

* Cephalic phase :- all secretion in this phase occur before entering the food into stomach. (preparing for receiving food)

* Things that stimulate this phase :-

see food / thinking about food / smell of food / taste of food.
all these things send signals to the brain to secrete gastric juice. (to hypothalamus) → send impulse to dorsal nucleus of vagus → impulse to stomach → give impulse to different cells of stomach (Chief cell, parietal cell ...) to produce gastric juice

* The main content of gastric juice is HCl & pepsin

* Things that inhibit this phase :- any thing stimulate sympathetic stress / emotional upset by (thoracic segment T1-T4) → inhibit secretion then to stomach vessel

* parietal cells secrete HCl * chief cells secrete pepsinogen.

* Gastric phase :- 2/3

* Stimulation of this phase :-

- 1) Distention (stretch) → when food enter the stomach after receptive relaxation → distention → that stimulate stretch receptor → that stimulate vagus nerve → brain → vagus nerve trigger HCl & pepsin production (Vago-Vagal reflex)
- 2) partially digested proteins → (afferent) → (efferent) motor

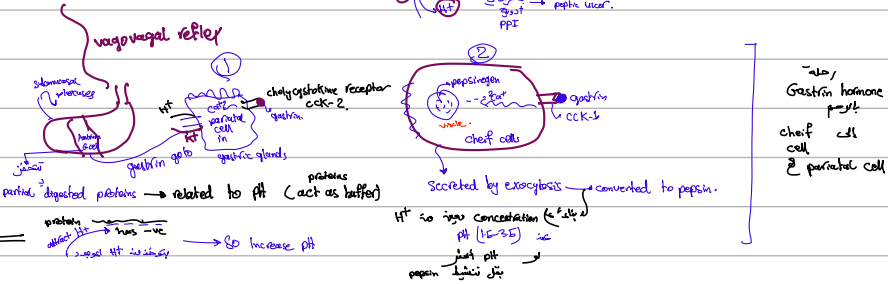
This can be assisted by submucosal & myenteric plexus (short reflex)

* Intraum → has special cell → Enterochromaffin G cells

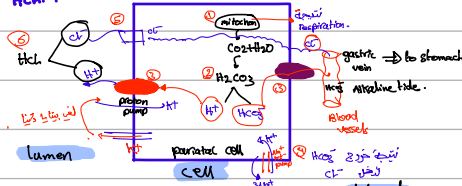
G cells → responded to partially digested proteins & then secrete Gastrin hormone

Gastrin travel through blood → Go to receptor (Cholecystikine-2 receptor) in parietal cell →

→ Increase Ca²⁺ level inside the cell → activate pump → pump into stomach → assist pepsin. PPI



* Formation of HCl :-



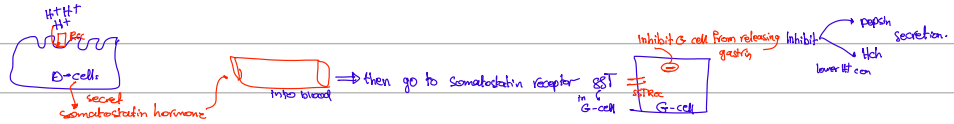
acidic

* Inhibitory of this phase: 1) sympathetic stimulation inhibit parasympathetic from production of gastric juice.

2) Somatostatin → secreted by Delta cell → stimulated in very low pH (H⁺)

* Delta cell (D-cell) in antrum is located.

if H⁺ concentration in lumen is high: =

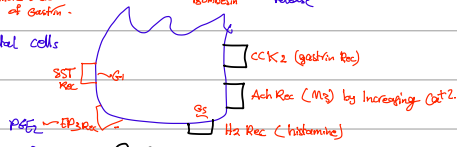


* If we don't need SST to inhibit gastric secretion - vagus nerve send signal to release ACh go to D cell → prevent release SST

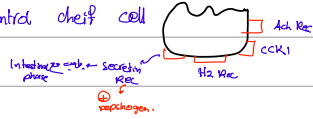
⊖ gastrin when it is released, go to D-cell & inhibit its work.



