Peptic Ulcer

History of Presenting Illness

There is no symptom complex that can adequately differentiate gastric from duodenal ulcers and nonulcer dyspepsia

Burning epigastric pain 1.5 to 3 hrs after meals
→ **Wakes patient up at night**
exacerbated by fasting
improved with meals
improved with antacids
- **BLACK STOOLS**

GASTRITIS is made WORSE WITH FOOD

Peptic Ulcer may be felt as a gnawing pain in the chest, back, mid-abdomen, or either upper quadrant

Melena = *at least 60 mls of uper Gi bleeding*
Really need at least 150 – 200 for the "black tarry"
Takes 8 hours to turn black

Differential Diagnoses

Biliary Colic
Cholelithiasis
Gastritis, Acute
Gastritis, Chronic
Gastroesophageal Reflux Disease
Mesenteric Artery Ischemia
Myocardial Ischemia
Pancreatic Cancer
Pancreatitis, Acute ← *80% of pancreatitis presents at 1 am (?…)*
Pancreatitis, Chronic
Drug-induced dyspepsia
Duodenitis
Functional (nonulcer) dyspepsia
Gastric infections
Infiltrative diseases of the stomach

The important causes of upper GI bleeding
- Duodenal ulcer
- Gastric ulcer
- Gastric erosions
- Ulcerative esophagitis
- Esophagogastric varices
- Mallory-Weiss tear
- Carcinoma, lymphoma
- Angiodysplasia

only 20-25% of patients with symptoms suggestive of peptic ulceration are found on investigation to have a peptic ulcer.

Pertinent Findings on History

Nausea
Vomiting,
Dyspepsia,
- **belching,**
- **bloating,**
- **distention,**
- **fatty food intolerance**

Heartburn
Chest discomfort
Anorexia, weight loss
Hematemesis

Red Blood + Clots?
= per rectum (massive duodenal bleed)
Cardiovascular effects eg.
- **tachycardia, palpitations**
- **weak pulse**
- **SYMPTOMS OF SHOCK**

Anaemia (indicates chronicity)

May be Embarrassing:
Could all that blood be coming from the patient's chronically bleeding NOSE??

ALCOHOL
TOBACCO
NSAIDs
And COCAINE

HISTORICALLY chronic peptic-ulcer sounding intermittent food-relieved epigastric pain…
…SUDDEN TURN FOR WORSE: SEVERE CONSTANT AND GENERALISED WITH RIGID ABDOMEN AND TACHYCARDIA → !! Perforated Ulcer !!

Findings on Examination : few and non-specific

- **Epigastric tenderness**
- **Guaiac-positive blood in stool** resulting from occult blood loss
- **Melena** resulting from acute or subacute gastrointestinal bleeding
- **Succussion splash** resulting from partial or complete gastric outlet obstruction
- **SOMETIMES: stigma of chronic liver disease, eg.**
  distended abdomen, ecchymoses, jaundice

Table 154-1: Commonly Used Tests to Detect *Helicobacter pylori* Infection

Test	Advantages	Disadvantages
INVASIVE (ENDOSCOPIC BIOPSY-BASED)		
Biopsy urease test	Quick, simple	Rapid test not fully sensitive, 24-h test not fully specific
Histology	Widely available; may give additional histologic information	Sensitivity dependent on experience
Culture	Permits determination of antibiotic susceptibilities	Sensitivity dependent on experience
NONINVASIVE		
Serology	Cheap and convenient	Cannot be used for early follow-up
^{11}C or ^{14}C urea breath test	Safer and cheaper than endoscopy	Low-dose irradiation in ^{14}C test

Tests and Investigations

Full Blood Count
To make sure they are not anaemic

Coagulation assay
To make sure that you can operate on them

Chest X-ray
Looking for gastric abnormality
Also looking to explain any cardiovascular side-findings

Serum Biochemistry
UREA is elevated in *Helicobacter Pylori* infections

INDICATIONS FOR IMMEDIATE ENDOSCOPY
Those over 45 years of age with new onset of dyspeptic/ulcer symptoms or with other symptoms suggesting malignancy, such as
- **fever,**
- **weight loss,**
- **early satiety,**
- **vomiting,**

IS THE BLEEDING CONTINUING?
Mesenteric angiography may point to the site of ulcer

EXHALED UREA breath tests
Urea is ingested; in the presence of urease produced by H pylori, *labeled carbon dioxide (heavy isotope, carbon-13, or radioactive isotope, carbon-14) is produced in the stomach, absorbed into the bloodstream, diffused into the lungs, and exhaled.*

GASTROSCOPY and/or ENDOSCOPY with subsequent BIOPSY
PLUS: *Rapid urease tests* *for H. Pylori*

The presence of H pylori *in gastric mucosal biopsy specimens is detected by testing for the bacterial product urease. Three kits (CLOtest, Hpfast, Pyloritek) are commercially available, each containing a combination of a urea substrate and a pH sensitive indicator. One or more gastric biopsy specimens are placed in the rapid urease test kit. If* H pylori *are present, bacterial urease converts urea to ammonia, which changes pH and produces a color change.*

Follow-up endoscopy should be performed at least 4 weeks after cessation of all anti-Helicobacter drugs

How is this diagnosis made? **Via BIOPSY + HISTOLOGY**
Reveal many H.Pylori organisms, with infiltration by marcophages + neurophils
MUST CHECK for dysplasia, metaplasia, neoplasia

Management

BASICALLY:
1. *Cauterise*
2. *Kill microbe*
3. *reduce stomach acidity*
4. *maintain mucosa integrity*

@ GASTROSCOPY:

- **Find site of bleeding**
- **Inject offending vessel with adrenaline to vasoconstrict**
- **Use heater probe to cauterise ulcer**
- ***REPLACE LOST BLOOD***
- ***Cease taking the offending NSAIDs!!***

PROTON-PUMP INHIBITORS ARE ONLY INDICATED if the ulcer is larger than 1 cm

CLOSE THE PERFORATION SURGICALLY if perforated

POST-CAUTERISATION → need **high-dose Proton Pump Inhibitor therapy** to prevent rebleeding

SHORT TERM:

Uncomplicated ulcer without H.Pylori might be effectively treated with PPIs ALONE

Else: MUST Eradicate H. Pylori:

Monotherapy ineffective; try TRIPLE DRUG THERAPY
7-14 days of PROTON PUMP INIBITOR + TWO ANTIBIOTICS
eg. omeprazole / clarithromycin / amoxicillin or metranidazole
CONTINUE PUMP INHIBITOR FOR 1-2 weeks AFTER

Rates of recrudescence or reinfection after successful eradication are low (approximately 1% per year).

Name	Drug 1[a]	Drug 2	Drug 3	Drug 4
OCA[b]	Omeprazole (20 mg bid)	Clarithromycin (500 mg bid)	Amoxicillin (1 g bid)	-
OCM[b]	Omeprazole (20 mg bid)	Clarithromycin (250 mg bid)	Metronidazole[c] (500 mg bid)	-
OBTM[d]	Omeprazole (20 mg bid)	Bismuth subsalicylate (2 tabs qid)	Tetracycline HCl (500 mg qid)	Metronidazole[c] (500 mg tid)

[a] In any of the three regimens, omeprazole may be replaced by lansoprazole (30 mg bid), pantoprazole (40 mg bid), ranitidine bismuth citrate (400 mg bid), or possibly ranitidine (150 mg bid). Most available data are for omeprazole.

