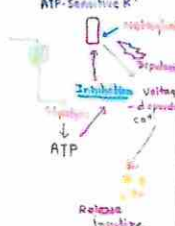
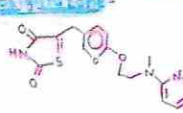
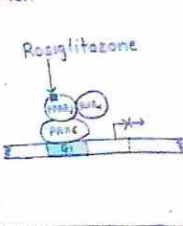
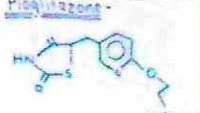

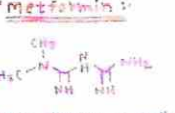
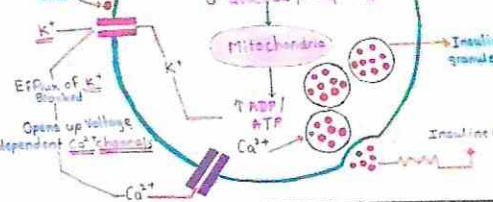
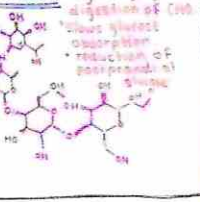
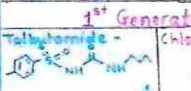
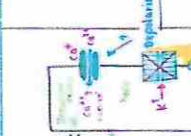
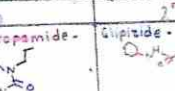

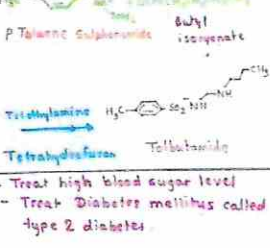
Miscellaneous:

- Articaine
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PRINCOP, Gondur



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<p>Repaglinide</p>  	<p>Meglitinide</p>  	<ul style="list-style-type: none"> Directly stimulate release of insulin from pancreatic beta cells and thereby lower blood glucose concentrations. They work by stimulating insulin secretion. Useful only in patients with some beta cell function. They are oral medications used to treat type-2 diabetes. They trigger production of insulin. 	<ul style="list-style-type: none"> Activation of the gamma isoform of the protein tyrosine kinase receptor. Insulin sensitizer by acting on adipose, muscle. Increase glucose utilization. Decrease glucose production leading to the increase feeding. Decrease hepatic gluconeogenesis. Increase insulin-dependent glucose. 	<p>Thiazolidenedione</p> <p>Rosiglitazone</p>  	<p>Proglitazone</p>  
<p>Bicyclics</p> <p>Intestinal Absorption of CHO ↓ Glucose ↑</p> <p>Glucose Utilization (Glycogenesis) ↑ ↑ Glycolysis via Anaerobic pathway ↑ Glucose uptake BY Stimulating enzyme GLUT 4 (Glucose transp. 4)</p>	<p>Metformin</p>  <ul style="list-style-type: none"> Acts directly or indirectly on liver to lower glucose production, and acts on the gut to the increase glucose utilization. Increase SUR and open the microsome. 	<p>Site of action for Sulfanyl ureas</p> 	<p>Glucosidase Inhibitors</p> <ul style="list-style-type: none"> Inhibit absorption of Carbohydrates Inhibit formation of glucosidase enzyme Inhibit enzymes that convert complex non-absorbable CHO into simple absorbable CHO 	<p>Acarbose - By delaying the digestion of CHO ↓</p> <ul style="list-style-type: none"> ↓ Glucose absorption ↓ production of postprandial glucose 	
<p>1st Generation</p> <p>Tolbutamide</p>  	<p>Chlorpropamide</p> 	<p>2nd Generation</p> <p>Glipizide</p>  <ul style="list-style-type: none"> ↑ Insulin release from pancreas Increase glycolysis stimulate release of insulin from pancreatic beta cells lower blood glucose concentration 	<p>Synthesis of Tolbutamide</p>  <ul style="list-style-type: none"> Treat high blood sugar level Treat Diabetes mellitus called type 2 diabetes. 	<p>Prepared by :-</p> <ul style="list-style-type: none"> Pratik Harindra Chaudhari Harshad Vilas Patil Krishna Ravesh Patil Shikha Preraj Malwar Shubho Harindra Korde Tanhu Smit Inekar 	



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