Antihistamine Drug

- H_1 Blocker
- H_2 Blocker
- H_3 Blocker
- H_4 Blocker

Conventional Antihistaminic

Highly Sedative
Diphenhydramine, Dimenhydrinate, Promethazine, Hydroxyzine

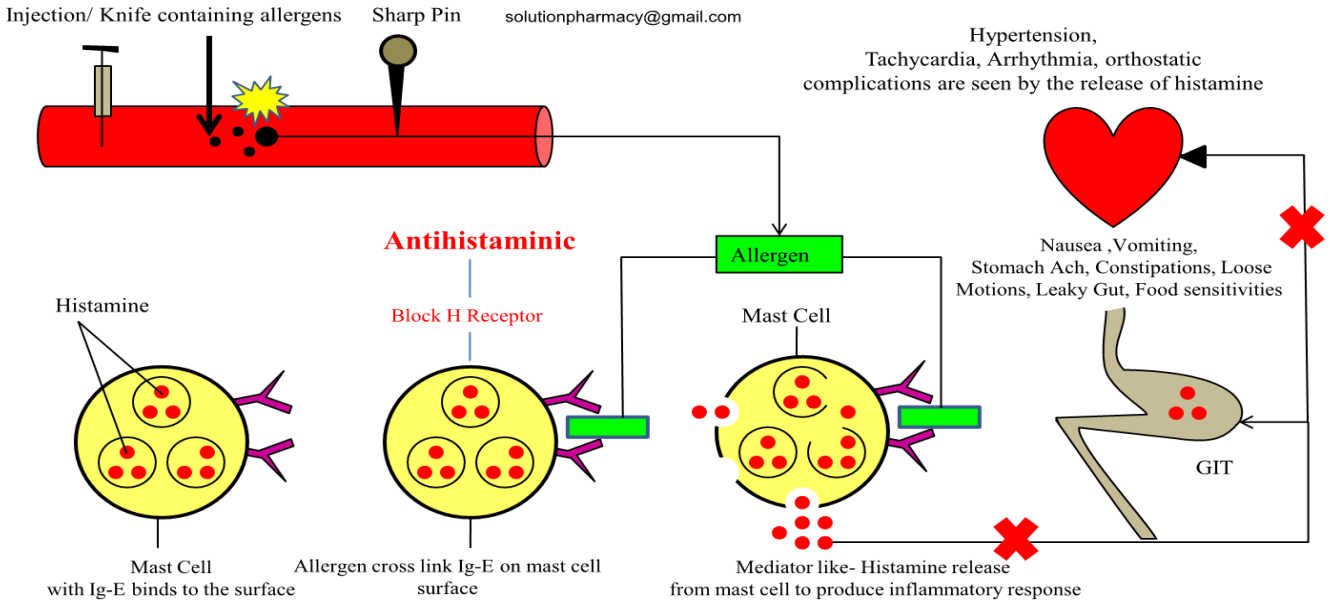
Moderately Sedative
Pheniramine, Cyproheptadine, Meclizine, Buclizine, Cinnarizine

Mild Sedative
Chlorpheniramine, Dexchlorpheniramine, Dimethindene, Triprolidine, Mebhydroline, Cyclizine

2nd Generation
Fexofenadine, Loratadine, Desloratadine, Cetirigine, Levocetrizine, Azelastine, Mizolastine, Ebastine, Rupatadine

Pharmacological Action

1. **Antagonism of Histamine** – Reverse Histamine induced bronchoconstriction, Intestinal and other smooth muscle
2. **Antiallergic Action** – Suppress type I Hypersensitivity reaction. Urticaria, itching and angioedema are well controlled.
3. **CNS** – Few of them depress CNS. Few are effective in preventing motion sickness. Few reduce tremor, Rigidity, antitussive
4. **Local anesthetics** – Some antihistaminic are strong and some are having weak membrane stabilizing property.
5. **Blood Pressure**- Most antihistaminic drug cause fall in BP on IV injection.