[b] These regimens may be given for 7 to 14 days; meta-analysis suggests that 14-day regimens are slightly more effective.

[c] The optimal dose of metronidazole is not known. Tinidazole (500 mg bid) can be used in place of metronidazole.

[d] Data for this regimen are mainly from Europe and are based on bismuth subcitrate. Omeprazole is given for 10 days, and the other three agents are given on days 4 through 10.

LONG TERM: preventative and maintenance measures

Address LIFESTYLE FACTORS: reduce intake of coffee, alcohol, tobacco, NSAIDs

Non-Healing Ulcer? → **MAINTENANCE THERAPY with Proton Pump Inhibitor or Histamine-2 Receptor Antagonists**

Prognosis

The mortality rate is approximately 1 death per 100,000 cases.
The hospitalization rate is approximately 30 patients per 100,000 cases.

Epidemiology

4 million individuals (new cases and recurrences) affected per year in the US
Lifetime prevalence =12% in men and 10% in women.
PUD affects approximately 4.5 million people annually.
15,000 deaths per year occur as a consequence of complicated PUD.
PUs are estimated to occur in 6 to 15% of the western population.
The death rates have decreased by >50% over the past 30 years.

Sex:

- Prevalence has shifted from predominance in males to similar occurrences for both sexes.
- Lifetime prevalence is approximately 11-14% for men.
- Lifetime prevalence is approximately 8-11% for women.

Age:

- Age trends for ulcer occurrence reveal declining rates in younger men, particularly for duodenal ulcer, and increasing rates in older women.

Trends reflect complex changes in risk factors for PUD, including age-cohort phenomena with the prevalence of *H pylori* infection and the use of NSAIDs in older populations.

Pharmacology of Gastric Secretion Therapies

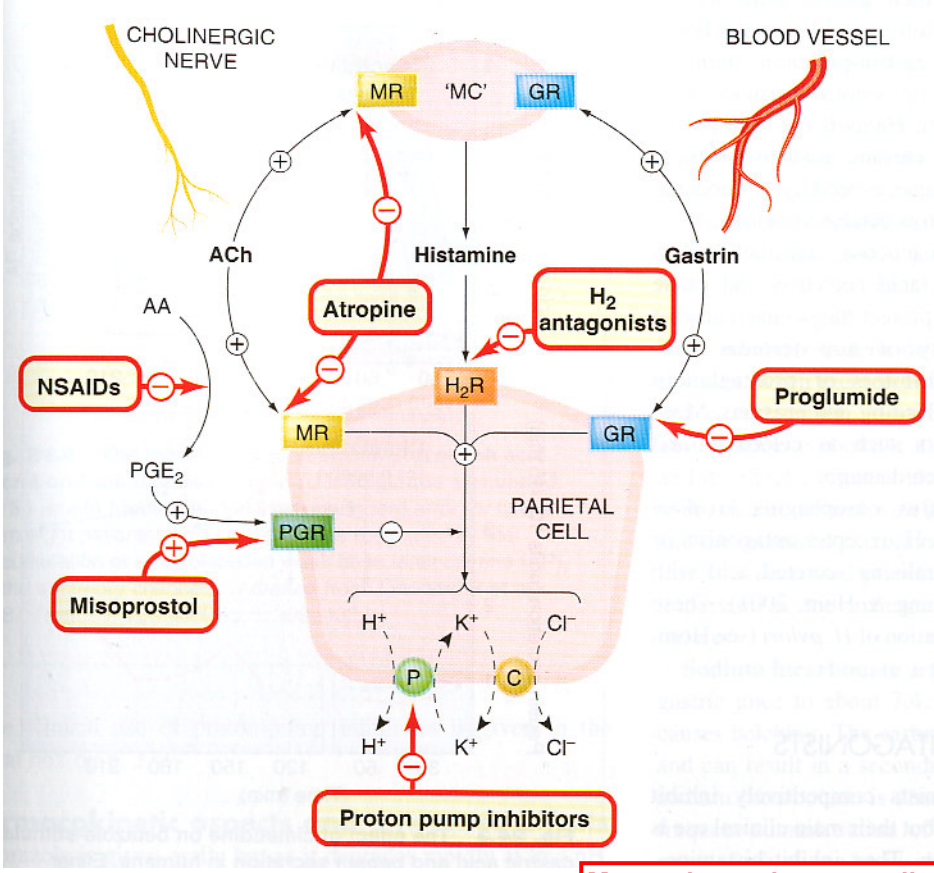


Fig. 24.2 **Schematic diagram showing the one-cell and two-cell hypotheses of the action of secretagogues on the acid-secreting gastric parietal cell, giving the site of action of drugs influencing acid secretion.** Acetylcholine and gastrin may act mainly directly on their receptors (the one-cell hypothesis) or partly directly and partly by releasing histamine (the two-cell hypothesis). ('MC', mast-cell-like, histamine-secreting cell; Hist, histamine; ACh, acetylcholine; MR, muscarinic receptor; H_2R, histamine H_2-receptor; GR, gastrin receptor; PGR, prostaglandin E_2 (PGE_2) receptor; AA, arachidonic acid; NSAIDs, non-steroidal anti-inflammatory drugs; P, proton pump (H^+/K^+-ATPase); C, symport carrier for K^+ and Cl^-.)

Magnesium salts cause diarrhoea
Aluminium salts cause constipation

Antacids Very brutally neutralise gastric acid

Magnesium hydroxide, aluminium hydroxide, sodium bicarbonate (!! CO_2 Belching !!)

Histamine 2 Receptor Antagonists

Since gastric parietal cells are stimulated by histamine to produce acid
via histamine binding to the H2 receptor,
at concentrations which are not enough to stimulate blood vessels
(this histamine comes from adjacent "mast cell-like cells")
(there is a slow basal secretion of histamine)

cimetidine, ranitidine, famotidine and nizatidine

Proton Pump Inhibitors

inactivate H+/K+ATPase; thus no protons are pumped into the lumen
and thus pH does not decrease.
PPIs do not eradicate H.Pylori, but they do suppress its growth

omeprazole, lansoprazole, pantoprazole, rabeprazole, esomeprazole

Cytoprotective Mucosa-loving Drugs:

