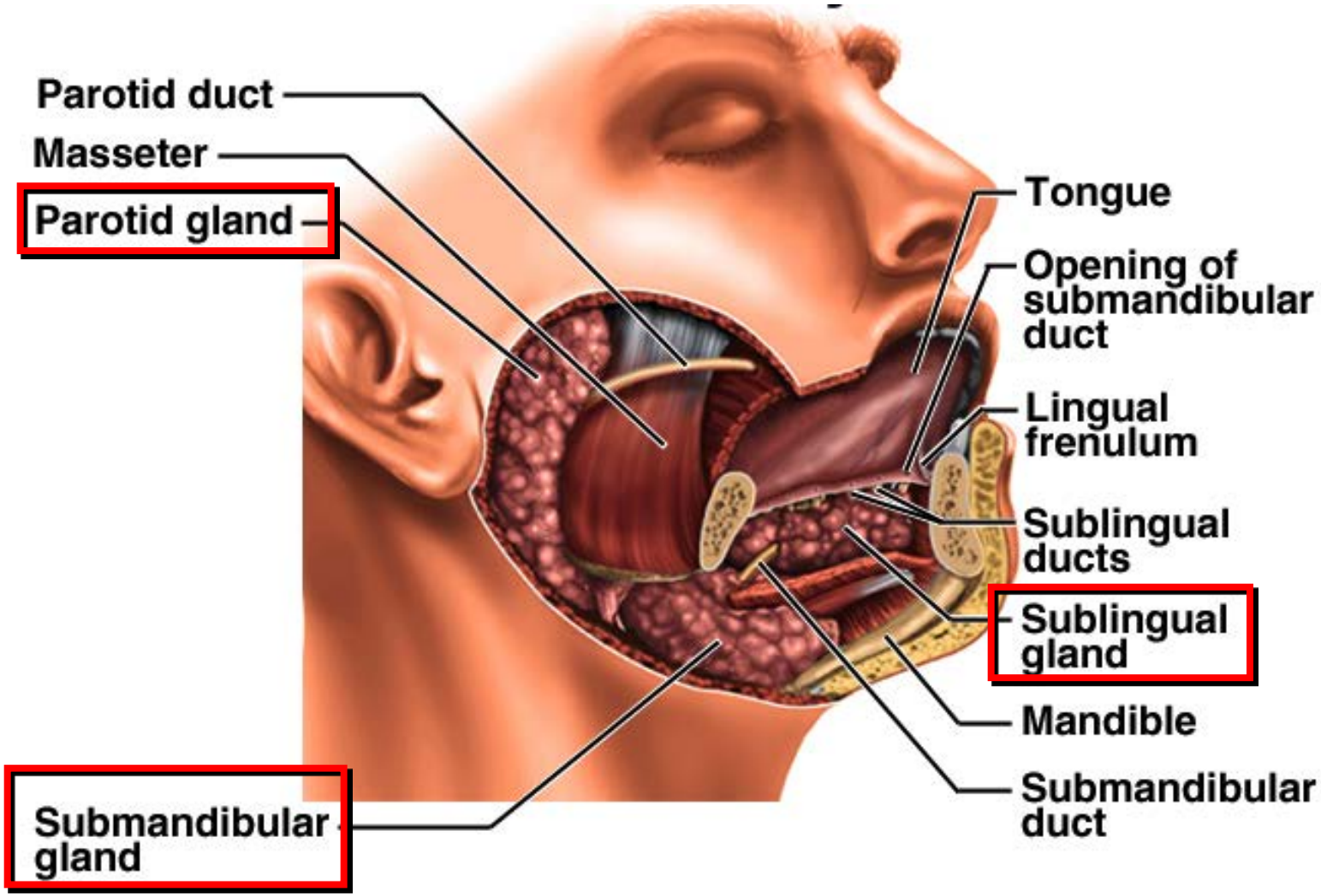
SECREÇÃO SALIVAR

Relação de *algumas* drogas que inibem a salivação

- anti-colinérgicos
- anti-adrenérgicos
- anti-depressivos
- anti-psicóticos
- ansiolíticos
- sedativos
- anti-histamínicos
- anti-parkinsonianos
- anti-hipertensivos
- diuréticos
- anti-espasmódicos
- anti-prostáticos
- mio-relaxantes
- anti-inflamatórios não esteroidais
- analgésicos
- vasodilatadores
- anti-acne
- anti-lipemicos
- anti-eméticos
- anti-diarreicos
- antibióticos
- anti-epiléticos
- anti-ulcerosos
- vitaminas

Douglas-Cisternas, 2004

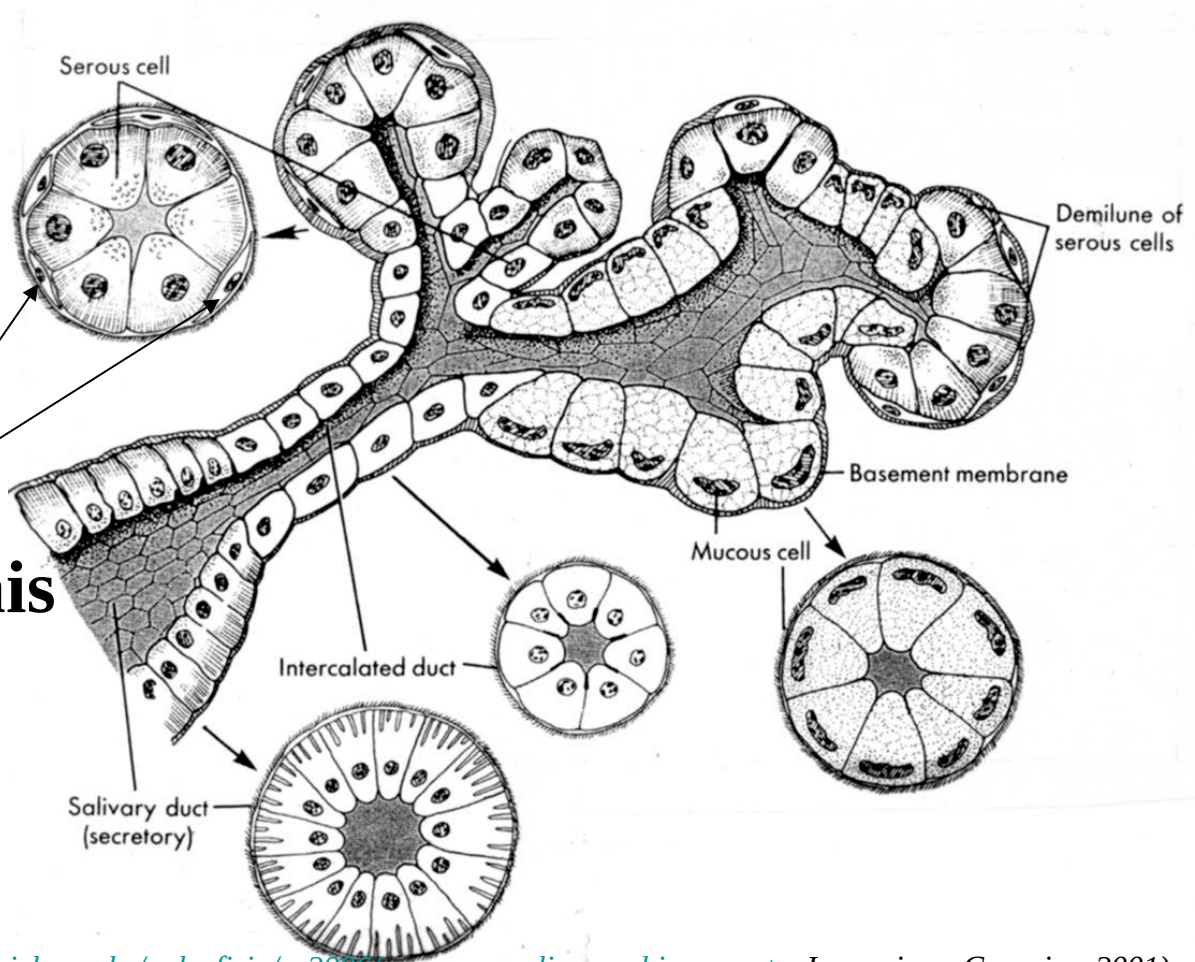
Glândulas salivares



saladin, 2001

Histologia das glândulas salivares anexas

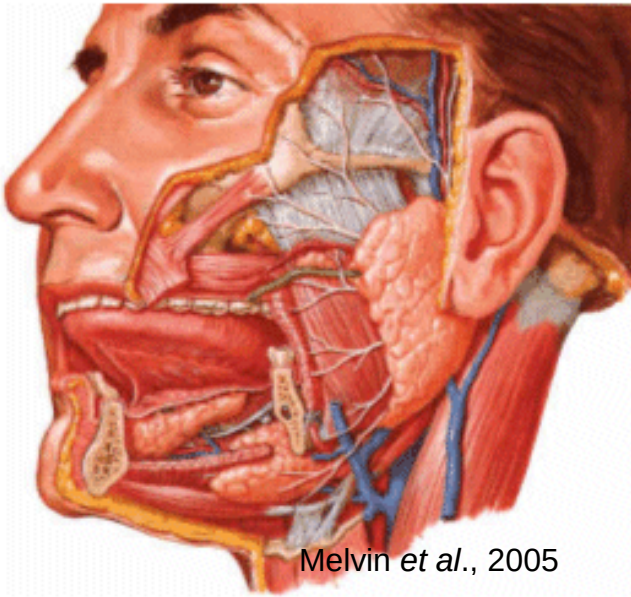
- **Células acinares**
- **Células ductais**
- **Células mioepiteliais**



http://www.fisio.icb.usp.br/aulasfisio/cv2006/secrecao_salivar_robinson.ppt - Junqueira e Carneiro, 2001)

Informações gerais sobre a saliva

- Produção diária: 1,0 - 1,5 L
- pH: 6,8-7,2
- Hipotônica em relação ao plasma
- Regulação exclusivamente neural



Melvin *et al*., 2005

A multifuncionalidade da saliva

adapted from M.J. Levine, 1993 http://www.umich.edu/~bmsteach/logatin/salivarygland/lectures/download/Chem_Comp_&_Funct.ppt

XEROSTOMIA CONGÊNITA

- Boca seca
- Cáries
- Infecções da mucosa (Periodontites, gengivites, candidíase, abscessos, etc.)
- Síndrome da ardência bucal
- Edentulismo *(perda dos dentes)*

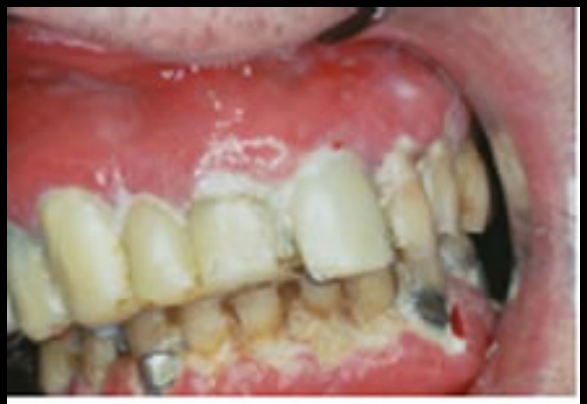
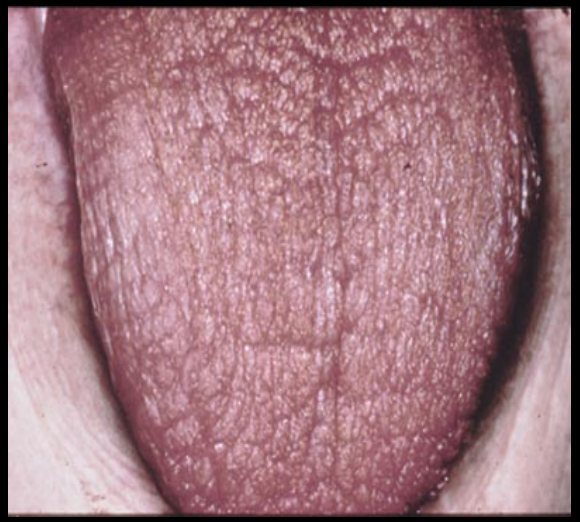
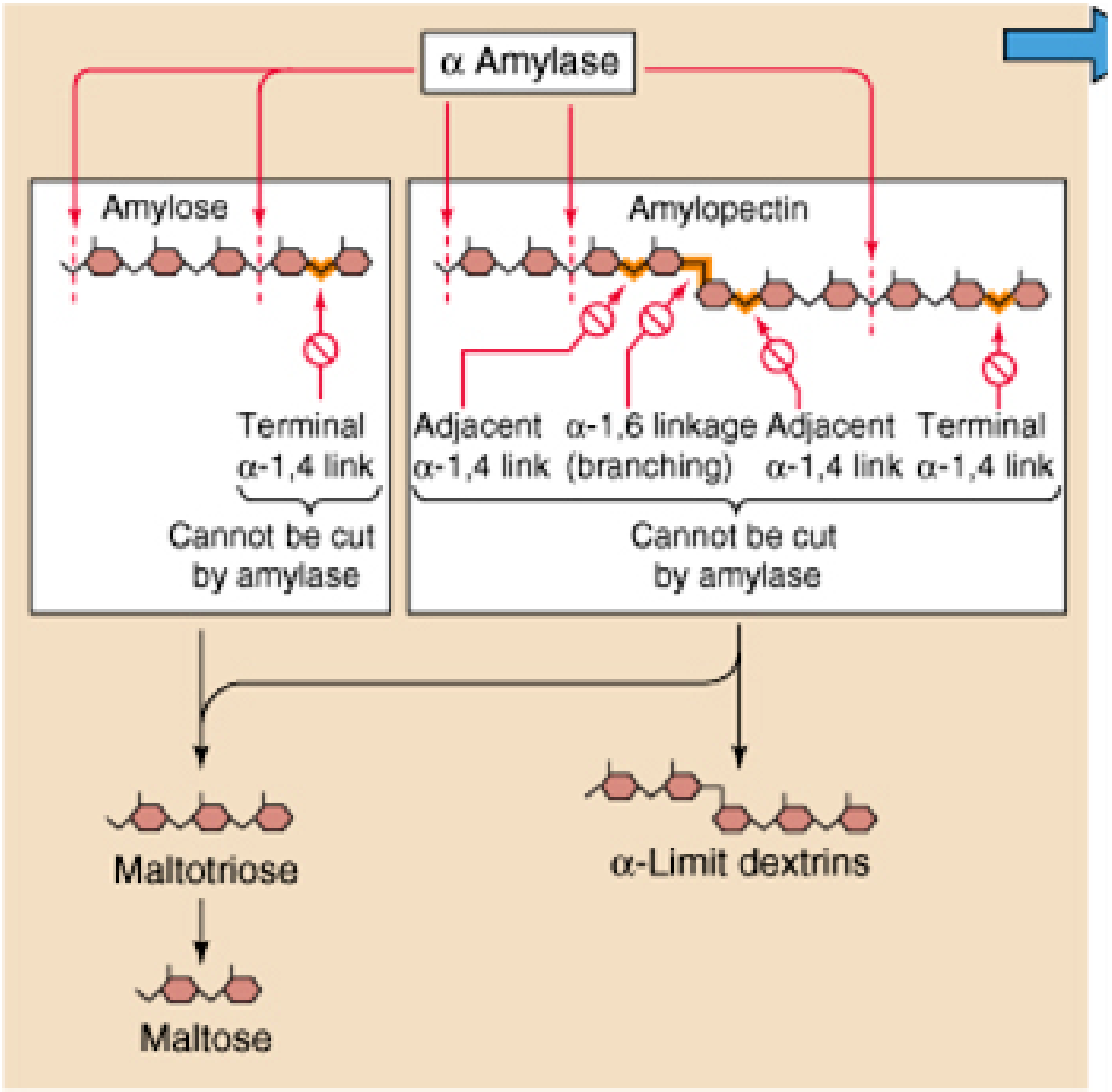


Figure 5. Oral debris accumulation resulting from xerostomia and other predisposing factors.

