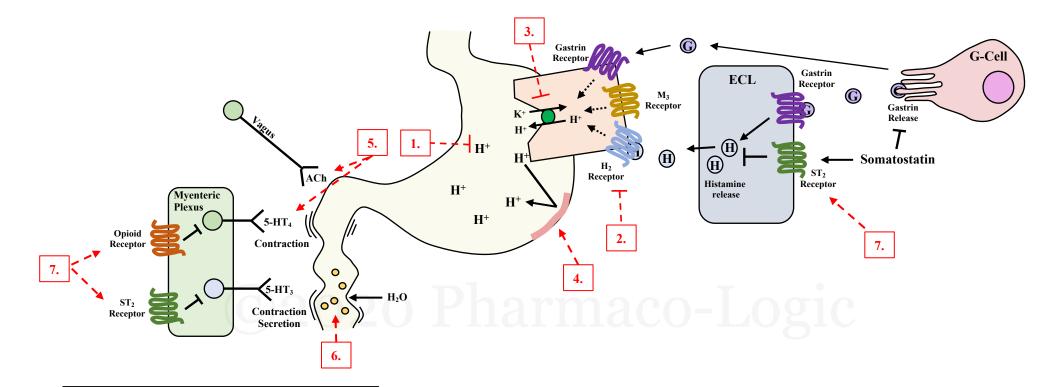
| 1. Antacids         MOA: Neutralize gastric acid, inhibit pepsin activity         Sodium Bicarbonate         Calcium Carbonate         - Hypercalcemia, Kidney stones, ↓ Drug absorption         (chelation), rebound acid <u>Aluminum Hydroxide</u> - Constipating <u>Magnesium Hydroxide</u> - Diarrhea         SE: Alkalization of the urine, ↓ drug absorption | <ul> <li>2. Antihistamines – H₂ receptor Antagonists         MOA: Inhibit parietal cell mediated acid production and release         USE: Peptic ulcer, gastritis, esophageal reflux – Night-time reflux      </li> <li><u>Cimetidine</u> – strong CYP inhibitor, Many DDI     </li> <li>SE: Antiandrogenic effects, gynecomastia, ↓ creatinine clearance, cross         BBB – dizziness, confusion, prolactinemia         <u>Ranitidine</u> </li> <li>SE: Thrombocytopenia, Decreased creatinine clearance     </li> <li><u>Famotidine</u>         Nizztidine – no subject to first pass metabolism     </li> </ul> | 3. Proton pump inhibitors<br>MOA: Irreversible inhibit H <sup>+</sup> /K <sup>+</sup> ATPase in the parietal cells<br>USE: Peptic ulcer, gastritis, esophageal reflux, Zollinger Ellison syndrome,<br>H. Pylori combo therapy<br><u>Omeprazole</u> – Prodrug<br><u>Esomeprazole</u> – Prodrug<br><u>Pantoprazole</u> – Prodrug<br><u>Lansoprazole</u> – Prodrug<br>SE: Risk for C. Diff infection, pneumonia, Hypomagnesemia, hypocalcemia,<br>reduced B12 absorption _ f_gastrin/ECL_production_Worsen Osteoporosis |
|--|--|--|
|  | <u>Nizatidine</u> – no subject to first pass metabolism  | reduced B12 absorption, ↑ gastrin/ECL production, Worsen Osteoporosis<br>DDI: Clopidogrel (CYP2C19 inhibition)   |



## 4. Mucosal Protectants

**MOA:** Coats and enhance mucosa protection from acid <u>Sucralfate</u> – creates a protective coat – not absorbed <u>Misoprostil</u> – PGE1 analog,  $\downarrow$  cAMP  $\rightarrow \downarrow$  acidic production **SE:** Diarrhea **CI:** pregnancy (abortive) <u>Bismuth Subsalisylate</u> – coats the GI tract – anti-inflammatory **SE:** Black tongue and stool, Constipation

### 5. Prokinetics

# MOA: Stimulate Enteric nervous system → GI motility

 $\begin{array}{l} \underline{\textit{Bethanechol}} - M_3 \text{ agonist} \\ \underline{\textit{Neostigmine}} - AChE \text{ inhibitor} \\ \underline{\textit{Metaclopramide}} - 5\text{-}HT_4 \text{ Agonist} \\ \textbf{SE: } D_2 \text{ antagonist - neuroleptic syndrome, Parkinsonian, EPS} \\ \underline{\textit{Domperidone}} - 5\text{-}HT_4 \text{ Agonist}/D_2 \text{ antagonist} \\ \underline{\textit{Cisapride}} - 5\text{-}HT4 \text{ Agonist} \\ \end{array}$ 

### 6. Laxatives

MOA: Stool softeners, increase GI fluid, Stimulate GI motility Bulk Forming Laxatives – *Psyllium, Methylcellulose* SE: Bloating, gas, Cramping, constipation Surfactant/Lubricant Laxatives – *Mineral oil, Docusate* SE: Diarrhea (mineral oil), allergy (Docusate), gas, cramping Osmotic Laxatives – *Magnesium hydroxide, Lactulose*–used for portal systemic encephalopathy → promotes NH<sub>4</sub>+ excretion

**SE:** Diarrhea, nausea, cramping, gas, dehydration **Stimulant Laxatives** – *bisacodyl, senna, castor oil,* **SE:** Cramping, incontinence, electrolyte imbalance

#### 7. Anti-diarrheals MOA: Decrease GI stimulation

 Opioids
 – Loperamide (does not cross BBB), diphenoxylate, Eluxadoline

 SE: Constipation – Diphenoxylate formulated with atropine

 CI: Inflammatory diarrhea

 Colloidal Bismuth – Coats GI tract

 Kaolin – Clay substance, indigestible

 Pectin – Gel forming polysaccharide, indigestible

 Cholestvramine, cholestipol – decreased absorption of fat-soluble drugs

 Octreotide
 – somatostatin analog – useful for GI tumors and AIDs

 SE: Decreased GI, pancreatic, biliary secretions, Hypoglycemia, cholelithiasis, Steatorrhea