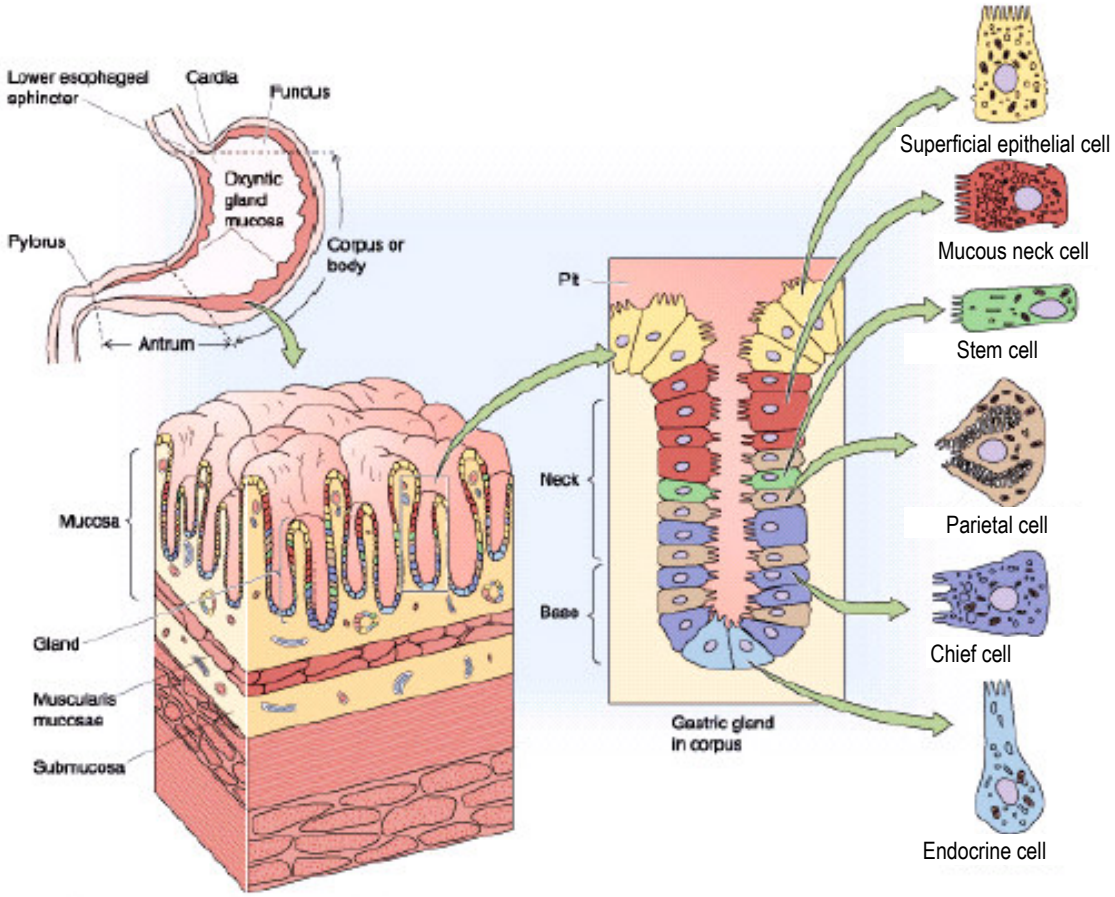
Sucralfate = a complex of aluminium hydroxide and sulfated sucrose
In presence of acid will release aluminium
Released aluminium acquires a strong negative charge and binds to +ve groups in proteins, glycoproteins, etc. **The result is thick mucus which limits the transit of H+**
Its like a band-aid for your mucosa. Allows the ulcer to re-epithelialise
NEEDS ACID!! DO NOT GIVE WITH ANTACIDS!!

Carbenoxolone**:** natural liquorice root product. Promotes mucus.

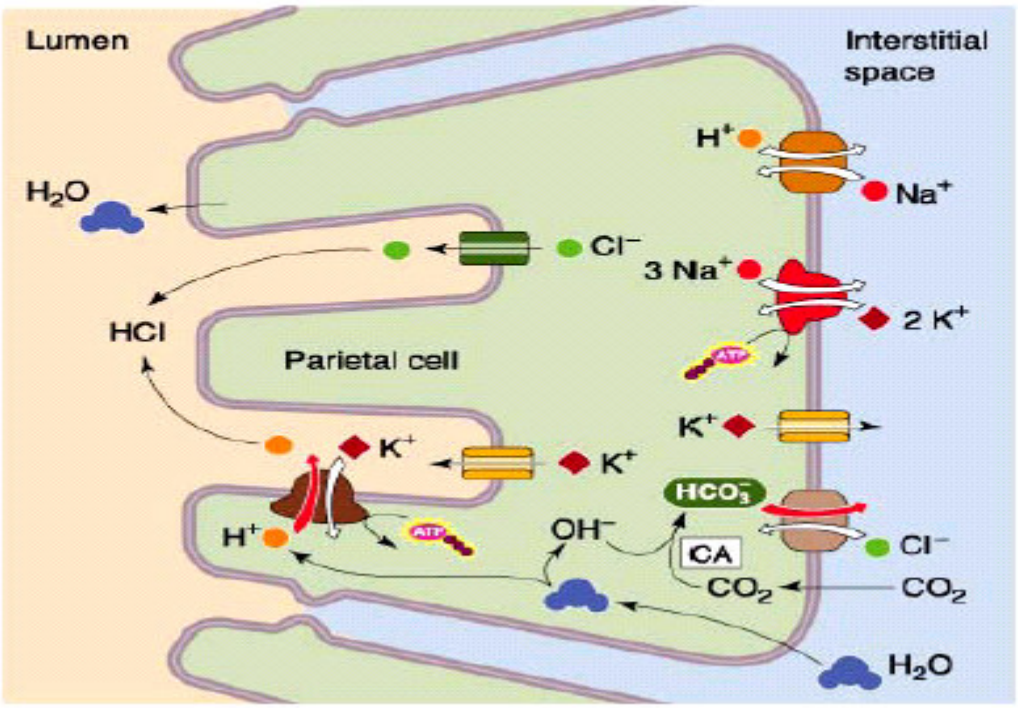
Prostaglandins by nature inhibit gastric acid secretion. Eg. ***misoprostol***

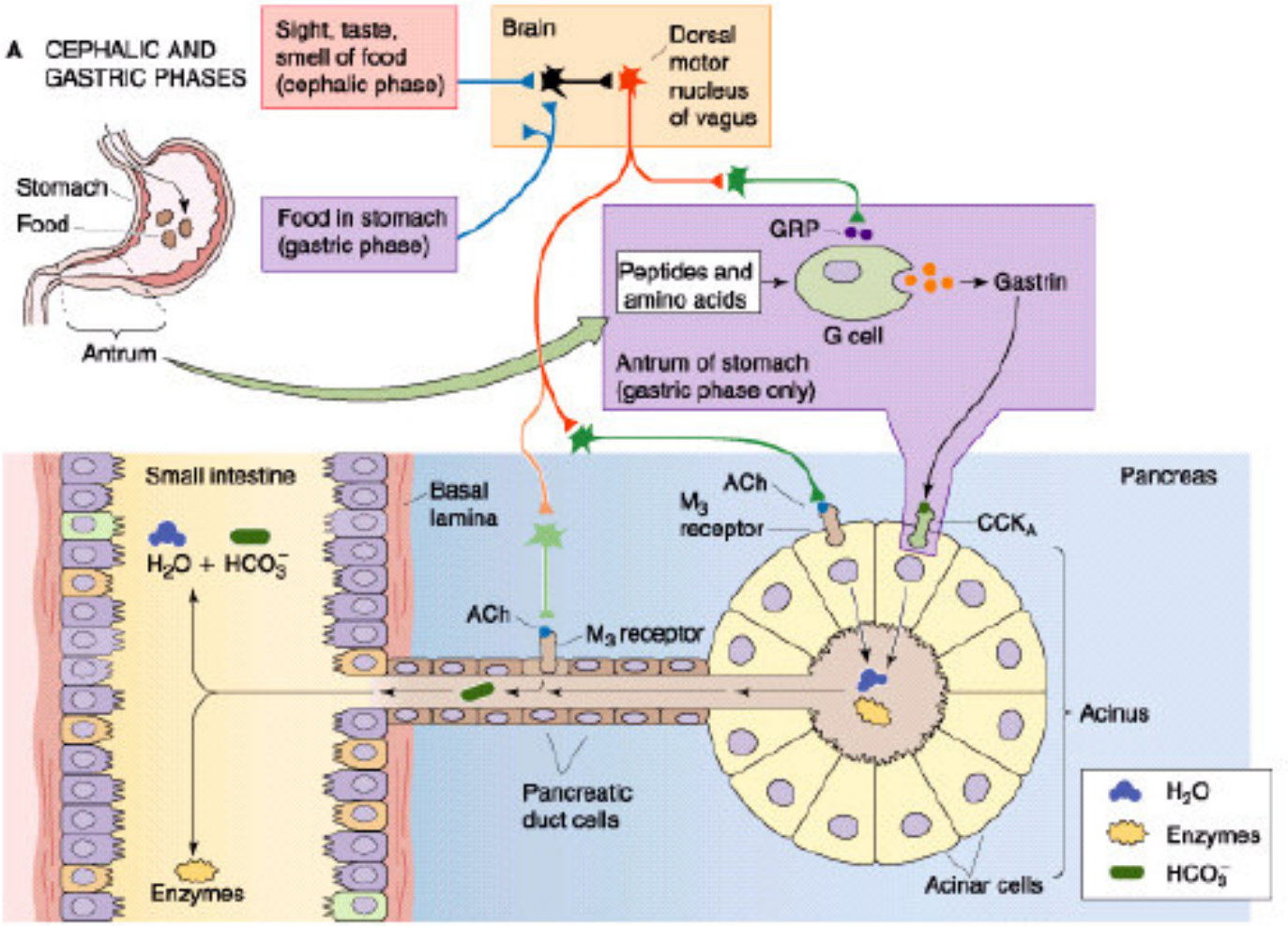
Bismuth Chelate: is toxic to H. Pylori and seems to coat the ulcer walls.
!! may cause black tongue and faeces, nausea, vomiting, and encephalopathy.