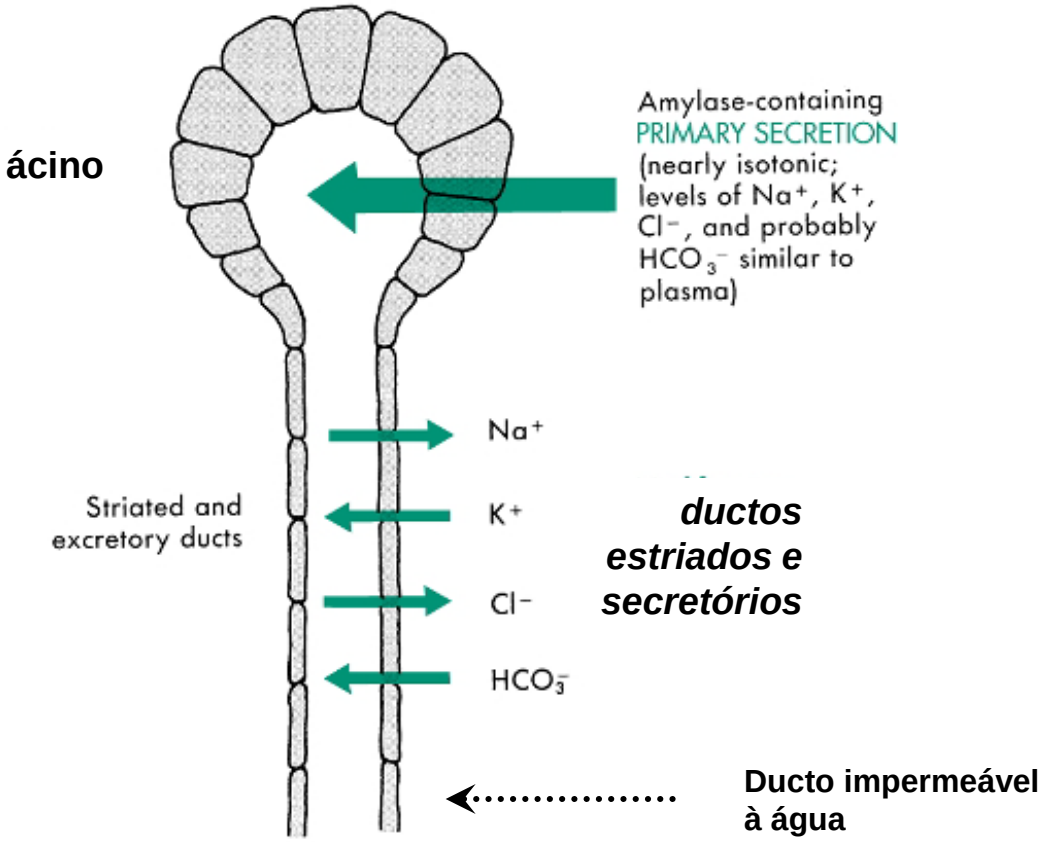


AMILASE SALIVAR ou PTIALINA

pH 6,9
Boca: 3-5%
Estômago 25-30%



Secreção Salivar: 2 estágios

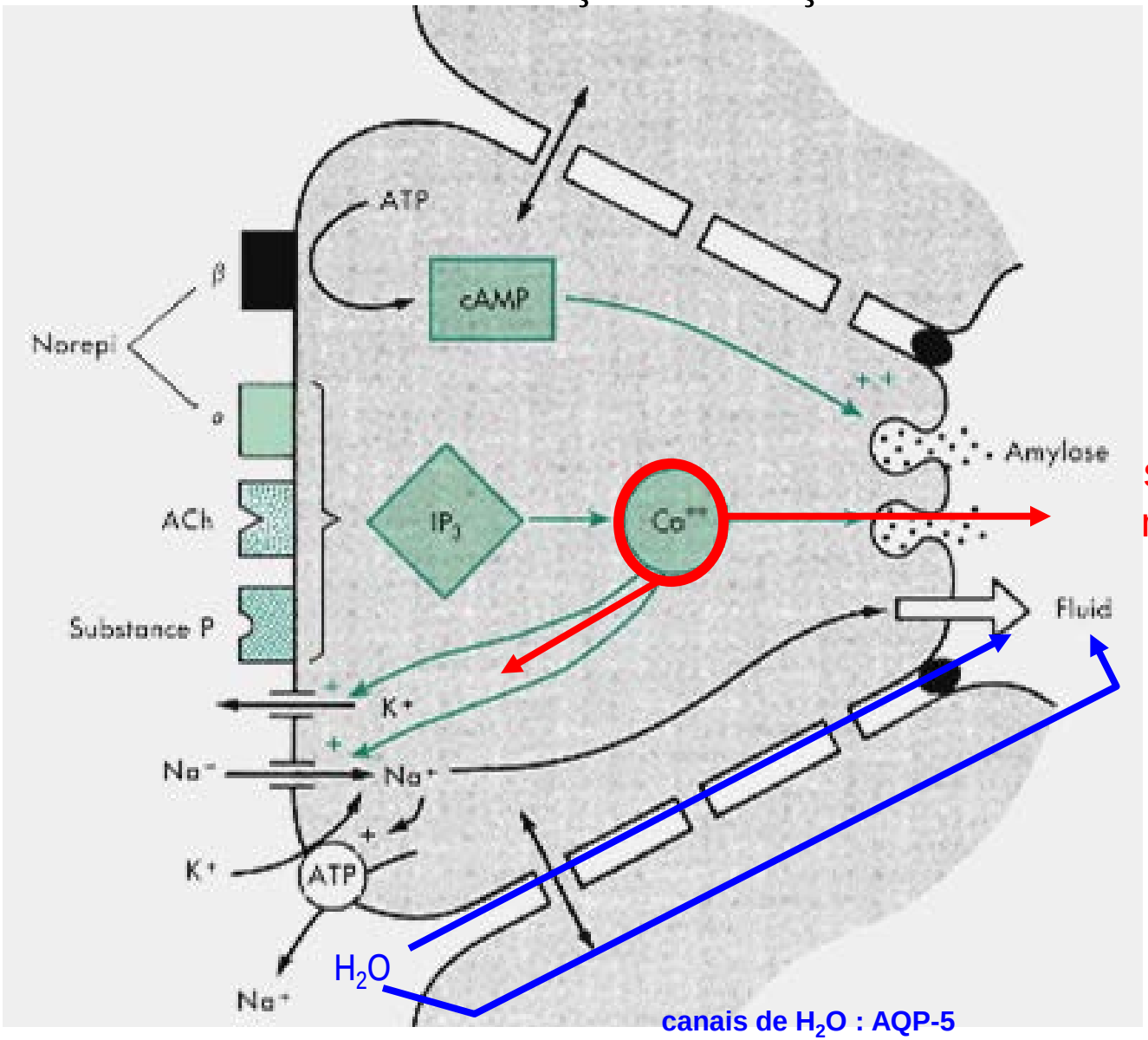


Berne et al., 2004

Mecanismos celulares de estimulação da secreção salivar nos ácinos

SIMPA

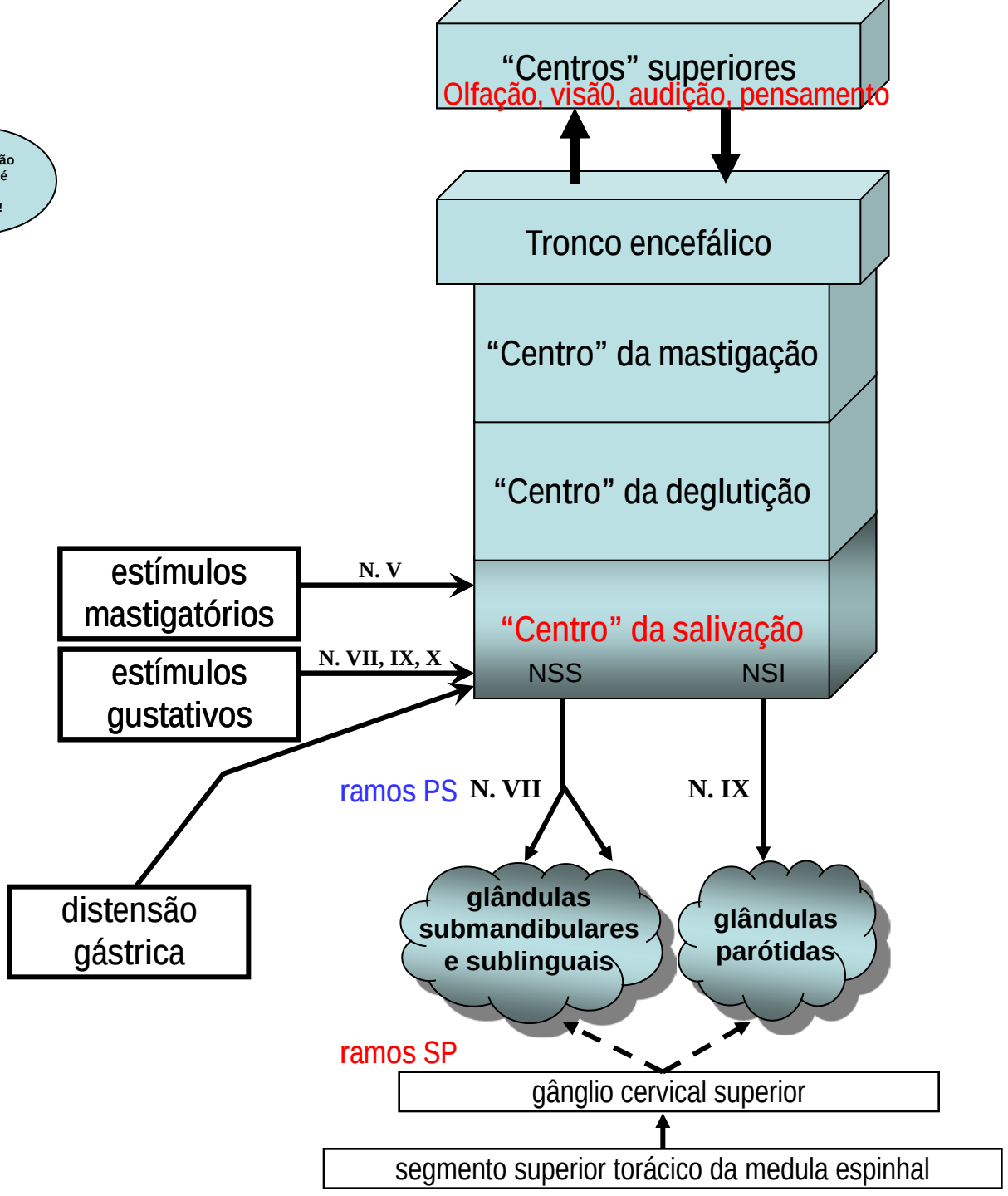
PARA



secreção maior em volume

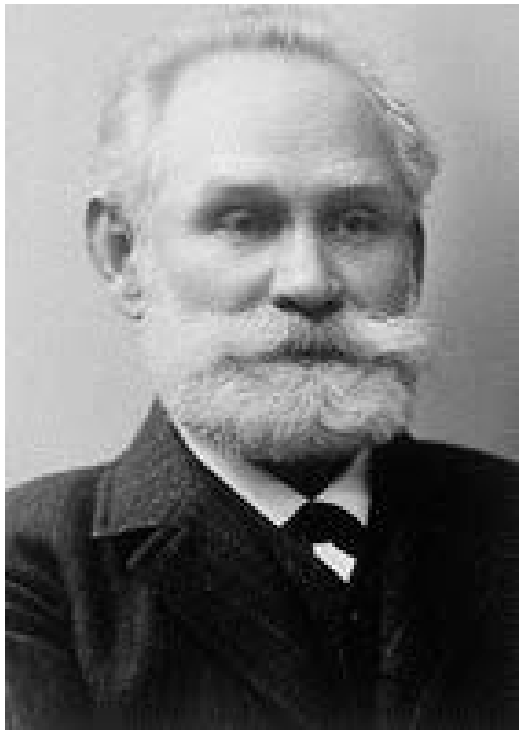
Fig. 32-7 The cellular mechanisms whereby norepinephrine (Norepi), acetylcholine (ACh), and substance P evoke salivary secretion. Norepinephrine acting onα-adrenergic receptors, acetylcholine, and substance P increases intracellular Ca2+. Norepinephrine acting on β-adrenergic receptors increases intracellular levels of cyclic AMP (cAMP). Effectors that increase cellular cAMP elicit a primary secretion that is richer in amylase than is the secretion evoked by agents that increase intracellular Ca2+. **Substances that increase intracellular Ca2+ produce a greater volume of acinar cell secretion than do agonists that increase intracellular cAMP.** (From Peterson OH. In Johnson RL, editor: Physiology of the gastrointestinal tract, New York, 1981, Raven Press.) Berne et al, 2004

I-OLFATÓRIO
II-ÓPTICO
III-OCULOMOTOR
IV-TROCLEAR
V-TRIGÊMEO
VI-ABDUCENTE
VII-FACIAL
VIII-VESTÍBULO-COCLEAR
IX-GLOSSOFARÍNGEO
X-VAGO
XI-ACESSÓRIO
XII-HIPOGLOSSO

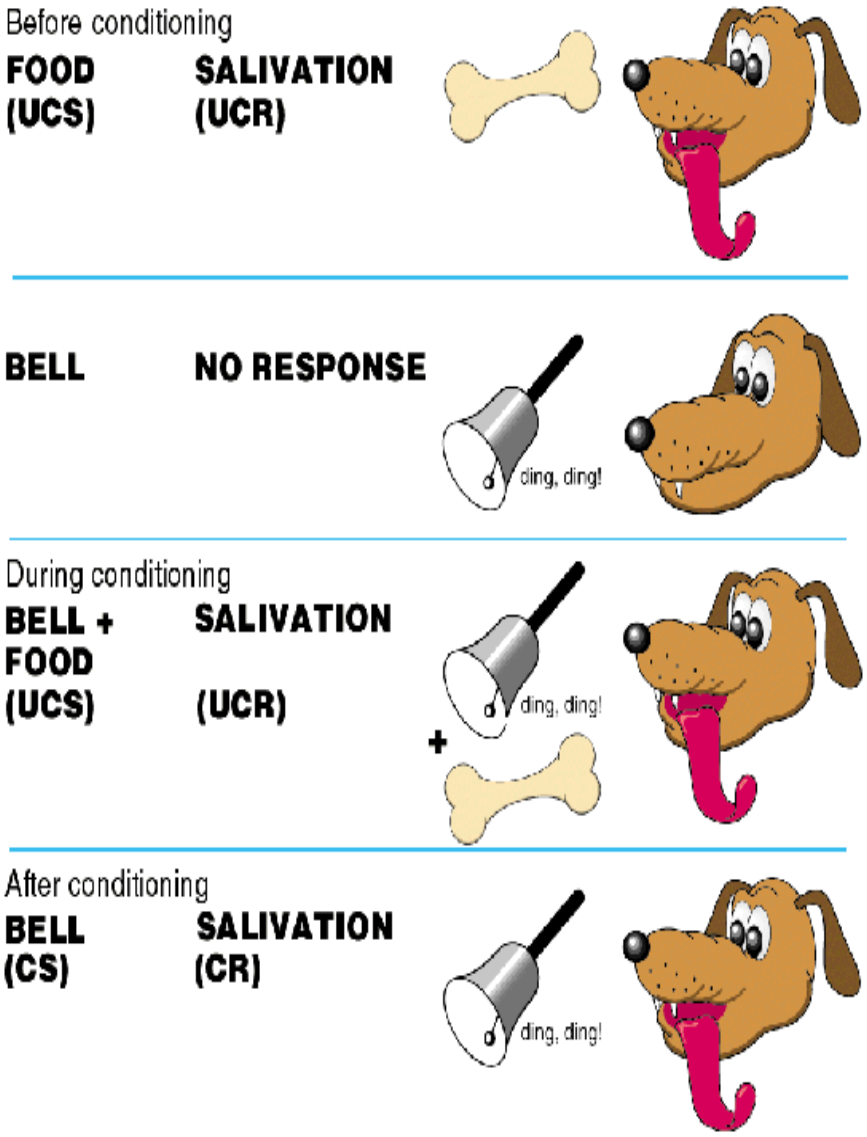


Reflexos condicionados

Pavlov recebeu o Prêmio Nobel em 1904 de Fisiologia e Medicina, por suas pesquisas.

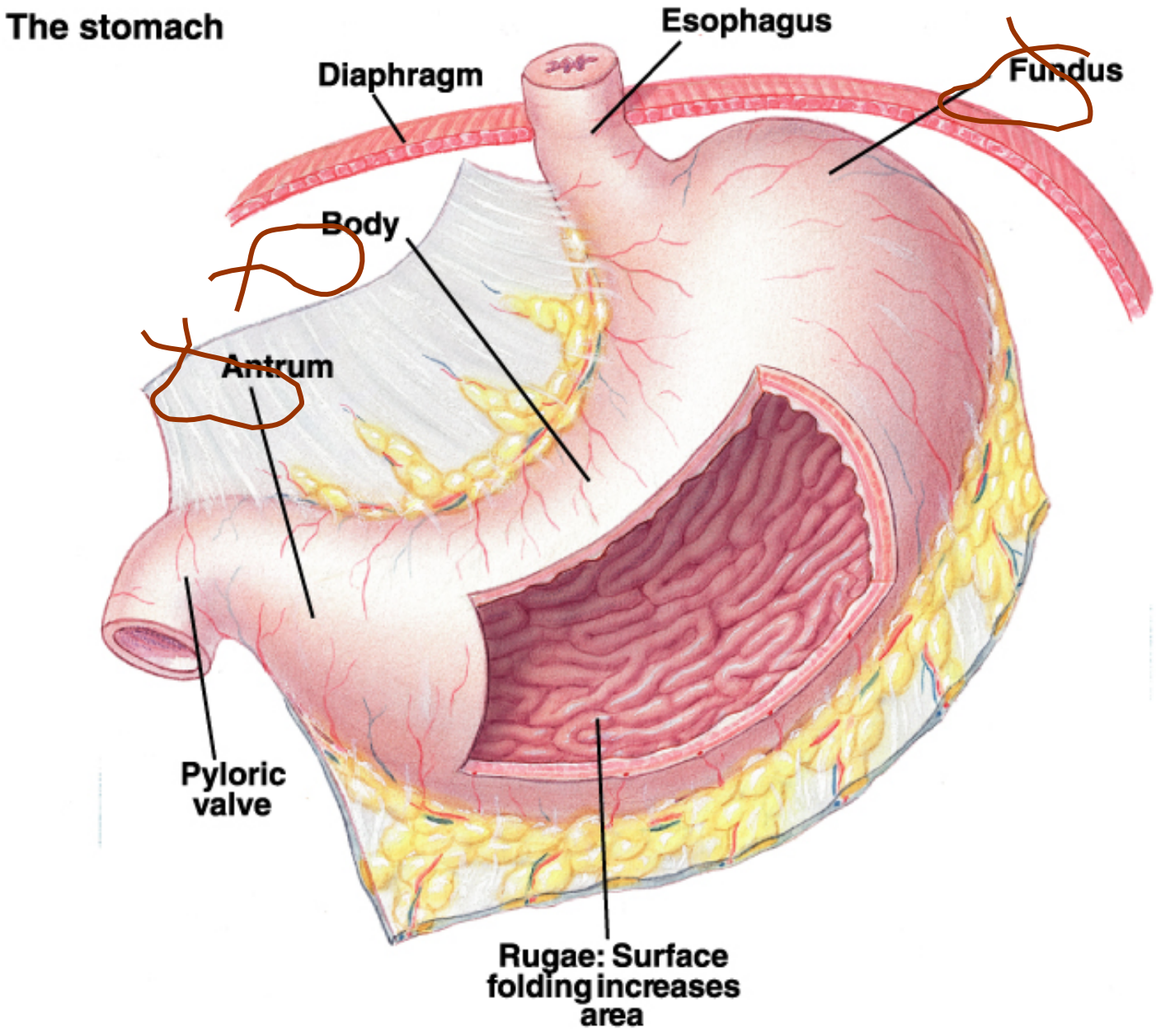


Ivan Petrovich **Pavlov** (1849 - 1936)