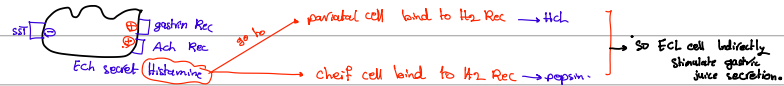
* Molecules that control parietal cells:

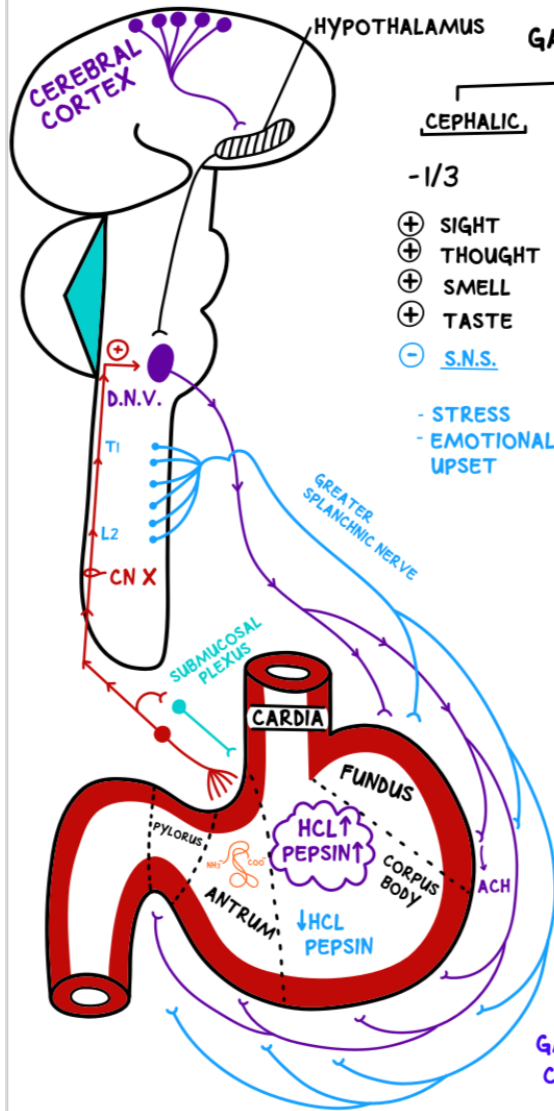


* Molecules that control chief cell:



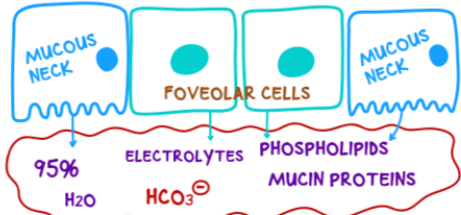
* ECL cell → enterochromaffin like cell → exist mostly in body of stomach.