Stomach secretes acid and pepsinogen



Model for gastric acid secretion





Gastrointestinal disturbances

These are the commonest adverse effects of NSAIDs,
rel.risk = 3 to 5 times that of non-NSAID users.
20% of long-term NSAID users will have evidence of ulcers.
30% of all cases of massive gastrointestinal haemorrhage in the elderly are from NSAIDs.
misoprostol + famotidine = reduces the incidence of NSAID induced ulceration.

Renal effects

NSAIDs block the synthesis of PGI2 and PGE2.
(PGI2 and PGE2enhance glomerular filtration and inhibit the tubular reabsorption of sodium).
Thus, NSAIDs can cause sodium retention and consequent oedema.

Drug interactions

All NSAIDs have the potential to interfere with platelet function
The renal clearance of lithium is decreased by some NSAIDs
Some NSAIDs reduce the renal clearance of methotrexate

Risk factors in the elderly

Crap liver = increased plasma half-liife of NSAIDs
The elderly are more likely to be taking other medications for concurrent disease so the risks of drug interaction are greater in this group.

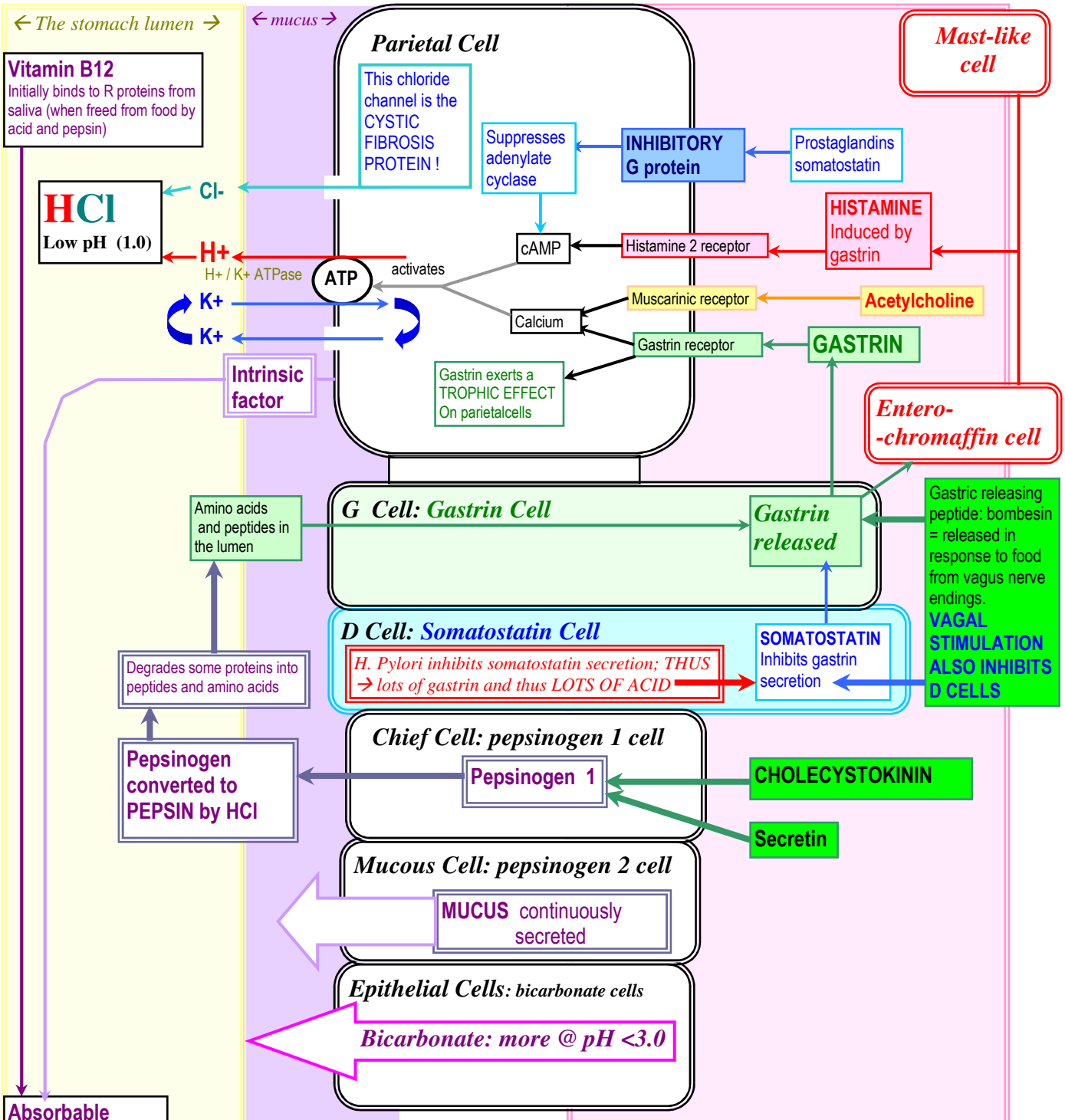
Speaking of the elderly… key word for the barrier: multidimensional holisitic approach

Common Comorbidities in the elderly and their functional implications

Degenerative arthritis contributes to 25 % of all physical disability in the elderly. Give physio instead of NSAIDs.
Chronic Airflow Limitation
Dementia occurs in approximately 15% of elderly over 75 years.
Visual and hearing impairment . 5 % of elderly over 65, and 25 % over 85 have visual impairment. Around 5 % of elderly over 65 and 50 % over 85 have hearing impairment.
Proper medication use .. Adverse drug reactions account for 5 to 10 % of hospital admissions.
Social isolation : 40% of elderly females over 65 live alone; 55% of over 80 year olds.

The current elderly population comes from a generation that do not question medical opinion. BEWARE

GASTRIC SECRETION and its neurohormonal control



Food-stimulated acid secretion : the physiologic stimulus for acid secretion if food. Traditionally, food-stimulated acid secretion has been divided into three phases.