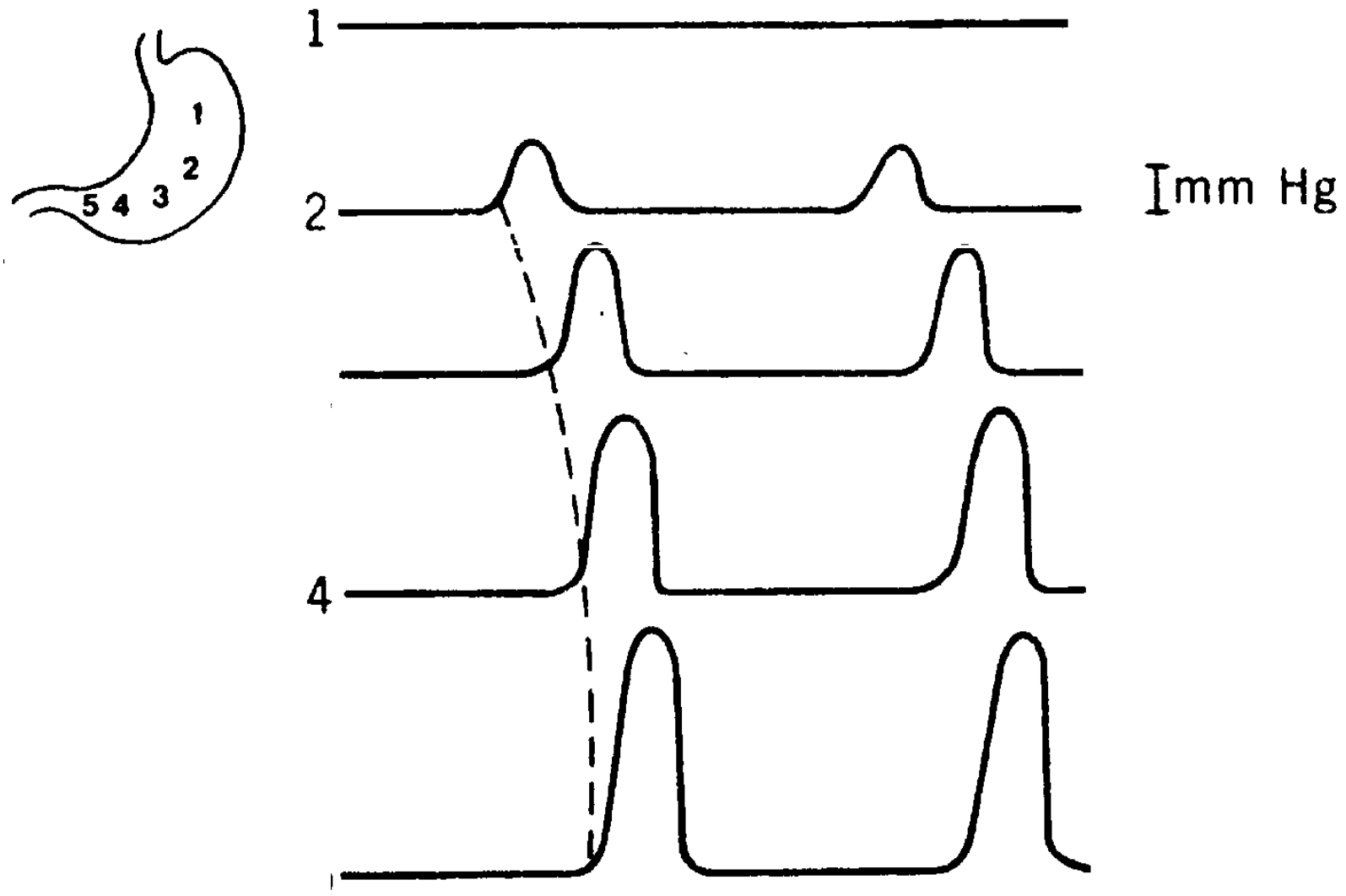


http://nobelprize.org/medicine/laureates/1904/pavlov-bio.html

MOTILIDADE GÁSTRICA

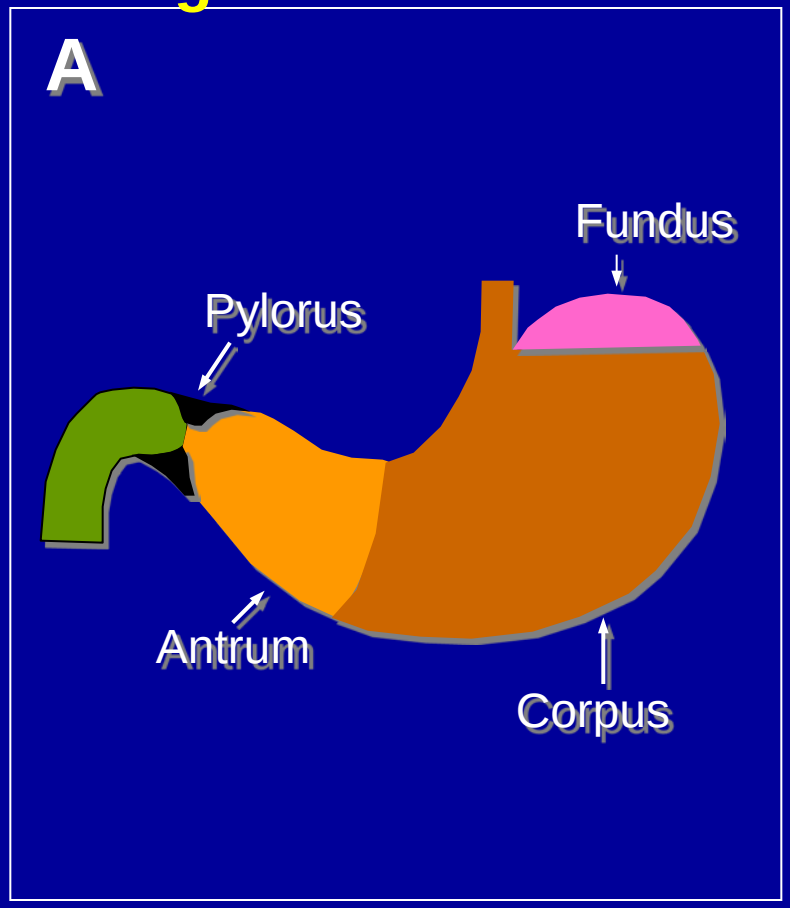


REGISTRO DA ATIVIDADE CONTRÁTIL DO ESTÔMAGO

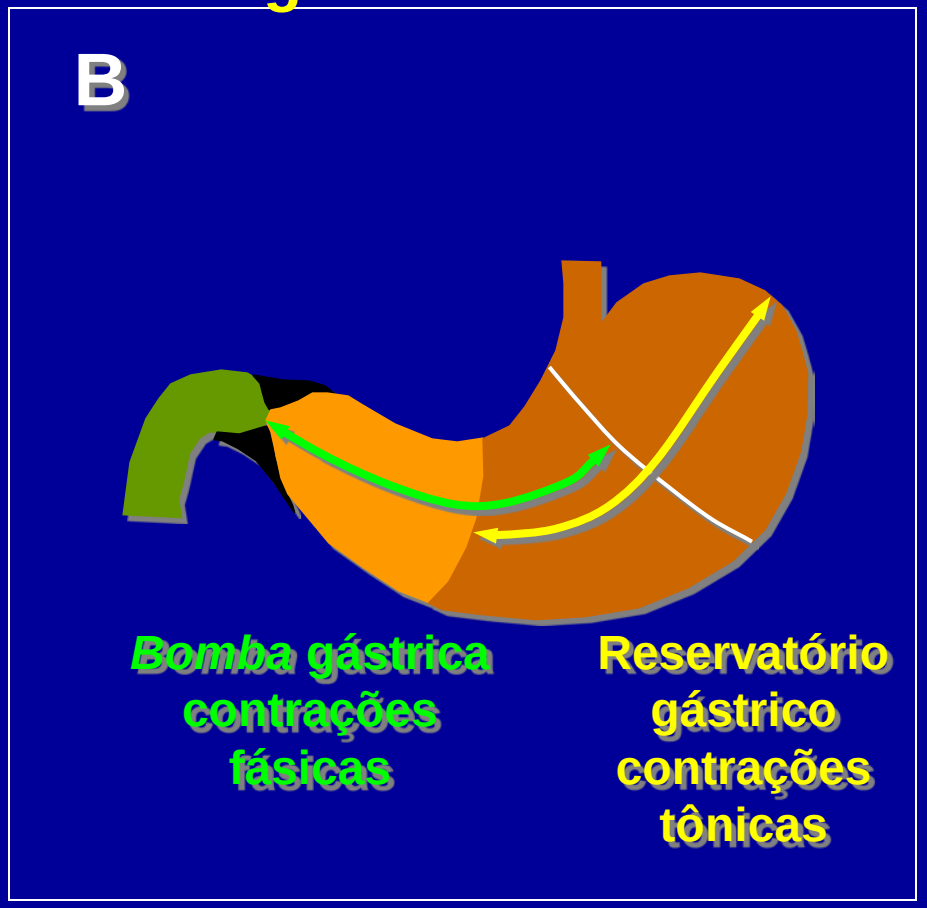


O estômago pode ser dividido em:

3 regiões anatômicas



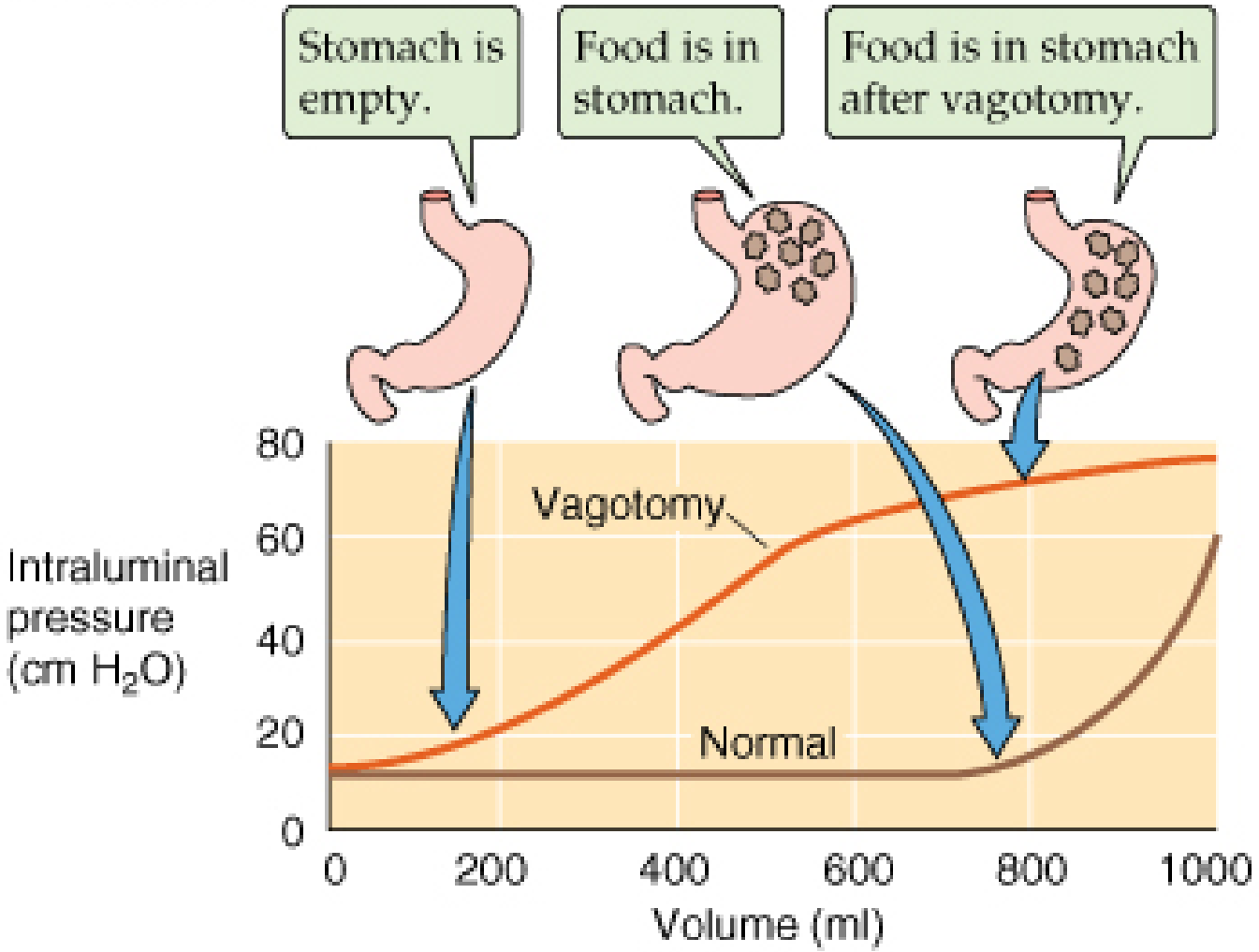
2 regiões funcionais



website original: http://www.wzw.tum.de/humanbiology/data/motility/34/?alt=english

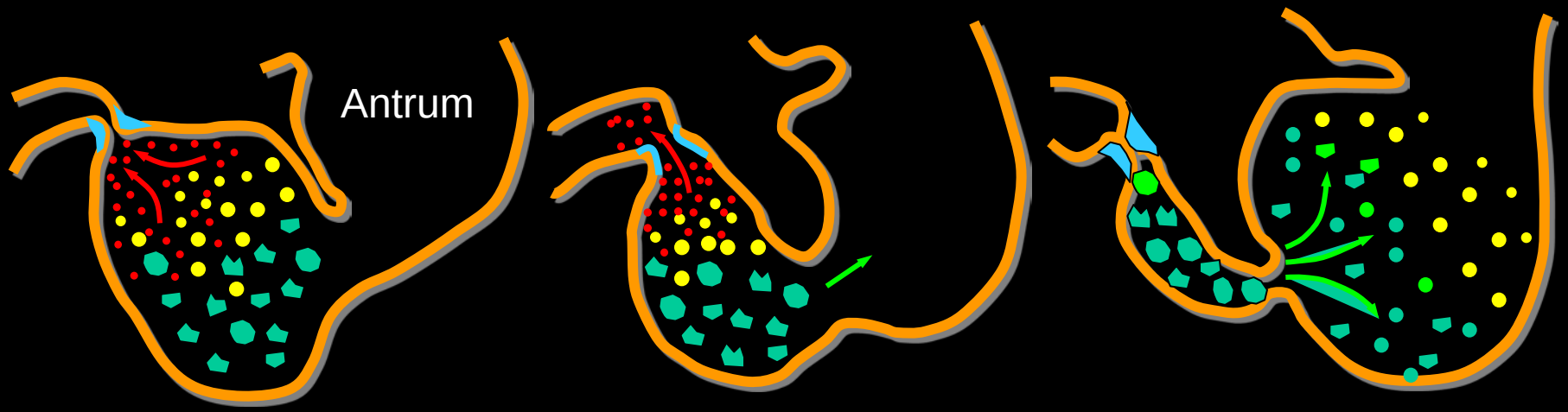
Relaxamento gástrico

(receptivo e adaptativo)



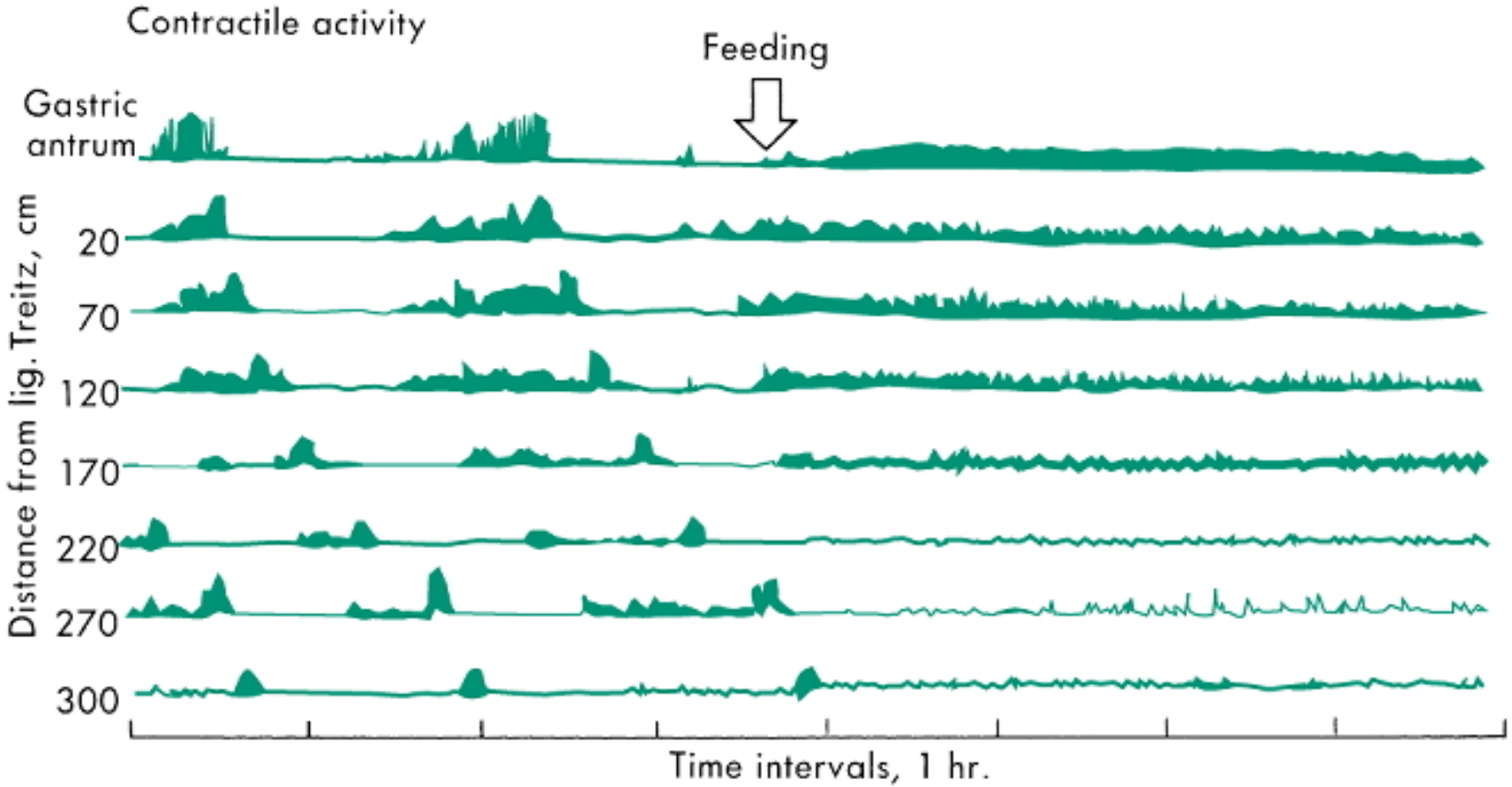
Antro gástrico: Função de propulsão, esvaziamento e moagem