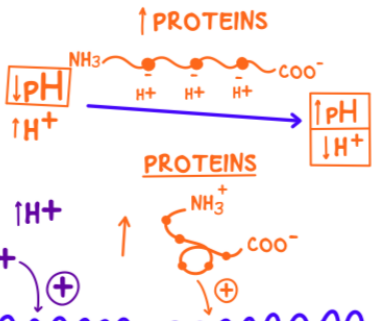


GASTRIC SECRETIONS

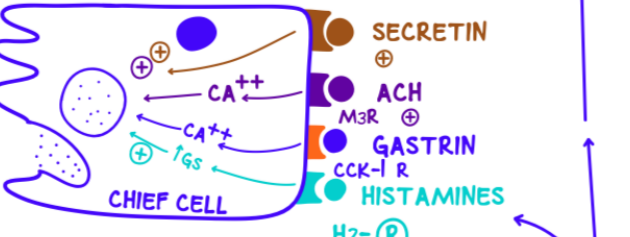
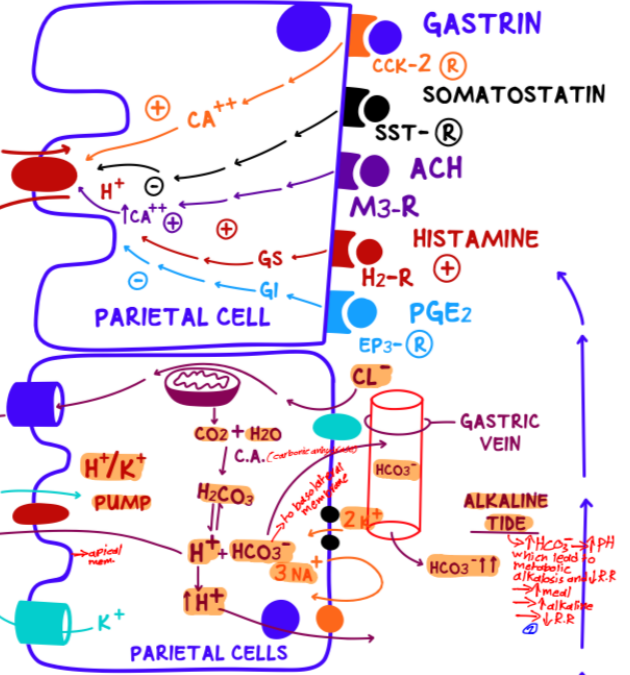
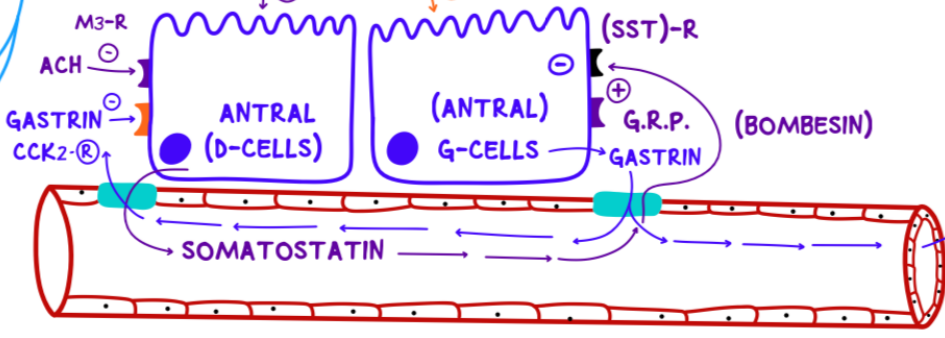
- CEPHALIC**
 - 1/3
 - ⊕ SIGHT
 - ⊕ THOUGHT
 - ⊕ SMELL
 - ⊕ TASTE
 - ⊖ S.N.S.
 - STRESS
 - EMOTIONAL UPSET
- GASTRIC**
 - 2/3
 - ⊕ DISTENTION (STRETCH)
 - ⊕ PARTIALLY DIGESTED PROTEINS
 - ↳ pH (RELATED)
 - ⊖ S.N.S.
 - STRESS
 - DEPRESSION
 - ANXIETY
 - ⊖ SOMATOSTATIN
 - ↓↓↓ pH (↑↑↑ H⁺)
- INTESTINAL**



MUCOSAL BARRIER
PREVENTS CORROSIVE DAMAGE BY HCL AND PEPSIN



(ACTIVE) PEPSIN
 ↑ H⁺ ⊕
 pH 1.8-3.5
 (INACTIVE) PEPSINOGEN
 ⊖



Q1) why you feel tired after having a big meal?

لما نتناول كمياته كبيرة منه الطعام ربح يزيد تركيز (H^+) في المعدة و لكن الدم اللي حوالينها المعدة ربح يزيد تركيز HCO_3^- فيه

هنا فيه عينا (chemoreceptors) موجودة في (Heart) او في (brain) وهذا (receptor) بجموع بتغير (pH) فلما يزيد (HCO_3^-) في الدم ربح ^{يقول} يزيد (respiratory rate) فبميسر مع الشخص (hyperventilation) فزيد (CO_2) وبقه (O_2) فبميسر عند الشخص (respiratory acidosis)

منه العاليه ما بتساعد على العضم وفيها
pepsinogen ما يتحول الى (pepsin)

برضو لما نتناول كمياته كبيرة من الطعام ربح تتدبر المعدة وتضغط على (diaphragm) فبتقل مساحتها (Thoracic cavity) فبتقل الشخص قادر يتنفس

Q4) what is the mechanism of drug that is given for treatment of (GERD)?

omeprazole → inhibit hydrogen-potassium pump to decrease stomach acidity

Histamine blockers (H_2 blockers) → block (H_2 receptors)

prostaglandine → inhibit hydrochloric acid production

Q2) what is the enzyme that is working in very acidic media?

pepsin

Q3) what kind of drugs that inhibit secretion and motility?

somatostatin

Q5) you have two patients: The first one given glucose orally and the second (IV)

who is the first that is going to have higher insulin in the blood??

في حال (IV) سيمه (glucose) لازم يروح للقلب عشان يضغط لباقي اعضاء الجسم اعليك استجابة الي انسولين بتوخذ وقت

في حال (orally) سيمه (glucose) بيمه مباشرة ل (GIT) والي بحتوي على (K cell)

صنه (K cell) بتفرز (glucose dependent insulinotropic peptide) والي بيوره بيمه تحفيز ل (insulin)

برضو افراز (gastrin) بحفز كمية قليلة من (insulin)

اعليك المريض الي بنه طبخ (glucose) عن طريقه العضم يرتفع عنده (insulin) اسرع

ملاحظات

بتقدر تعطى (somatostatin) في حال كان عند المريض (somatostatinoma) لانه بقدر يثبط نفسه