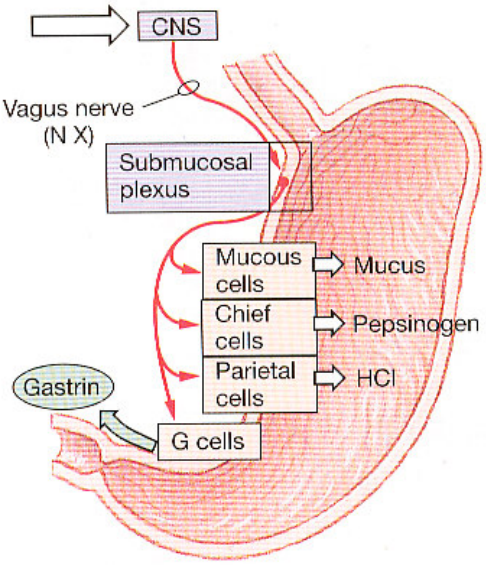
1. **Cephalic phase** acid secretion in response to the thought, sight and smell, taste is mediated by the vagus nerve. Vagal stimulation which may be elicited by sham feeding directly activates the parietal cells through muscarinic receptors and indirectly stimulates the G cells through GRP causing a small increase in gastrin.

2. **Gastric phase** of acid secretion occurs when food reaches the stomach and has two components: i) a physical component caused by distention of the stomach which stimulates modest acid secretion directly and ii) the more important component is the effect of food, primarily amino acids, in stimulating G cells to release gastrin. Gastrin release accounts for up to 90% of the gastric phase of acid secretion.

3. **Intestinal phase** begins with the entry of chyme into small intestine. The primary stimulatory factors are distention, proteins and products of protein digestion. Circulating amino acids may have a role in stimulating acid secretion without elevating gastrin levels. Under normal conditions, the intestinal phase only accounts for a small proportion of the acid secretory response to a meal.

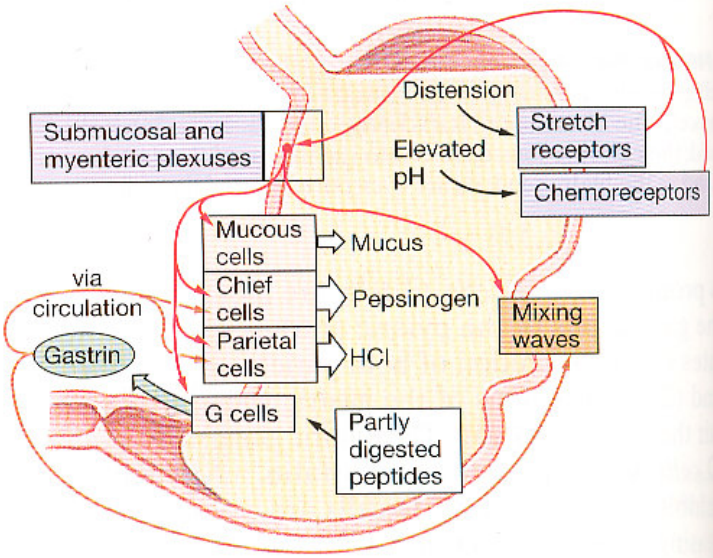
(a) The Cephalic Phase Sight, smell, taste, or thoughts of food

Function:
Prepare stomach for arrival of food
Duration:
Short (minutes)
Mechanism:
Neural, via preganglionic fibers in vagus nerve and synapses in submucosal plexus
Actions:
Primary: increased volume of gastric juice by stimulating mucus, enzyme, and acid production
Secondary: stimulation of gastrin release by G cells



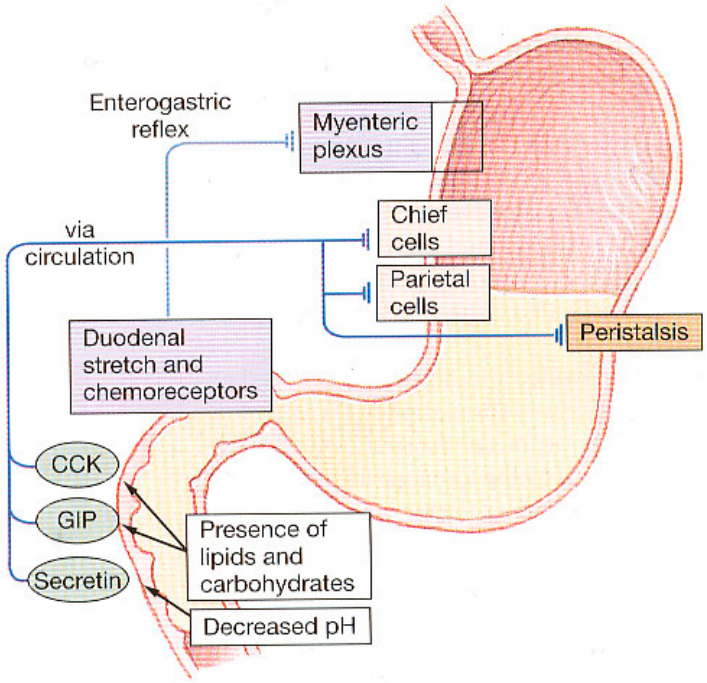
(b) The Gastric Phase

Functions:
Enhance secretion started in cephalic stage; homogenize and acidify chyme; initiate digestion of proteins by pepsin
Duration:
Long (3–4 hours)
Mechanisms:
Neural: short reflexes triggered by
(1) stimulation of stretch receptors as stomach fills
(2) stimulation of chemoreceptors as pH increases
Hormonal: stimulation of gastrin release by G cells by parasympathetic activity and presence of peptides and amino acids in chyme
Local: release of histamine by mast cells as stomach fills (not shown)
Actions:
Increased acid and pepsinogen production; increased motility and initiation of mixing waves



(c) The Intestinal Phase

Function:
Control rate of chyme entry into duodenum
Duration:
Long (hours)
Mechanisms:
Neural: short reflexes (enterogastric reflex) triggered by extension of duodenum
Hormonal:
Primary: stimulation of CCK, GIP, and secretin release by presence of acid, carbohydrates, and lipids
Secondary: release of gastrin stimulated by presence of undigested proteins and peptides (not shown)
Actions:
Feedback inhibition of gastric acid and pepsinogen production; reduction of gastric motility