Fase de propulsão **Fase de esvaziamento** **Fase de retropulsão**

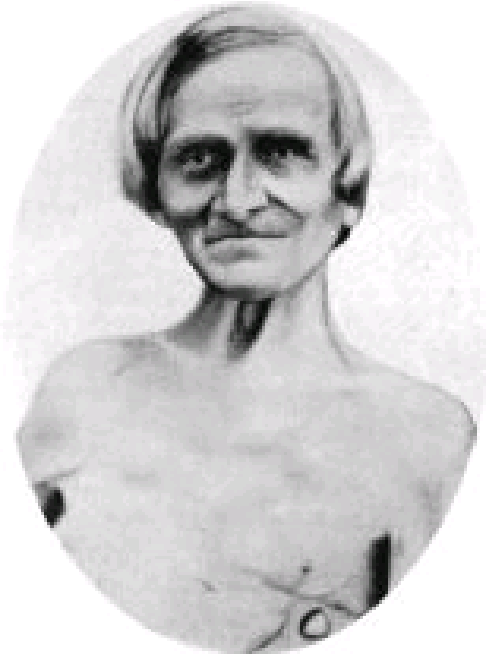


website original: *http://www.wzw.tum.de/humanbiology/data/motility/34/?alt=english*

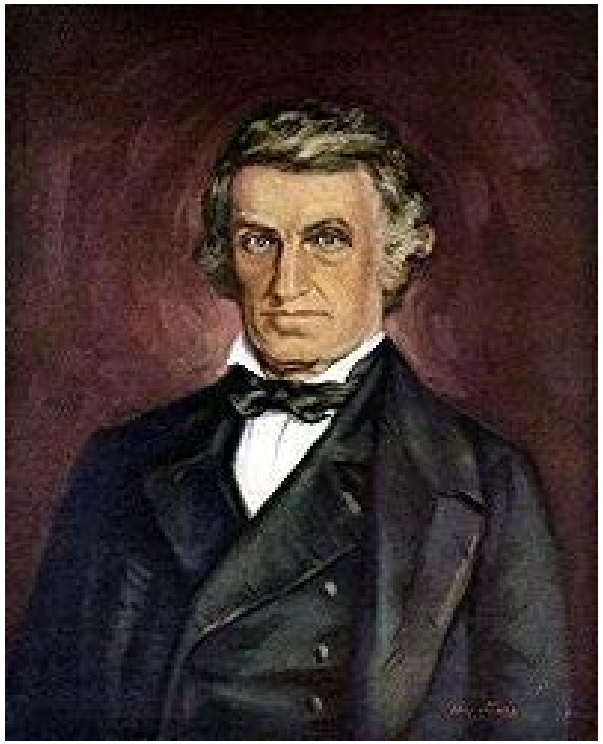
Motilidade durante o jejum:
complexo migratório mioelétrico (CMM, estômago ☞ Íleo terminal)



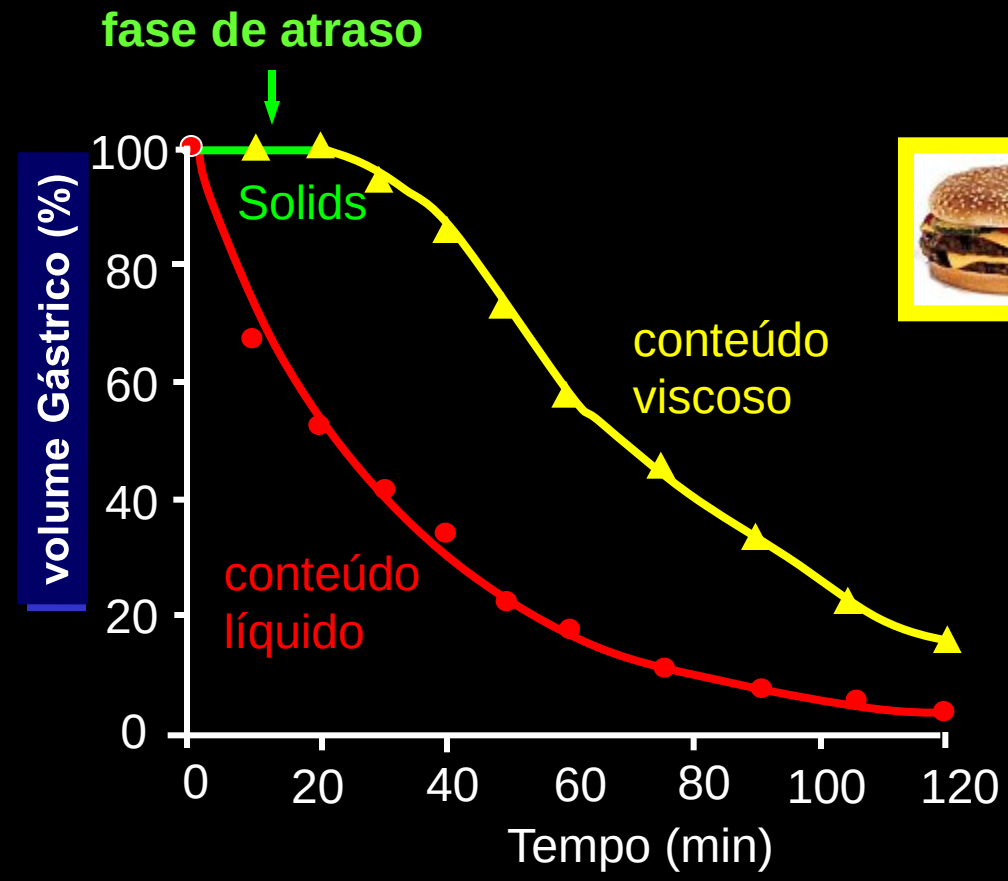
O pai da Fisiologia Gástrica



Alexis St Martin

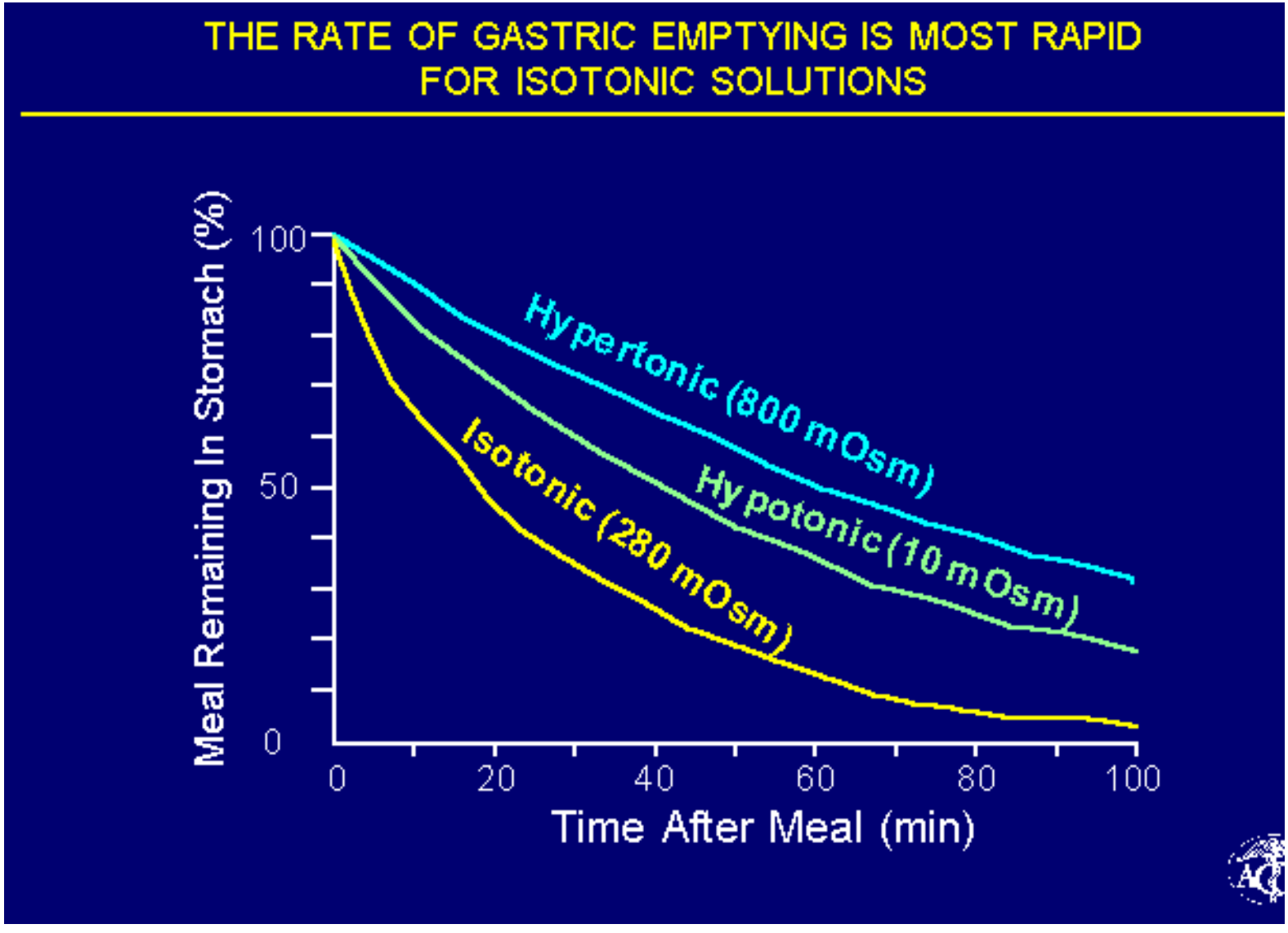


Willian Beaumont

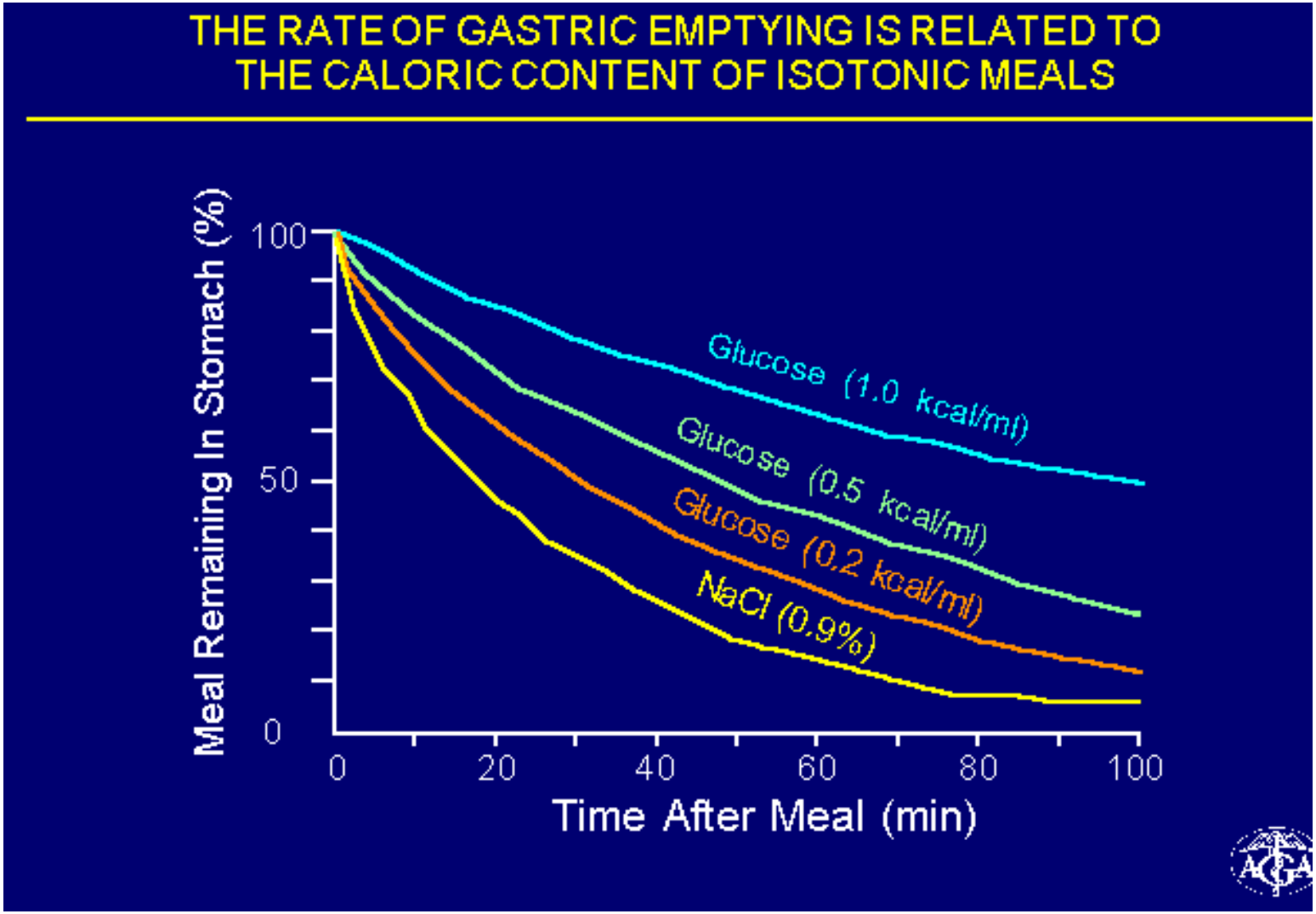


website original: http://www.wzw.tum.de/humanbiology/data/motility/34/?alt=english

Regulação esvaziamento gástrico



Regulação esvaziamento gástrico



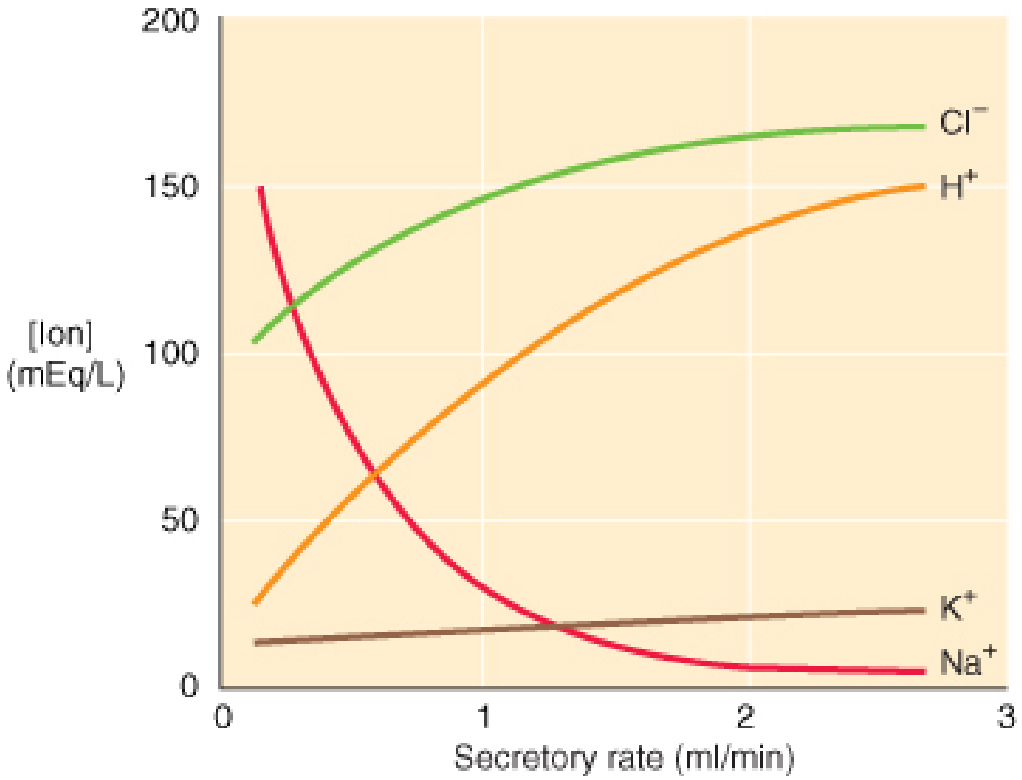
Importância da regulação do esvaziamento gástrico

1-Permitir um esvaziamento regulado dos conteúdos gástricos a uma velocidade consistente com a capacidade de processamento do quimo pelo duodeno.

2-Evitar a refluxo dos conteúdos duodenais.