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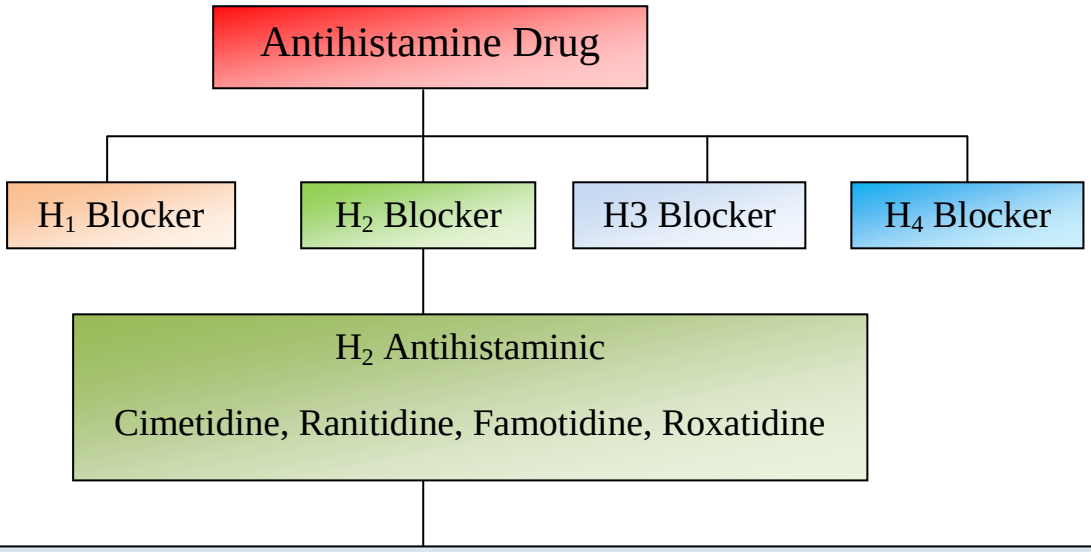
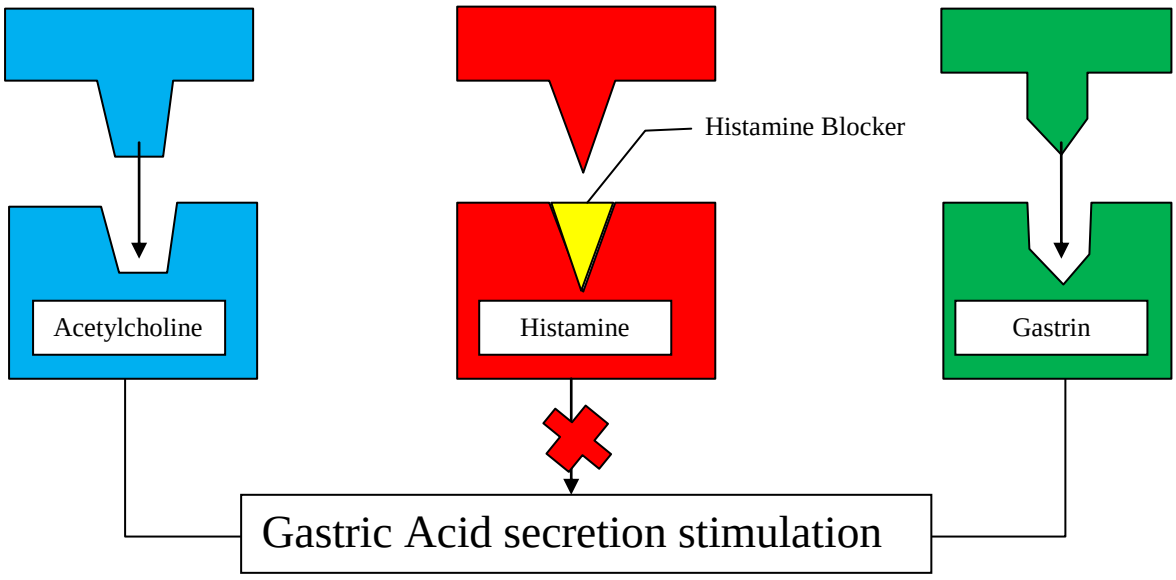
Classification and Mechanism of Action for Antihistamine Drug

Key Point to Understand- 'Histamine' is made up of two simple words- Histo (Tissue) + Amine. If we add them together the meaningful sentence will be- Amine released from tissue. Histamine is stored and release from mast cells. Other tissue like- Skin, gastric and intestinal mucosa, lungs, liver and placenta. Histamine receptors are of basically 02 types- (1) H1 and H2. H3 is also available. Histamine initiate allergic reaction thus antihistaminic drugs give relief from allergy by blocking any of the histamine receptor.



H_2 Receptor antagonist and regulation of gastric acid secretion

Gastric acid (Hcl) is secreted by the parietal cells from the mucosa of gastrointestinal tract, and that is stimulated by acetylcholine, histamine, and gastrin. The receptor medicated binding of acetylcholine, histamine, and gastrin result into activation of protein kinase which ultimately stimulate the H^+/K^+ ATP. Thus it is very simple that if someone is willing to inhibit the release of gastric acid he or she has to inhibit the binding of any of the above agent to their respective receptor. So the H_2 Receptor antagonist doesn't allow the agent to bind to the receptor and inhibit the release of gastric acid.



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