Pathophysiology of Vomiting

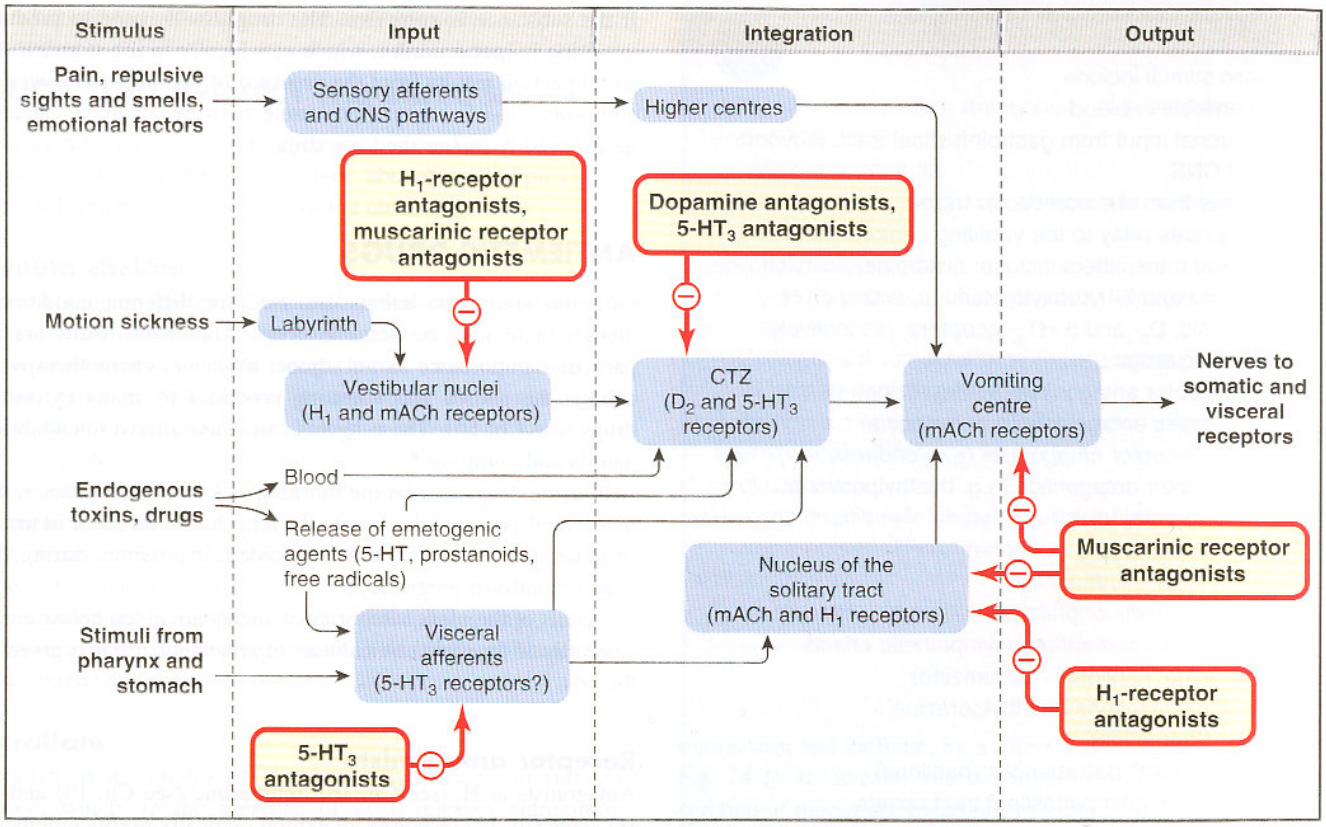
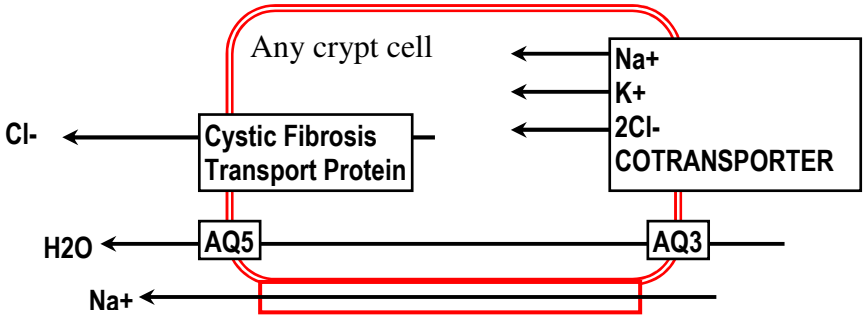


Fig. 24.5 Schematic diagram of the factors involved in the control of vomiting, with the probable sites of action of antiemetic drugs. The cerebellum may function as a second relay or gating mechanism in the link between labyrinth and CTZ (not shown). (ACh, acetylcholine; CTZ, chemoreceptor trigger zone; H_1, histamine H_1; M, muscarinic; D_2, dopamine D_2; 5-HT_3, 5-hydroxytryptamine type 3.) (Based partly on a diagram from Borison H L et al. 1981 J Clin Pharmacol 21: 235–295.)

Basic Sciences : the epithelium

Cholera toxin lock the crypt cells of the colon into their SECRETORY PHENOTYPE (i.e aquaporins are all deployed and H2O issues forth)
thus → gallons and gallons of watery diarrhoea

Take home message: H2O follows Solutes: THUS
If you eat something that you cannot absorb, you will cause DIARRHOEA



BICARBONATE SECRETING EPITHELIA:
Duodenum, pancreas, stomach, bile ducts

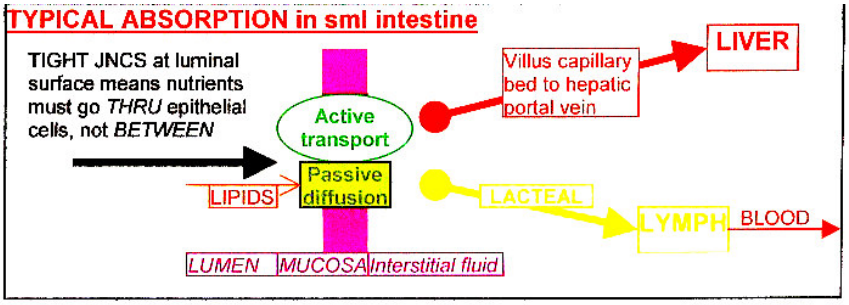
Sodium is used to co-transport glucose, amino acids:
Taking advantage of high lumen concentration
THUS: Mutant SGLT-1 = diarrhoea (cant absorb glucose or Na+)

DIGESTION & ABSORPTION OF NUTRIENTS

706_05

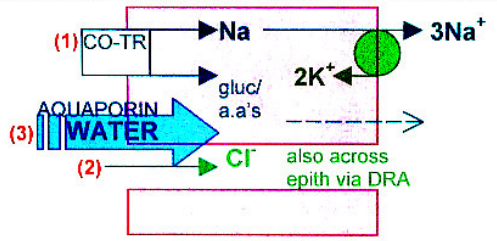
TYPICAL ABSORPTION in sml intestine

TIGHT JNCS at luminal surface means nutrients must go *THRU* epithelial cells, not *BETWEEN*



WATER & SALT – ABSORPTION @VILLUS

Water follows solutes: Na, Cl, HCO_3.
Solutes transported by ACTIVE TRANSPORT & LEAKY TIGHT JNCS
Main players: Na enters cell unwanted & pumped out by **SODIUM PUMP** (Na^+/K^+ ATPase) @ basolat membrane
(1) Na cotransported with glucose/aa OR
(2) Na exchanged for H+ etc. via NHES
→CREATES OSMOTIC GRADIENT FOR WATER TO FLOW DOWN



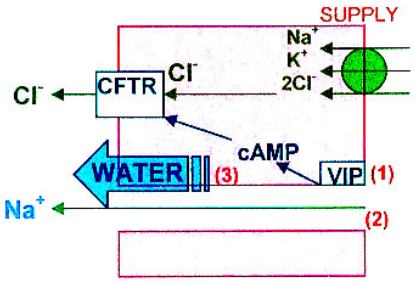
NHES mutn (no Na in), DRA mutn (no Cl in) → *DIARRHOEA*

WATER & SALT – SECRETION @CRYPT

(1) osmotic gradient estab by food in lumen OR
(2) crypt secretes NaCl, water follows

CFTR = cAMP dependent Cl channel
Main players:
SUPPLY = Na,K-2 Cl cotransporter
ACTIVATOR = VIP
CHANNEL = CFTR

→CF=CFTR mutation malabsorption
→bacterial toxins (*cholera toxin*) (1)keep cAMP high, (2)blocks villus/crypt differentiation→ MASSIVE DIARRHOEA
stops at cell turnover (days)