SECREÇÃO GÁSTRICA

Dois tipos de secreção gástrica:
Não Parietal
e
Parietal



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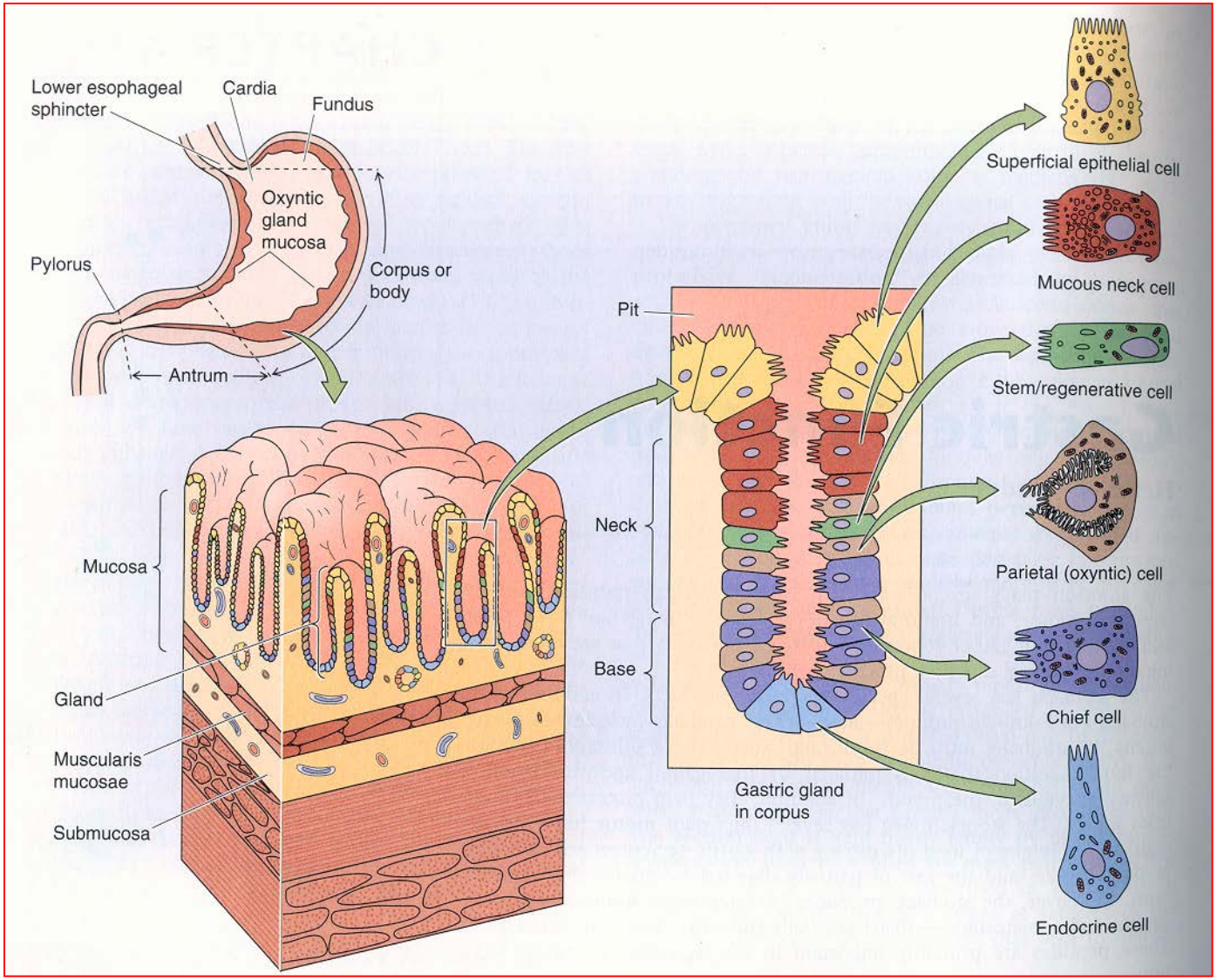
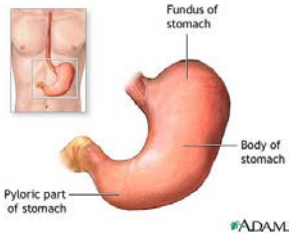
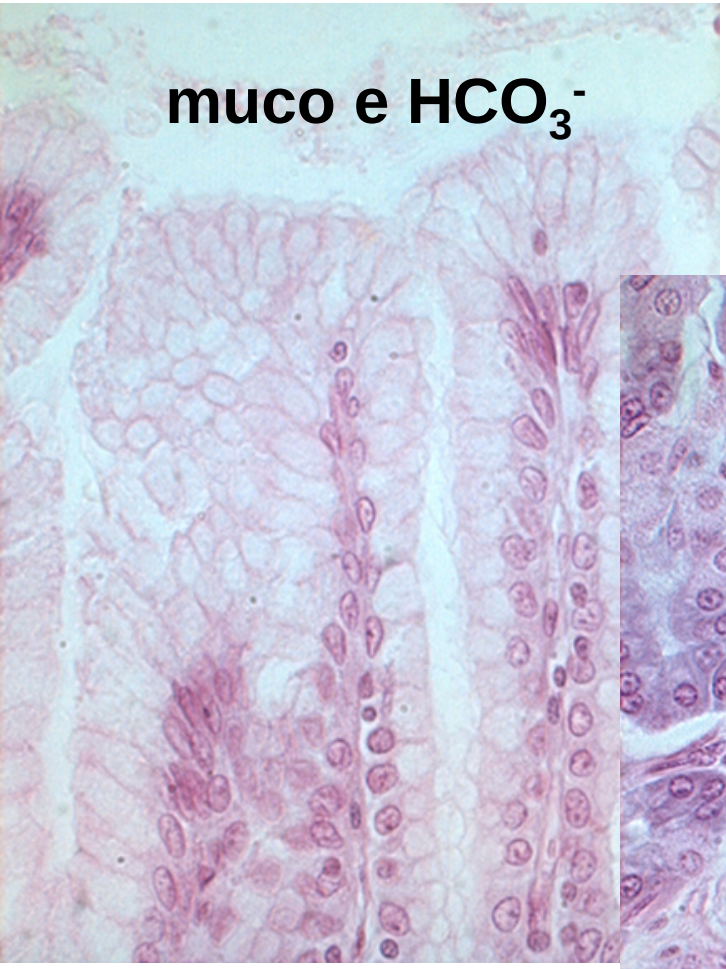

Lower esophageal sphincter
Cardia
Fundus
Oxyntic gland mucosa
Pylorus
Corpus or body
Antrum
Mucosa
Gland
Muscularis mucosae
Submucosa
Pit
Neck
Base
Gastric gland in corpus
Superficial epithelial cell
Mucous neck cell
Stem/regenerative cell
Parietal (oxyntic) cell
Chief cell
Endocrine cell

SECREÇÕES EXÓCRINAS



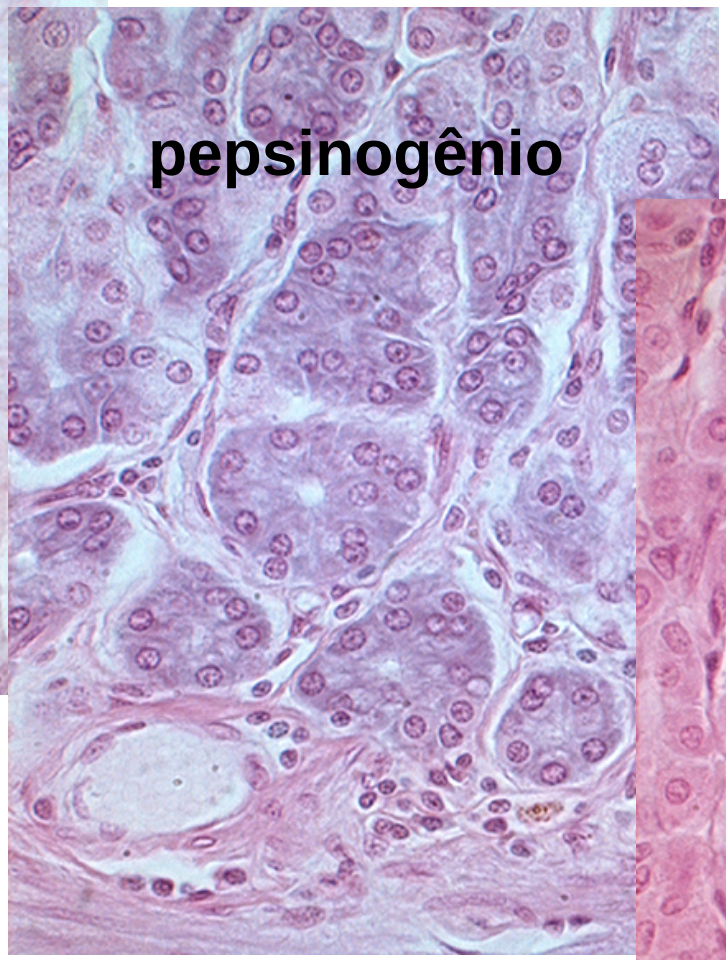
células mucosas

muco e HCO_3^-



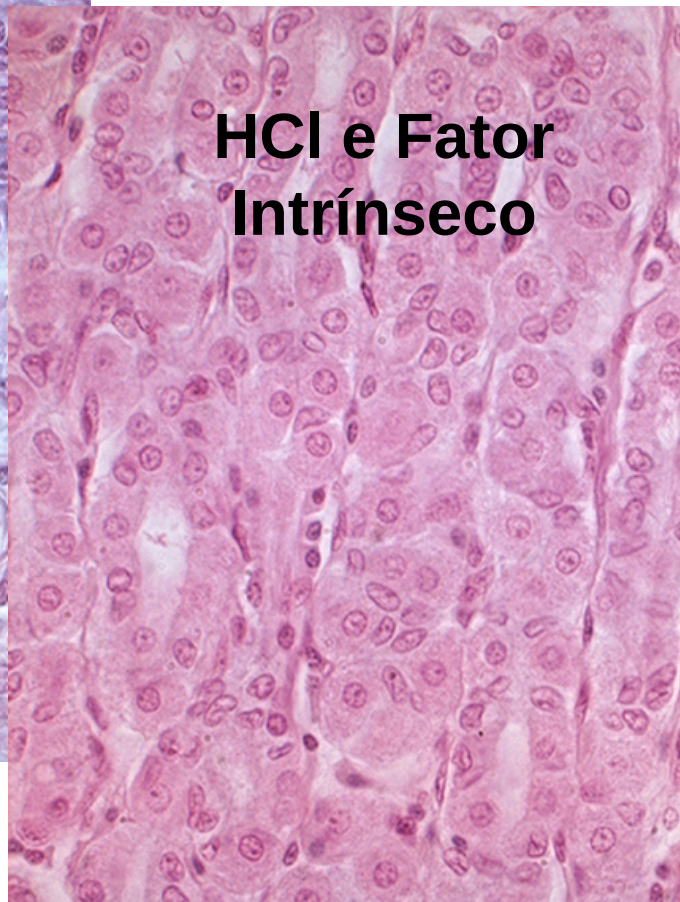
células principais

pepsinogênio



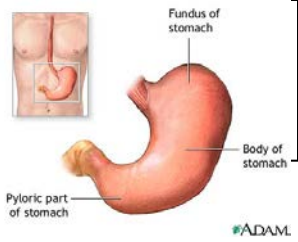
células parietais

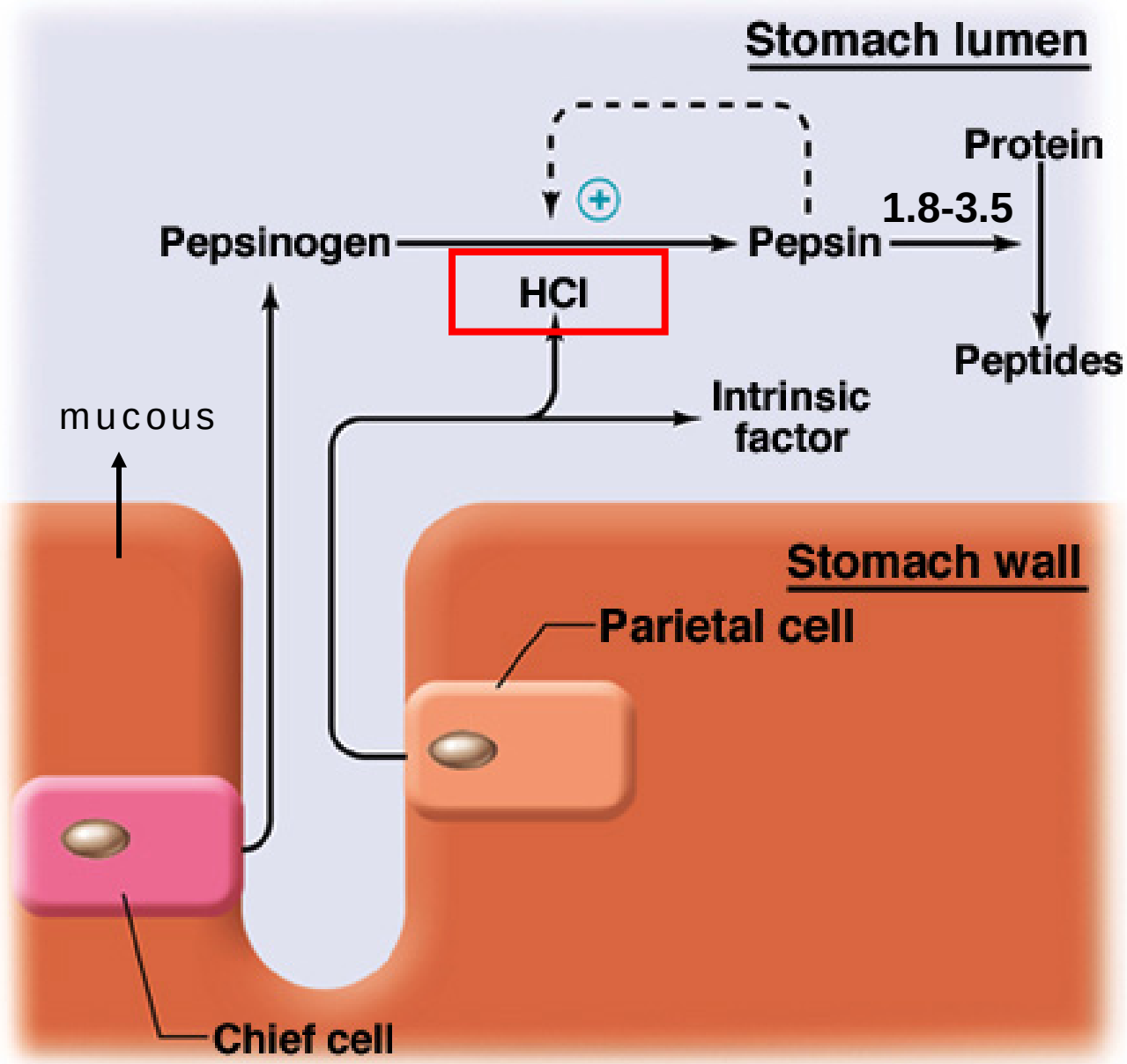
HCl e Fator Intrínseco



http://anatomy.iupui.edu/courses/histo_D502/D502f03/f03_labs/Lab11/Lab11f03.html

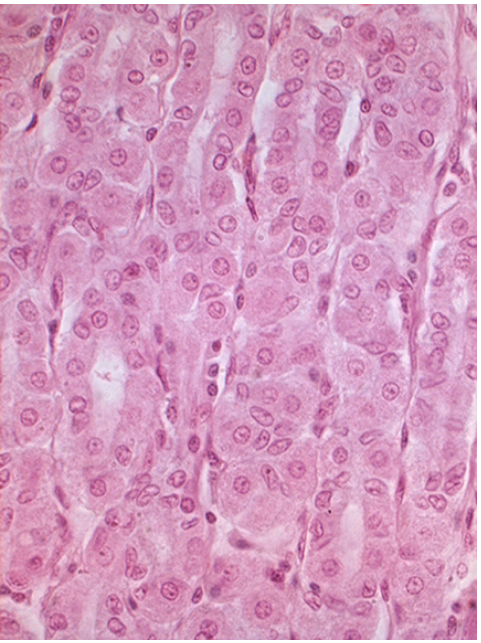
Interações das secreções gástricas

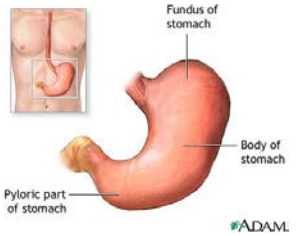




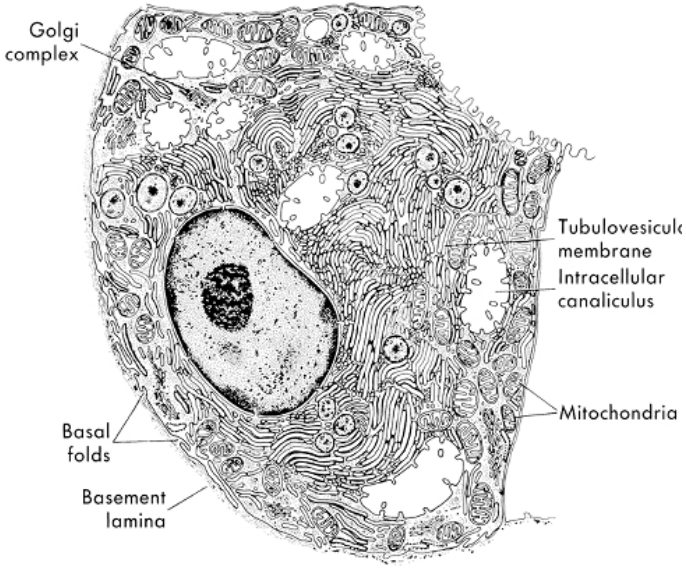
Digestive System (Vander, Sherman & Luciano, *Human Physiology*, Cap. 17, 2002, McGraw Hill

Secreção ácida gástrica: a célula parietal

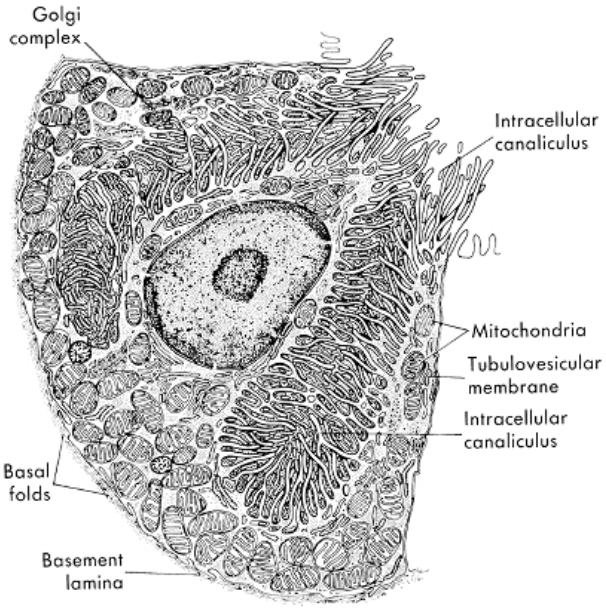




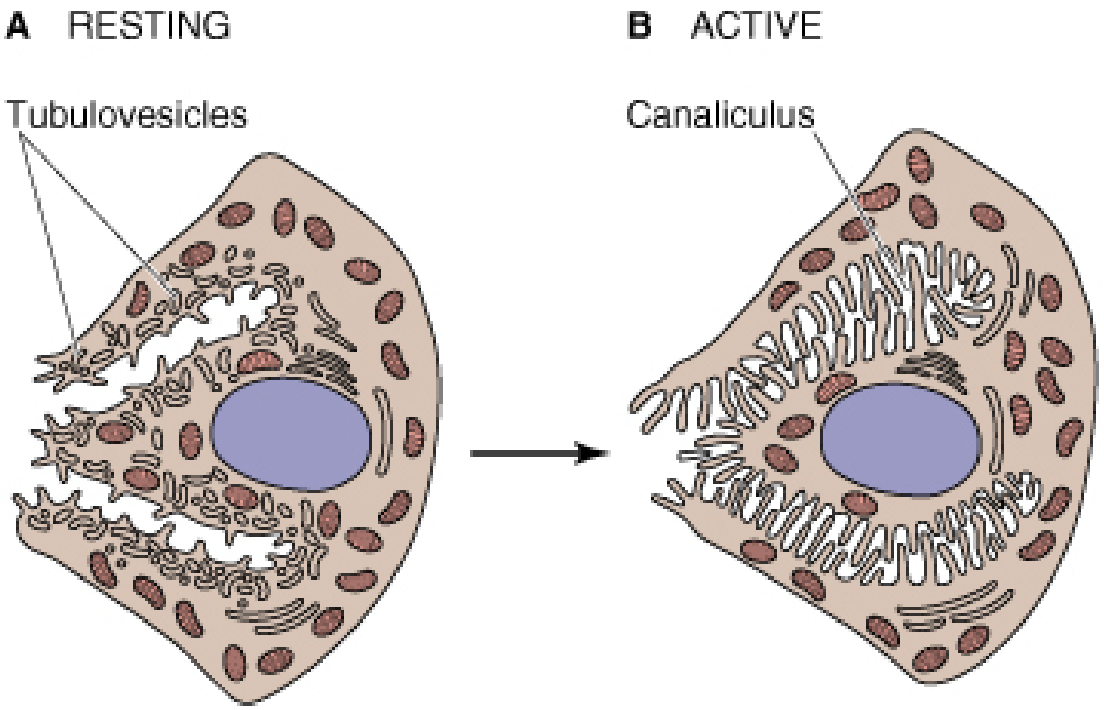
em “repouso”



em atividade secretora



(A) Drawing of a resting parietal cell with cytoplasm full of tubulovesicles and an internalized intracellular canaliculus.
(B) An acid-secreting parietal cell. Tubulovesicles have fused with the membrane of the intracellular canaliculus, which is now open to the lumen of the gland and lined with abundant, long microvilli. (From Ito S: In Johnson RL, editor: Physiology of the gastrointestinal tract, New York, 1981, Raven Press.)