Bicarbonate similarly secreted by GASTRO, DUO epithelium. **SUPPLY**=Na-bicarb contransporter , **CHANNEL**=CFTR

GLUCOSE

POLYSACCHARIDES
→salivary *AMYLASE*
MOUTH
→pancreatic *AMYLASE*
SML INTESTINE

OLIGOSACCHS & DISACCHS
→brush border E's:
MALTASE, LACTASE, SUCRASE *SML INTESTINE*

MONO-SACCHS

PEPTIDES

PROTEIN
→*PEPSIN*
STOMACH (HCl)

LGE POLYPEPS
→pancreatic E's
TRYPSIN, CHYMOTRYPSIN, CARBOXYPEPTIDASE
SML INTESTINE

SML POLYPEPS / PEPTIDES
→brush border E's
SML INTESTINE

LIPIDS

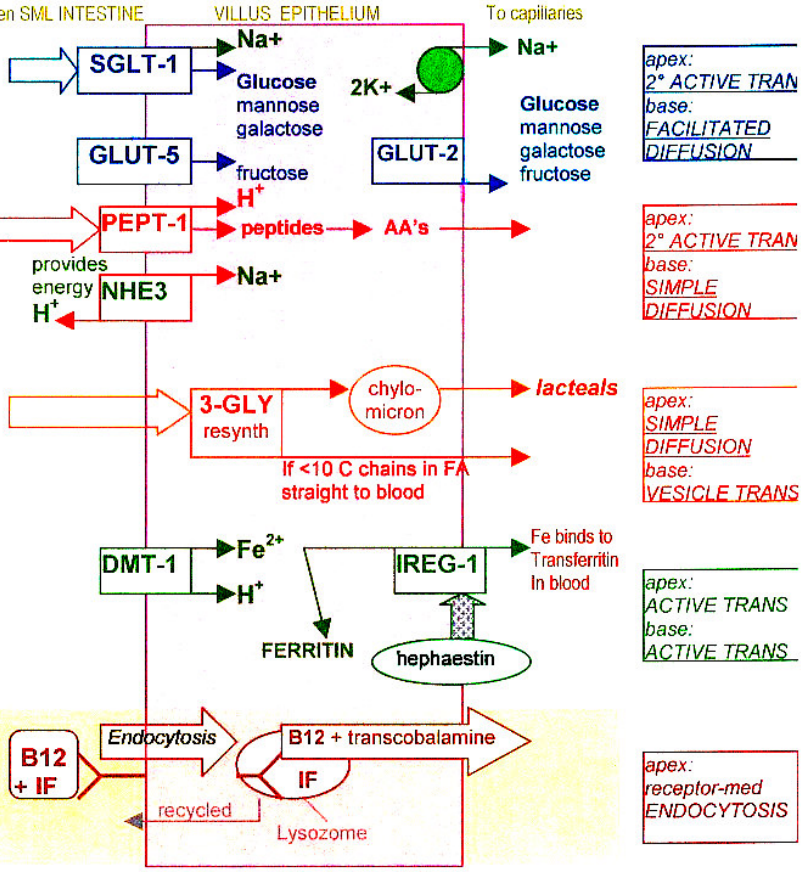
3-GLY'S (FAT GLOBULES)
→*BILE SALTS emulsification*
→*pancreatic LIPASE*
SML INTESTINE

MONO-GLY'S & FA'S (MICELLES)

IONS

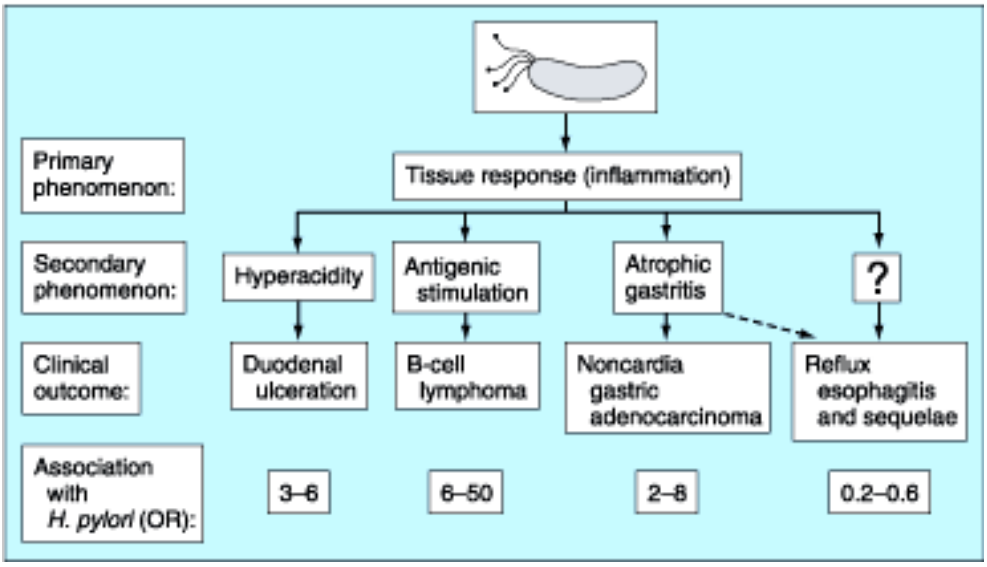
IREG-1 = Fe regulated transporter
→low Fe = open, Fe enters blood
→high Fe, stored as Ferritin
HEPHAESTIN regulates IREG expression

LARGE ORGANICS



Microbiology

H. pylori is a gram-negative, spiral, flagellate bacillus
= is noninvasive, living in gastric mucus
motile in the mucous environment,
urease protects it against acid by catalyzing urea hydrolysis to produce
→ **buffering ammonia.**←
H. pylori is microaerophilic and slow-growing and requires complex growth media.



Humans are the only important reservoir of H. pylori.
colonization is particularly common in childhood institutions.

The two major disease-associated H. pylori virulence factors described so far are
- **a vacuolating cytotoxin, VacA,**
- **a group of genes termed the cag pathogenicity island (*cag* PaI).**
  =*genes that confer enhanced virulence on* ***H. pylori*** *strains, at least partly by inducing epithelial cells to produce proinflammatory cytokines.*

primary peristalsis *is trigered by voluntary swallowing*
secondary peristalsis *is triggered by distension*

UPPER GI MOTILITY

Liquid fastest ; ~30 min
Fat slowest, ~2-3 hrs

upper oesophageal sphincter receives tonic excitatory innervation,
→ relaxes transiently to receive the bolus
→ constricts by reflex BEHIND the bolus
→ this wave sweeps down the oesophagus
The lower oesophageal sphincter is always tonically contracted
it also receives excitatory cholinergic innervation.
When a swallowing effort is made, non-adrenergic, noncholinergic inhibitory nerves to the lower oesophageal sphincter cause it to relax and admit the bolus into the stomach

food arrives in the stomach→
→ stretch receptors in the wall are activated
via a vago-vagal reflex, the smooth muscle of the *proximal stomach* relaxes *(adaptive relaxation)*
Within a few minutes of the start of a meal, regular peristaltic contractions (at a rate of three per minute) develop in the *distal stomach;*
these contractions mix and grind the solid food and transport it to the pylorus.
The muscle of the pylorus contracts in such a way that only liquids and tiny (approx. 2mm) food particles can pass through, provided there is a pressure gradient between the stomach and duodenum;
duodenal chemoreceptors prevent chyme emptying too rapidly.
After a mixed solid - liquid meal, the liquid component leaves the stomach at an approximately exponential rate,
with a mean half-emptying time of about 20 minutes.
The solid component empties, after a lag phase of about 5-20 minutes,
in linear fashion, with a mean half-emptying time of about 90 minutes.

FUNCTIONS OF THE GI

Saliva:

- Lubricant; protects teeth **(a mucoprotein)**
- High levels of Ca++, phosphate→ **KEEPS TEETH FROM DISSOLVING**
- **50-60 mmol/L of bicarbonate to neutralise bacterial acid**
- has some enzyme (eg. amylase) but no realdigestion happens here.
    - **AMYLASE** is really there to dissolve the polysaccharides so that bacteria cant use them for food (and swallowing will flush them away)
- ALSO HAS **LYSOZYME**: kills bacteria by dissolving their walls
    - **GROWTH FACTORS** to keep the oral mucosa healthy

Phase Triggers:
Cephalic: sight, smell of food
Gastric: food @ stomach
Intestinal: food @ upper gut

- **Salivary secretion is under both sympathetic and parasympathetic control:**
- **PARA SYMPATHETIC= thin watery saliva**
- **SYMPATHETIC = thick gooey saliva**

@ cephalic phase, both are secreted

-

Oesophagus:

Gut activity is regulated by its contents

Upper 2/3rds are under CNS control
Lower 1.3rd are internal nervous network
Downstream distension causes upstream contraction
ITS NOT REAL PERISTALSIS: "I cant believe its not peristalis"
→ because oesophagus requires spinal reflexes to contract

Stomach:

why all the acid? Barely any digestive properties…
BUT IT KEEPS THE GERMS OUT
Pepsin is the main worker, (pepsinogen→ activated by contact with low pH)
→endoprotease, breaks up proteins into petides to stimulate lots of GI stuff eg. gastrin secretion

STOMACH ONLY CARES ABOUT STOMACH:
produces self-preserving MUCUS which is a barrier
H+ tries to get in, HCO3- tries to get out, they meet half-way and neutralise each other
= MUCOSAL pH GRADIENT!! pH 1 @ lumen becomes pH 8 @ epithelium!!
REGULATED BY:
GASTRIN (for acid)
Cholecystokinin (reduces emptying rate)
+ many other Enteroendocrine hormones

Small Intestine:

The CLOSER to the duodenum, The more ACTIVE the transport

DUODENUM = a REACTION VESSEL:
Gastric contents + HCO3- to neutralise + Enzymes + BILE
(HCO3 comes from bile duct and pancreatic juices)
All those pancreatic enzymes are secreted as PRECURSORS which are then activated by ENTEROPEPTIDASES @ DUODENAL BRUSH BORDER
… this way, the pancreas does not digest itself
PLUS: brush border enzymes finish the job of digesting the small fragments of peptides
Cryps secrete, villi absorb.
As the cells migrate from the stem cells of the crypt up into the villus, they acquire brush-border enzymes.

BOWEL:

Boring job of storing faeces. Kept mainly in the caecum or rectum.
When triggered by eating, caecum content is pushed to the rectum.
MAINLY ABSORBS H2O and SALT
Like distal tubule, the colon removes ALL SALT before releasing its contents

PATHOGENESIS:

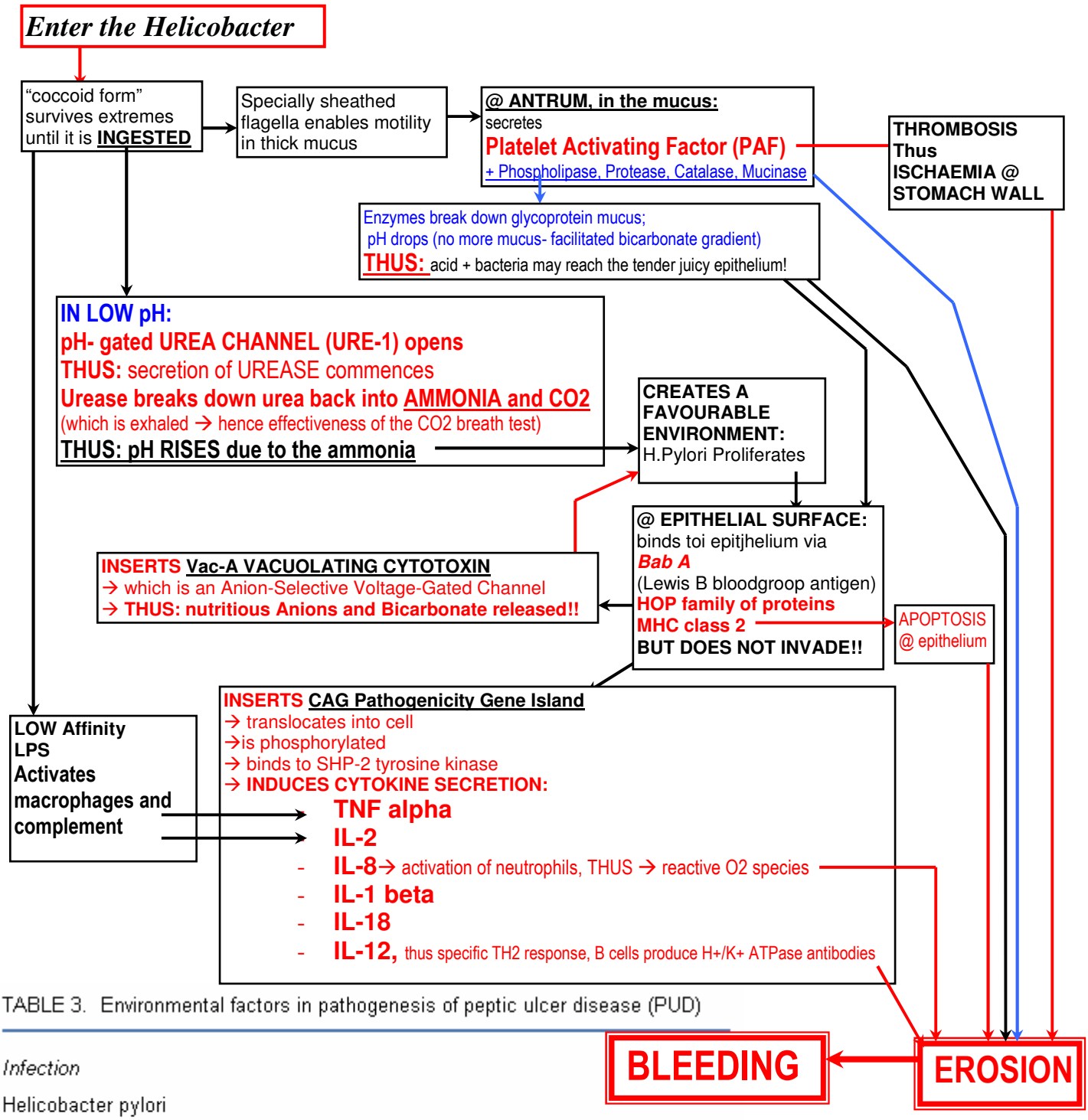


TABLE 3. Environmental factors in pathogenesis of peptic ulcer disease (PUD)

Infection
Helicobacter pylori
Drugs
NSAIDs
Smoking
Prevalence of PUD
Healing of duodenal ulcer (DU) and gastric ulcer (GU)
Death rates from PUD
Alcohol and caffeine-containing beverages
Acid secretion