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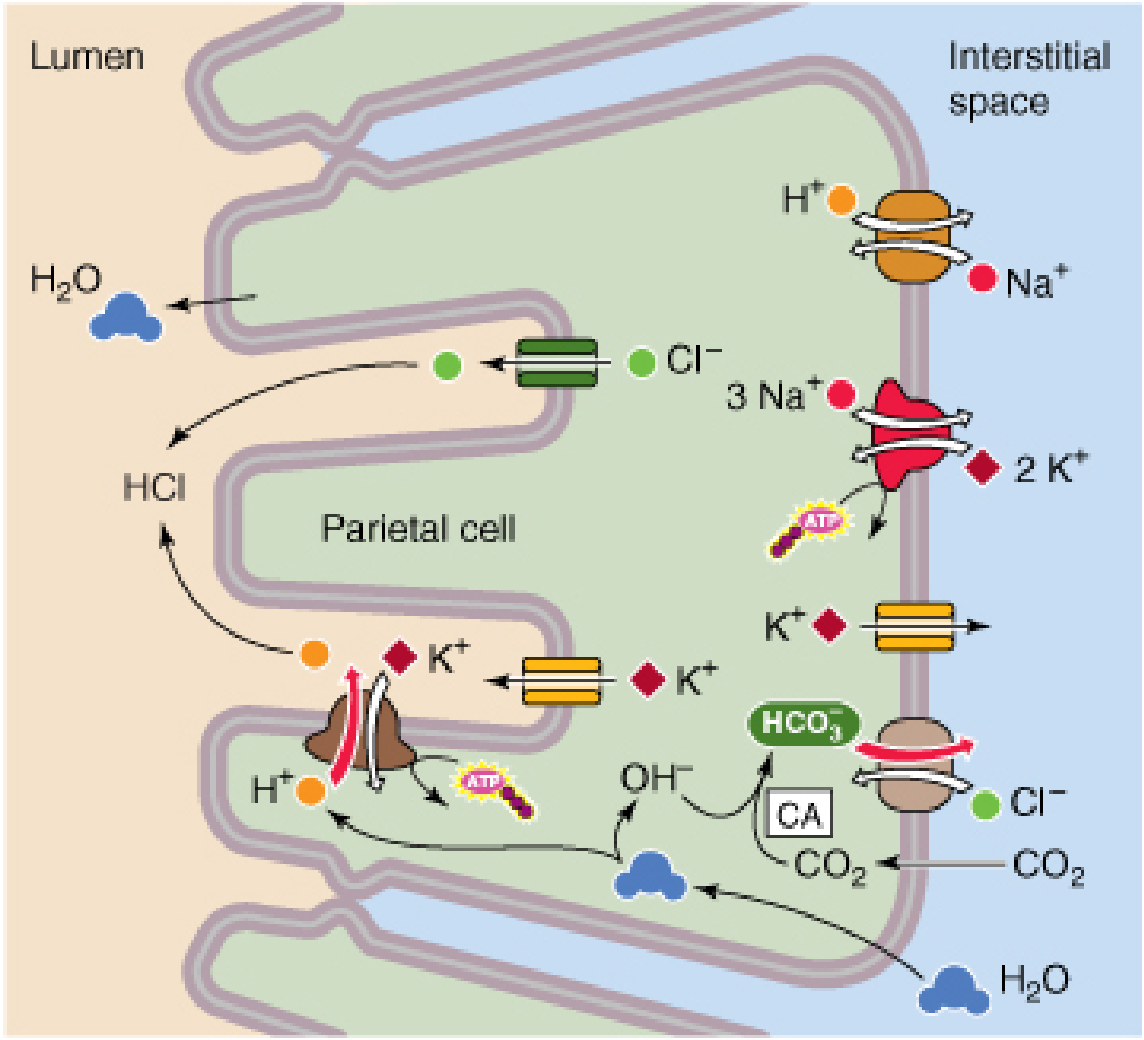
Mecanismos intracelulares de secreção ácida gástrica (célula parietal)



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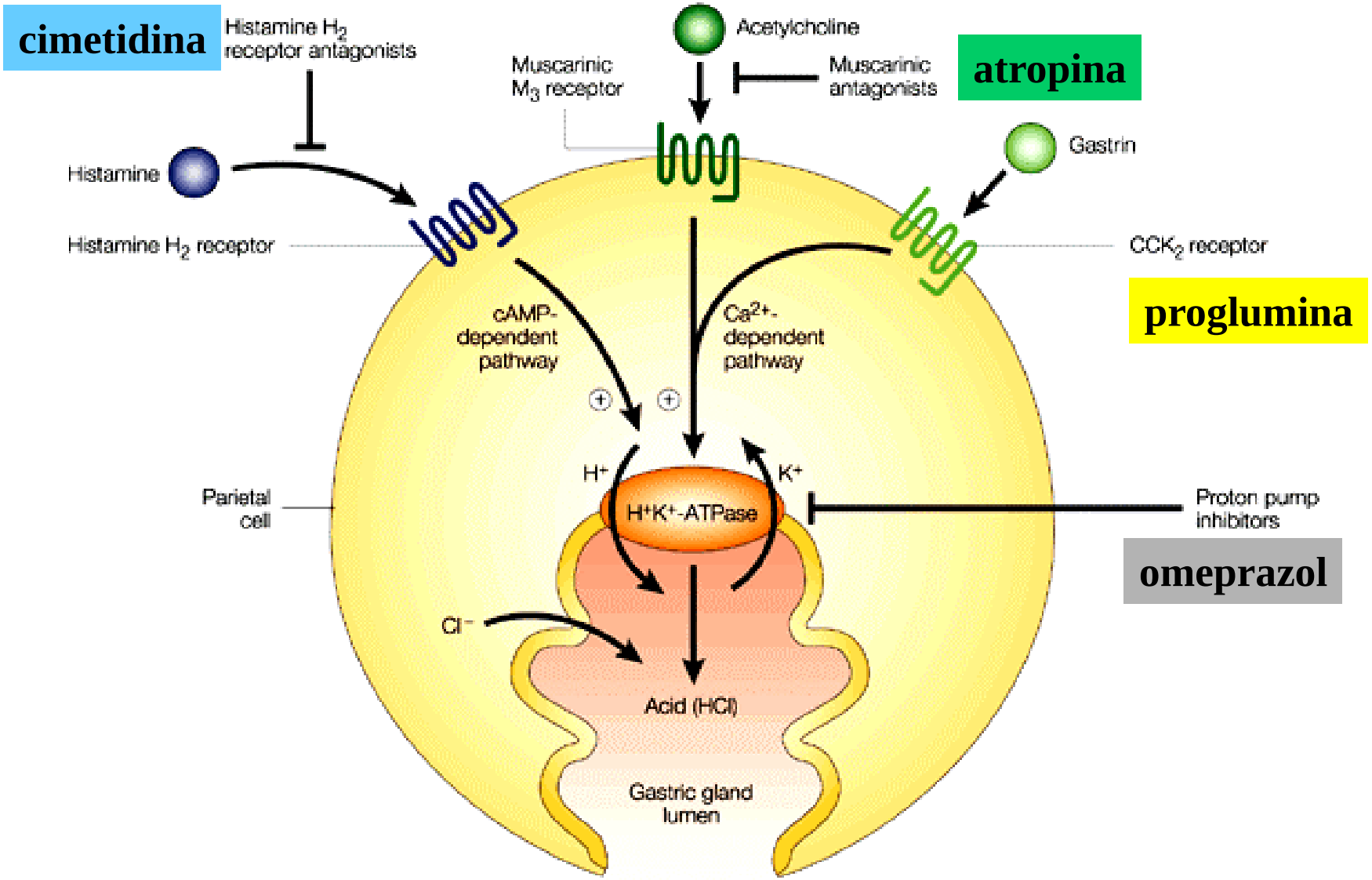
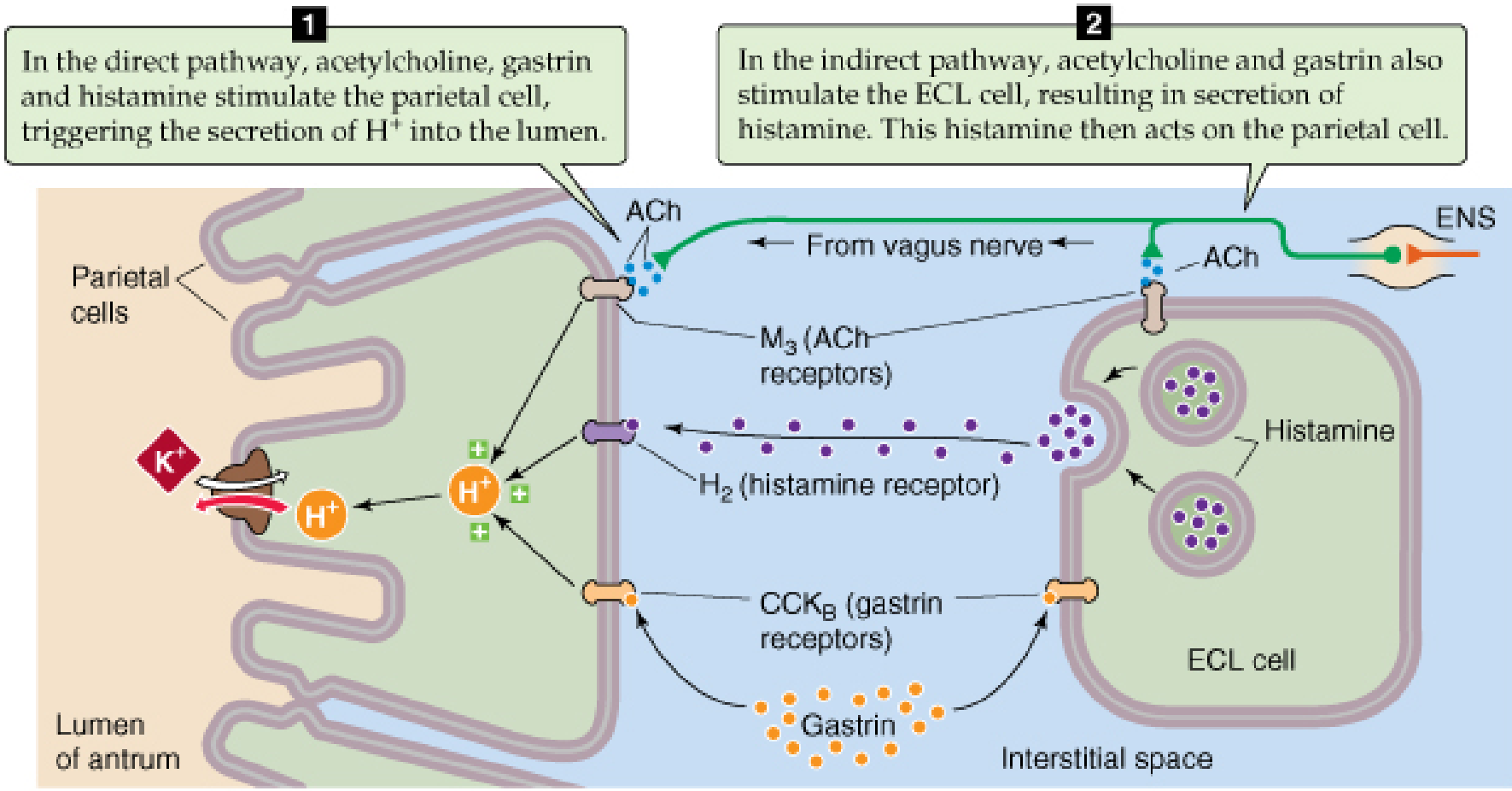
Bloqueadores da secreção ácida



Potencialização da secreção ácida



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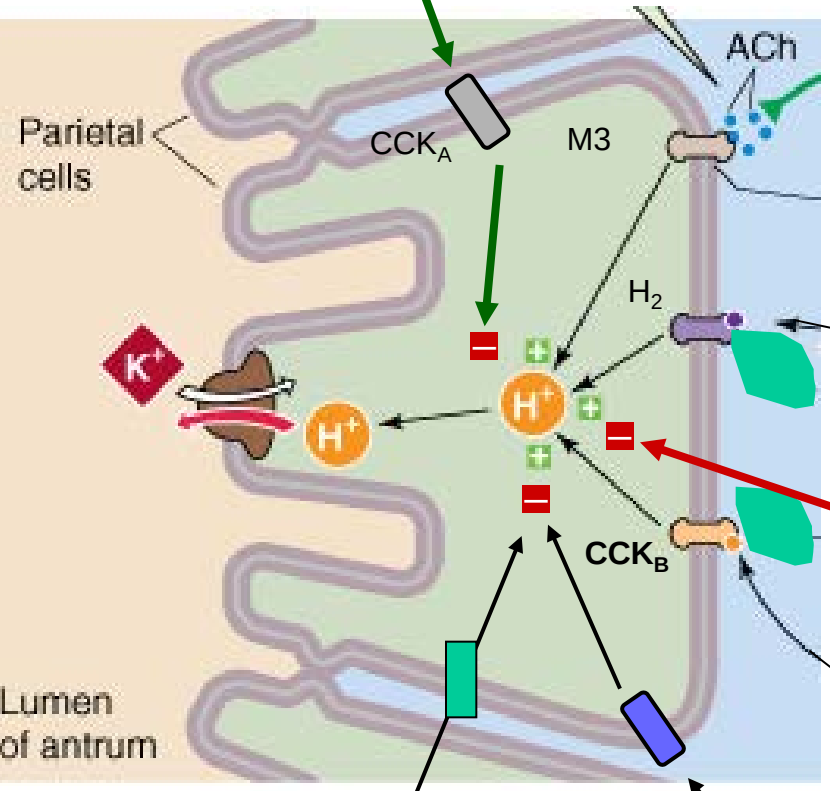
VIAS INIBITÓRIAS DA SECREÇÃO ÁCIDA

Cholecystokinin (CCK)

Enterogastronas:
-Somatostatina
-GIP
-Neurotensina
-Peptídeo YY



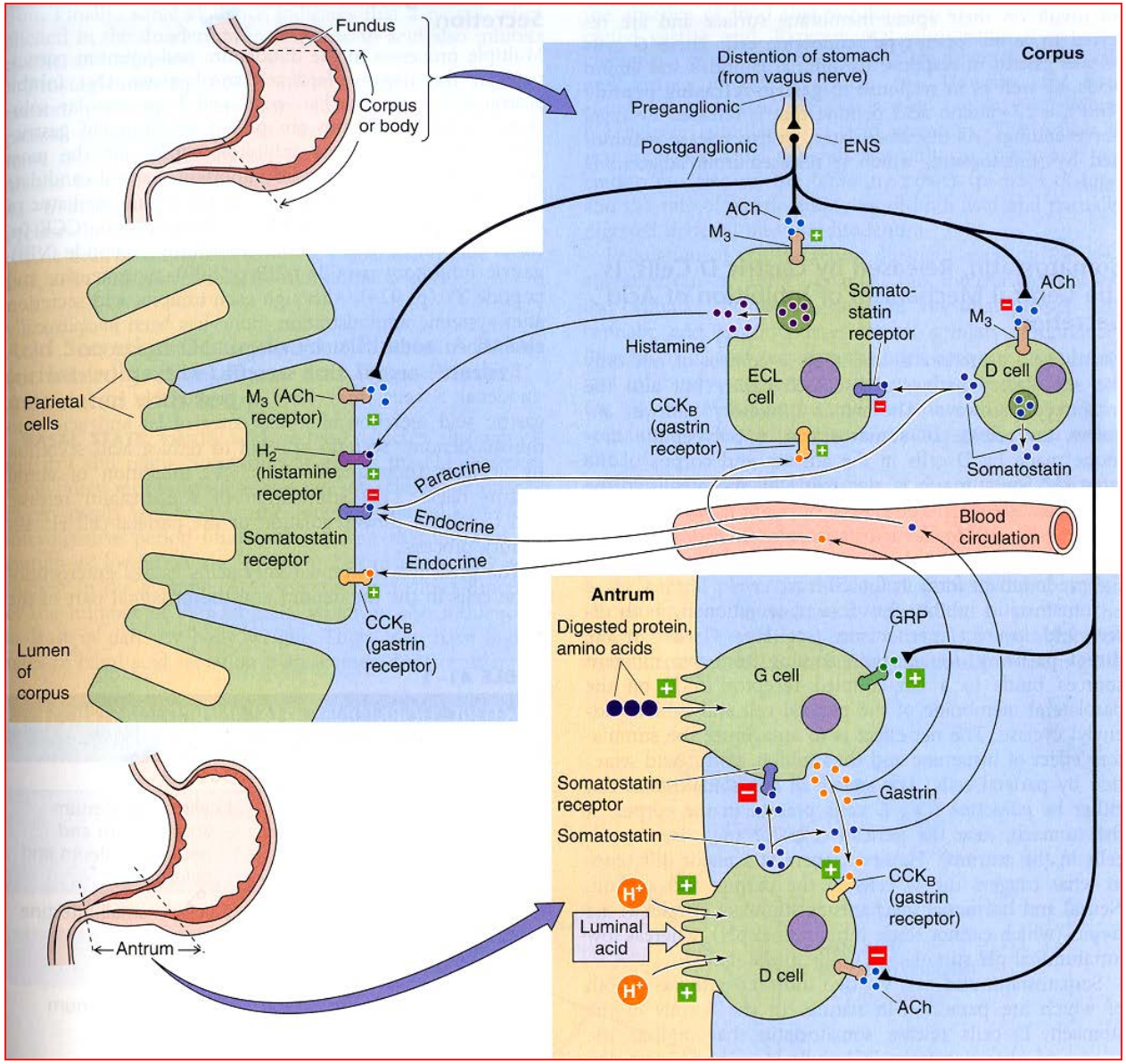
Histamine
ECL

Gastrin
G cells antrum

Secretin
(S cell small intestine)

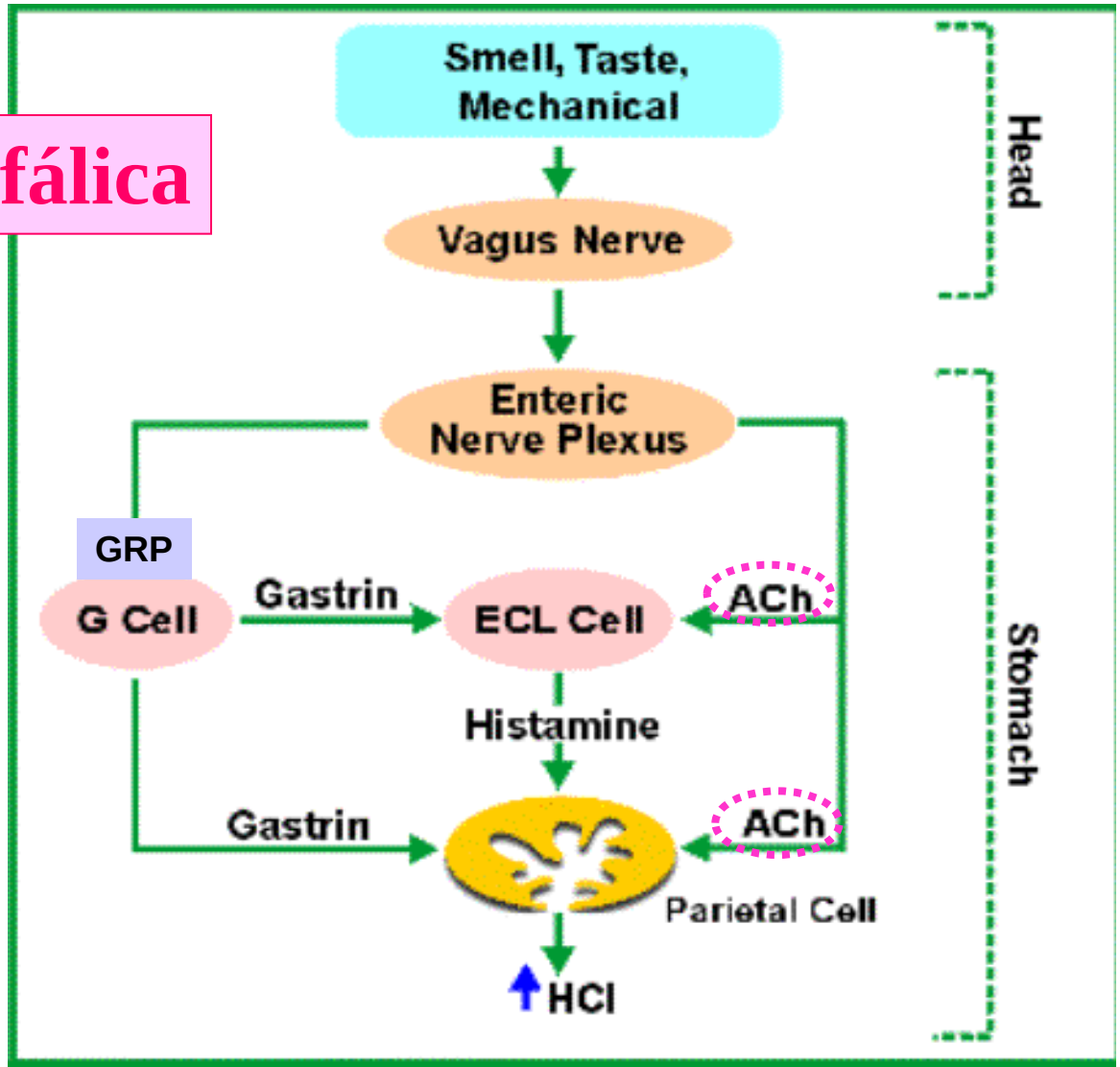
Prostaglandinas E2

Somatostatin
D cells (SS28)

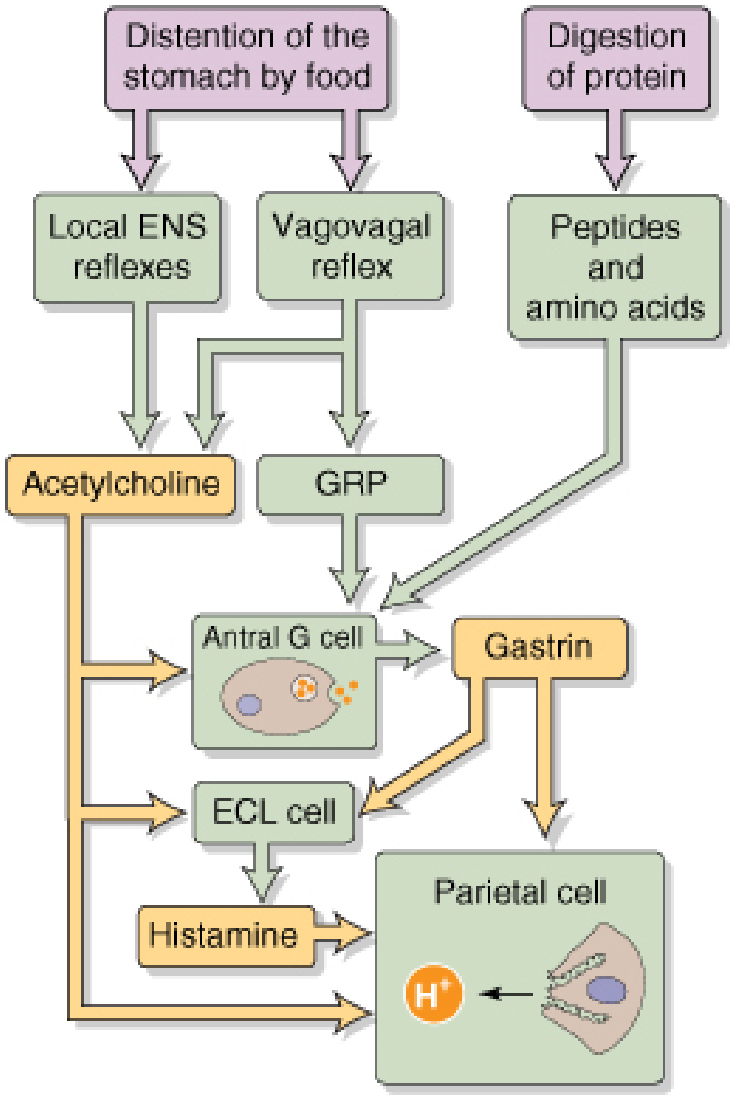

Fundus
Corpus or body
Distention of stomach (from vagus nerve)
Corpus
Preganglionic
Postganglionic
ENS
ACh
M3
Somato-statin receptor
Histamine
ECL cell
CCKB (gastrin receptor)
D cell
Somatostatin
Parietal cells
M3 (ACh receptor)
H2 (histamine receptor
Somatostatin receptor
Paracrine
Endocrine
Endocrine
Blood circulation
Lumen of corpus
CCKB (gastrin receptor)
Antrum
Digested protein, amino acids
GRP
G cell
Somatostatin receptor
Gastrin
Somatostatin
CCKB (gastrin receptor)
H+
Luminal acid
H+
D cell
ACh
Antrum

Fases da secreção gástrica

Fase cefálica



FASE GÁSTRICA



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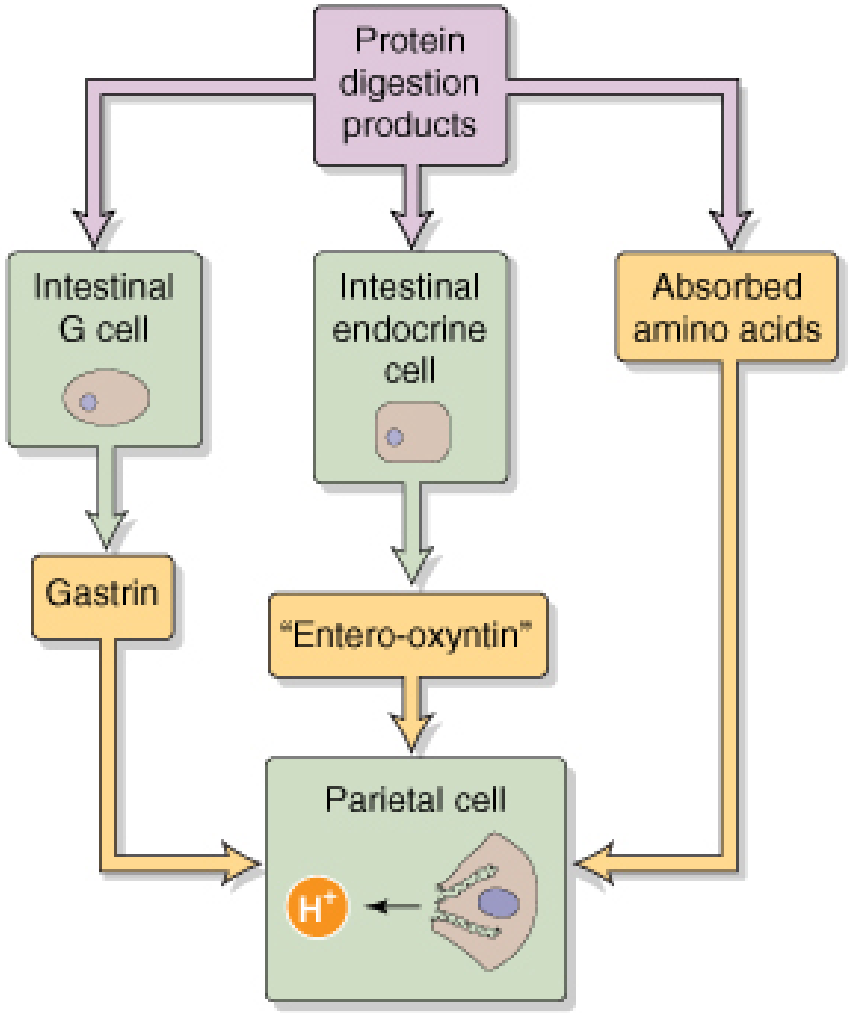
Via inibitória: acidez gástrica levando ao aumento da SS

FASE INTESTINAL



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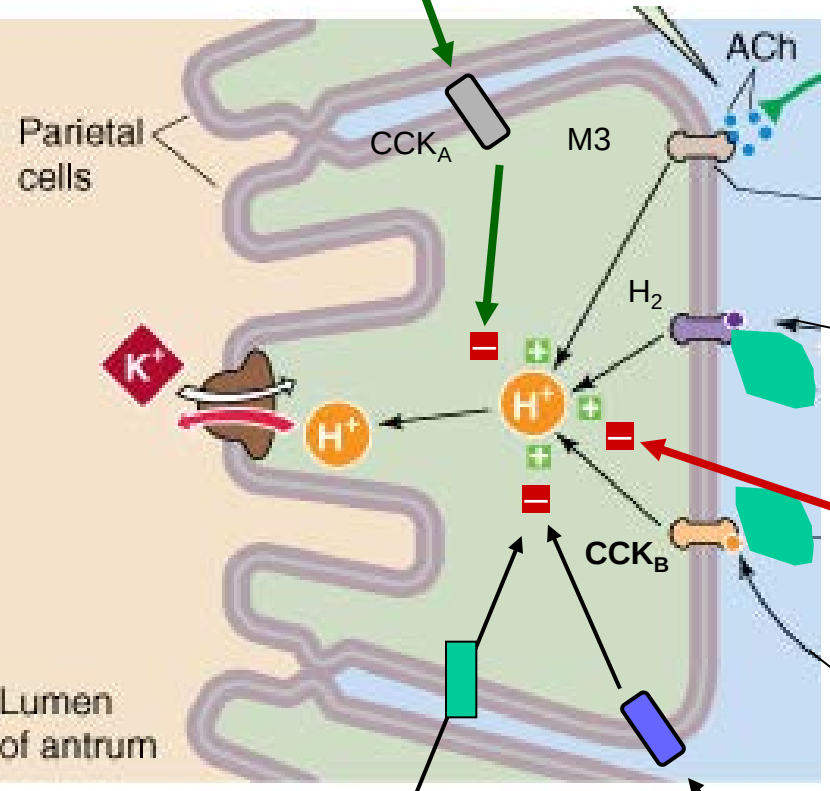
VIAS INIBITÓRIAS DA SECREÇÃO ÁCIDA

Cholecystokinin (CCK)

Enterogastronas:
-Somatostatina
-GIP
-Neurotensina
-Peptídeo YY



Histamine
ECL

Gastrin
G cells antrum

Secretin
(S cell small intestine)

Prostaglandinas E2

Somatostatin
D cells (SS28)

Por que a pepsina não digere o epitélio gástrico ?

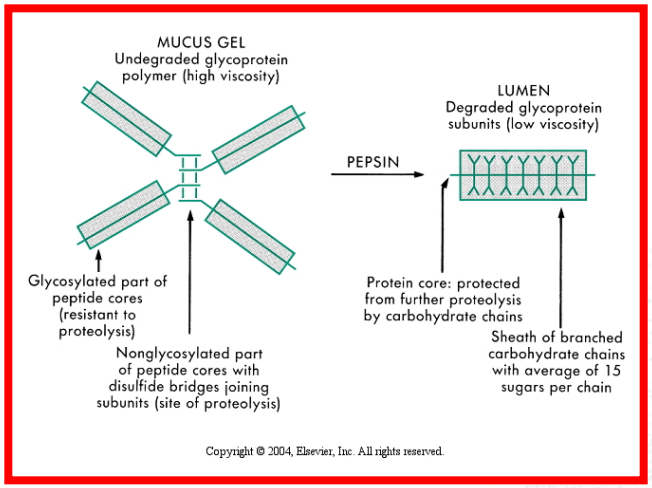
BARREIRA MUCOSA GÁSTRICA:

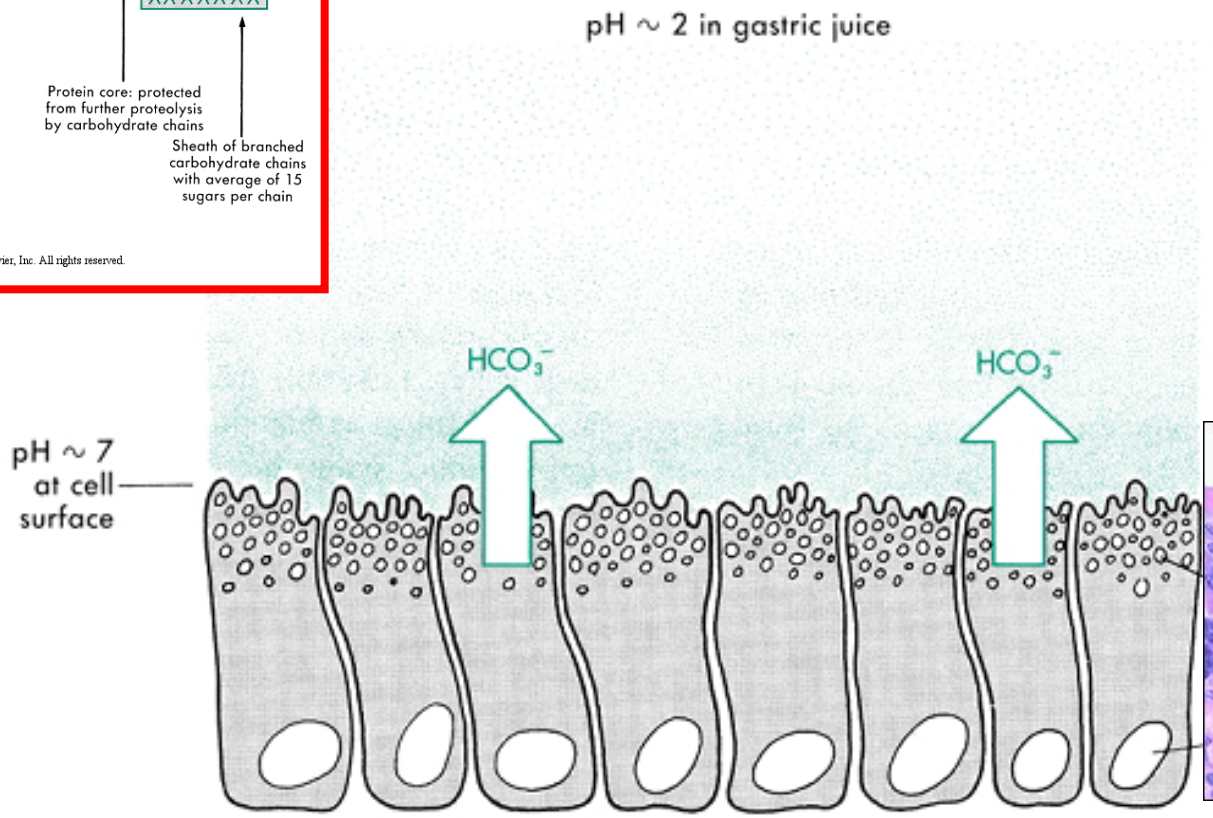
1-IMPERMEABILIDADE RELATIVA AO ÁCIDO DAS MEMBRANAS APICAIS DAS GLÂNDULAS GÁSTRICAS

2-PRESENÇA DE UMA CAMADA DE GEL-MUCOSO NA SUPERFÍCIE DO EPITÉLIO

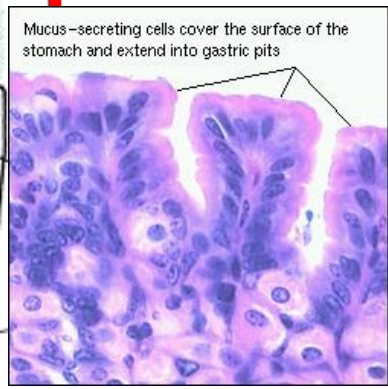
2-ALTA CONCENTRAÇÃO DE BICARBONATO NAS ADJACÊNCIAS DO EPITÉLIO

Proteção mucosa





camada mucosa (0,2 mm)



PGE2
(prostaglandinas são citoprotetoras)

⊕

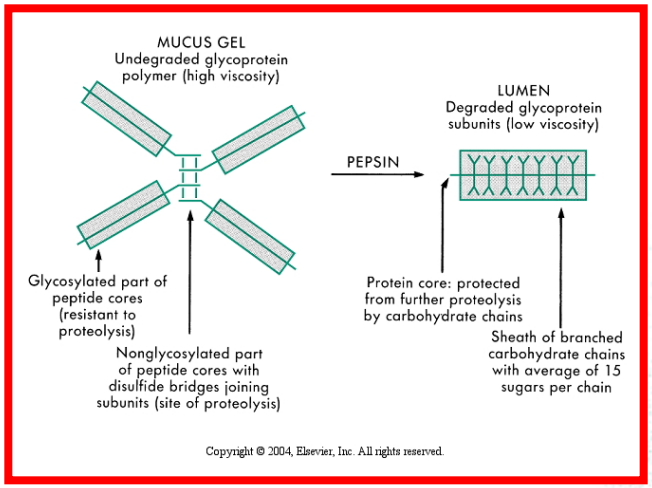
ACh, Food
(PS e SNE)

- ↑ secreção de muco e HCO_3^- pelas células epiteliais e mucosas
- ↑ fluxo sangüíneo

The protection provided to the mucosal surface of the stomach by the bicarbonate-containing mucus layer is known as the gastric mucosal barrier. In man, the mucus layer is about 0.2 mm thick. Buffering by the bicarbonate-rich secretions of the surface epithelial cells and the restraint to convective mixing caused by the high viscosity of the mucus layer allow the pH at the cell surface to remain near 7, whereas the pH in the gastric juice in the lumen is 1 to 2.

COX1: atividade ciclooxigenase da PGH_2-sintase). Berne et al., 2004 e outros: http://meds.queensu.ca/medicine/physiol/undergrad/phase2/phase2e/Ph2acid.htm

Proteção da mucosa



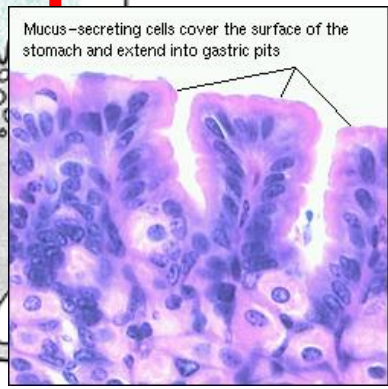
pH ~ 2 in gastric juice

camada mucosa (0,2mm)

Estresse

HCO_3^- HCO_3^-

pH ~ 7 at cell surface



NSAIDs
(drogas antiinflamatórias não-esteróides)

inibição da COX 1
(constitucional)

PGE2
(prostaglandinas são citoprotetoras)

⊕

ACh
(PS e SNE)

↑ secreção de muco e HCO_3^- pelas células epiteliais e mucosas

↑ fluxo sangüíneo

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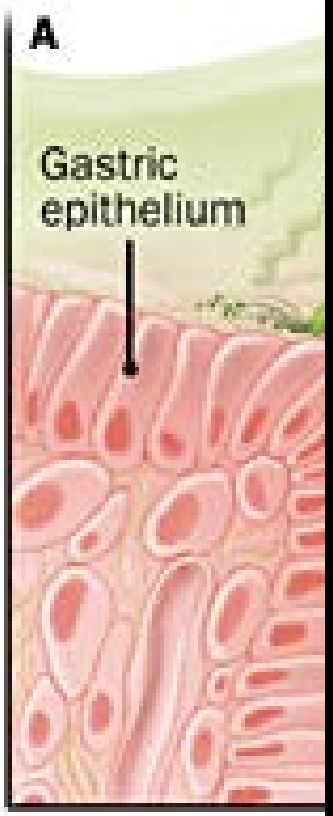
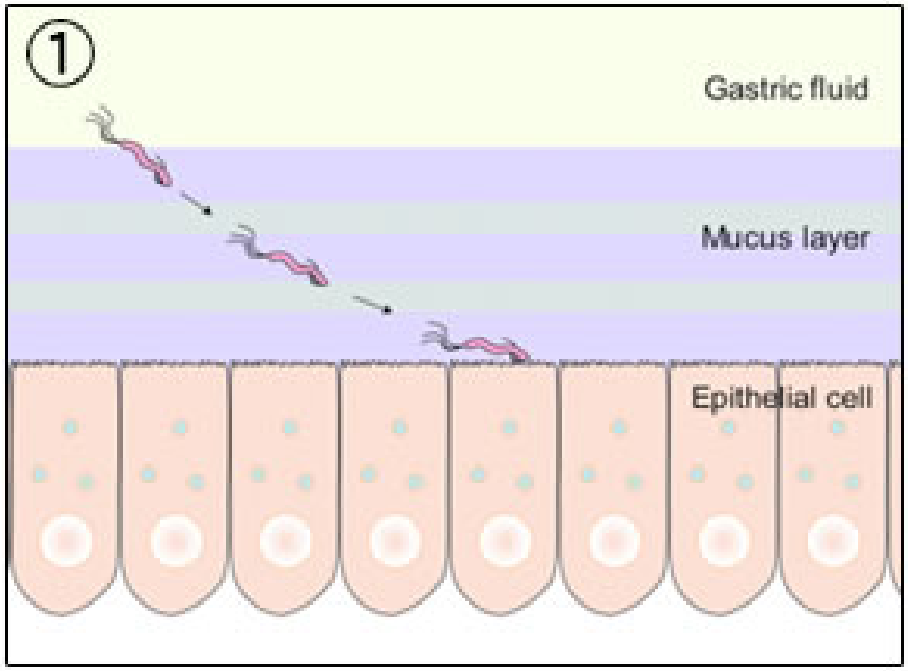
Helicobacter pylori

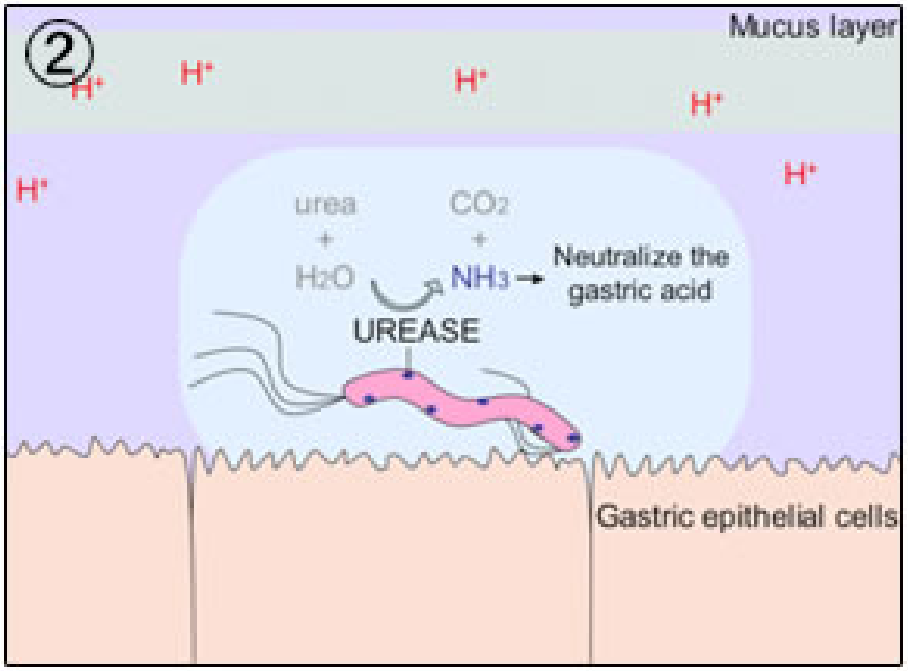


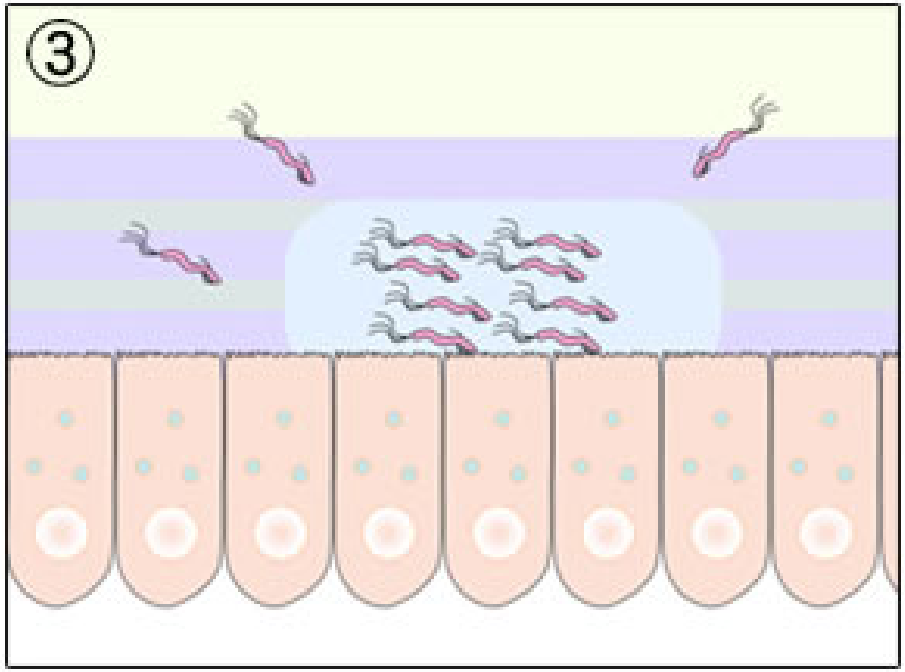
The Nobel Prize in Physiology or Medicine for 2005:
jointly to
Barry J. Marshall and **J. Robin Warren**
(University of Western Australia Nedlands)
for their discovery of
"the bacterium *Helicobacter pylori*
and its role in gastritis and peptic ulcer disease"

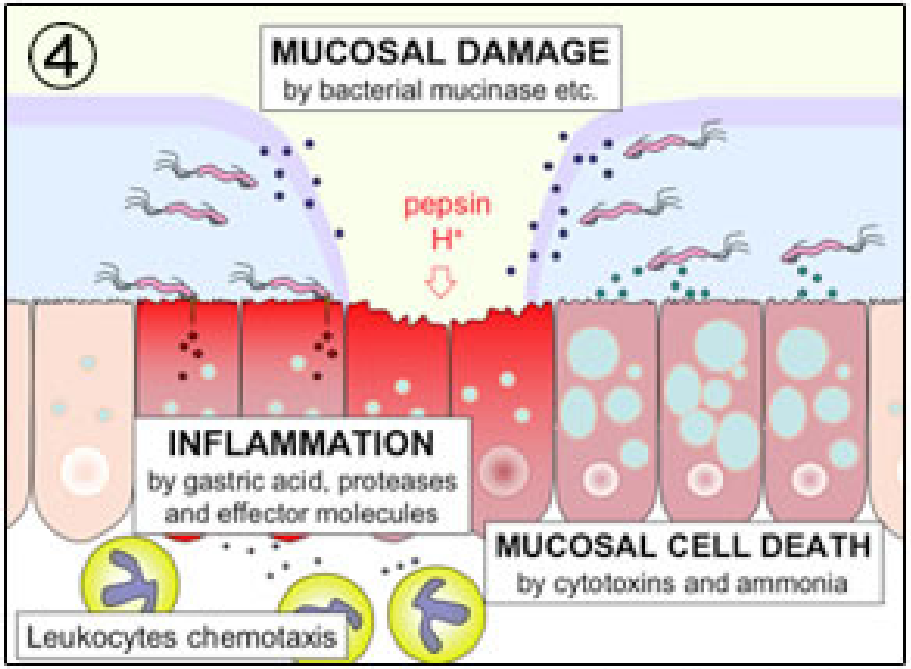
H. pylori is the etio e individuals to the development of gastric carcinoma. *H. pylori* colonizes in the human stomach. The method of *H. pylori* transmission is unclear, but seems to be person-to-person spread via a fecal-oral route. The prevalence of *H. pylori* in adults appears to be inversely related to the socioeconomic status. It is also thought that water is a reservoir for transmission of *H. pylori*.

http://hopkins-gi.nts.jhu.edu/pages/latin/templates/index.cfm?pg=disease2&organ=5&disease=16&lang_id=1



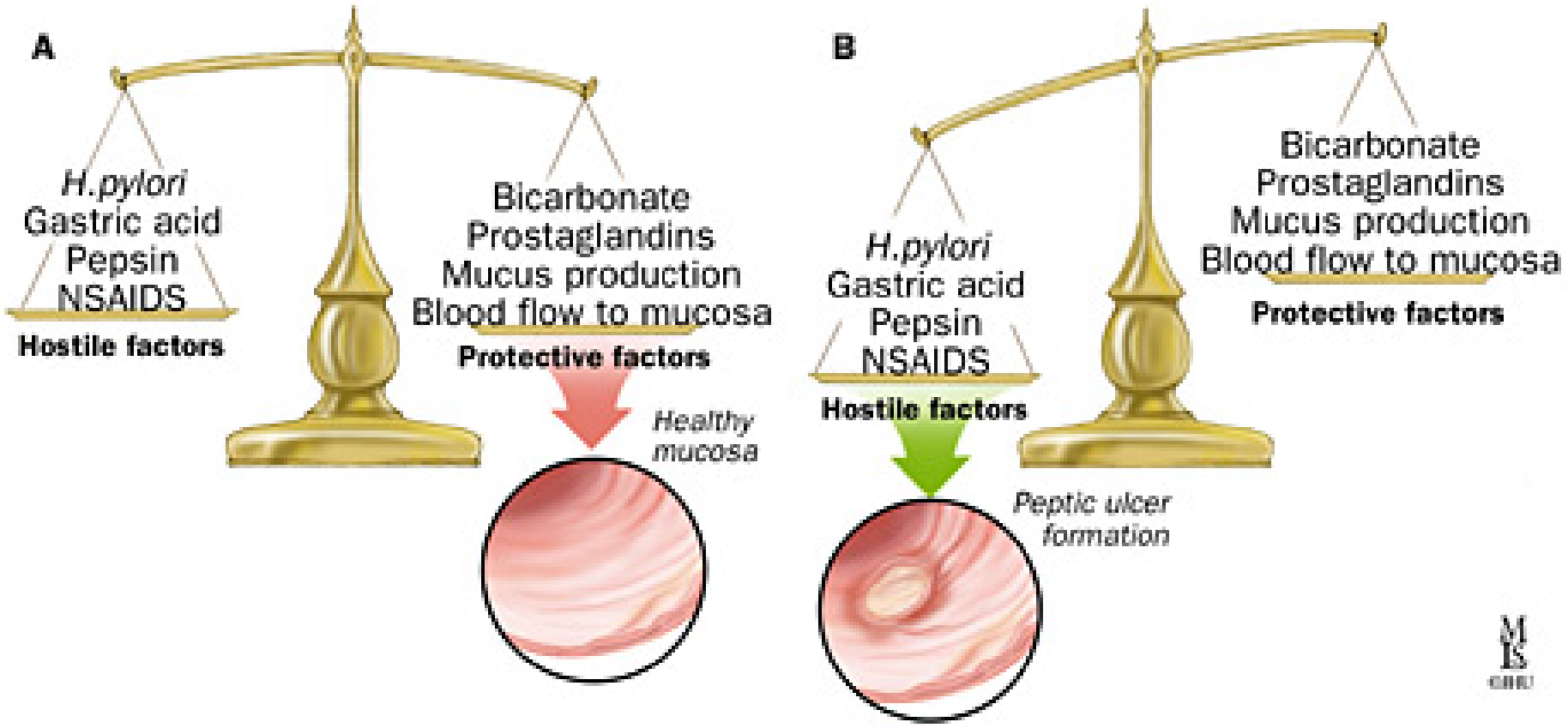






http://h.pylori.info/

A integridade do trato gastrointestinal superior depende do balanço entre fatores “hostis” e “protetores”



http://hopkins-gi.nts.jhu.edu/pages/latin/templates/index.cfm?pg=disease2&organ=5&disease=16&lang_id=1