Principles of Drug Therapy of Upper GI Disorders

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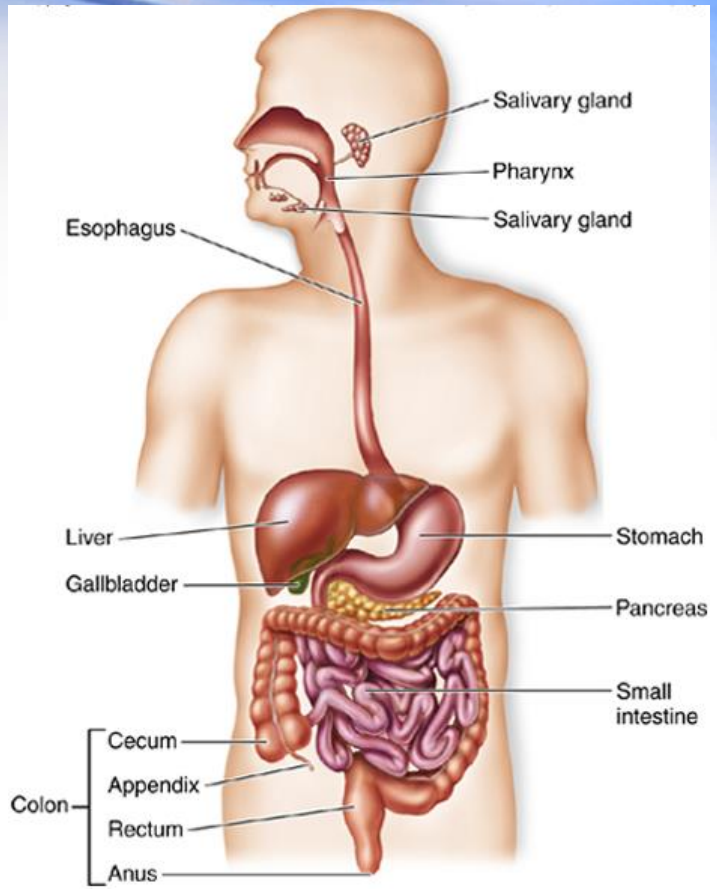
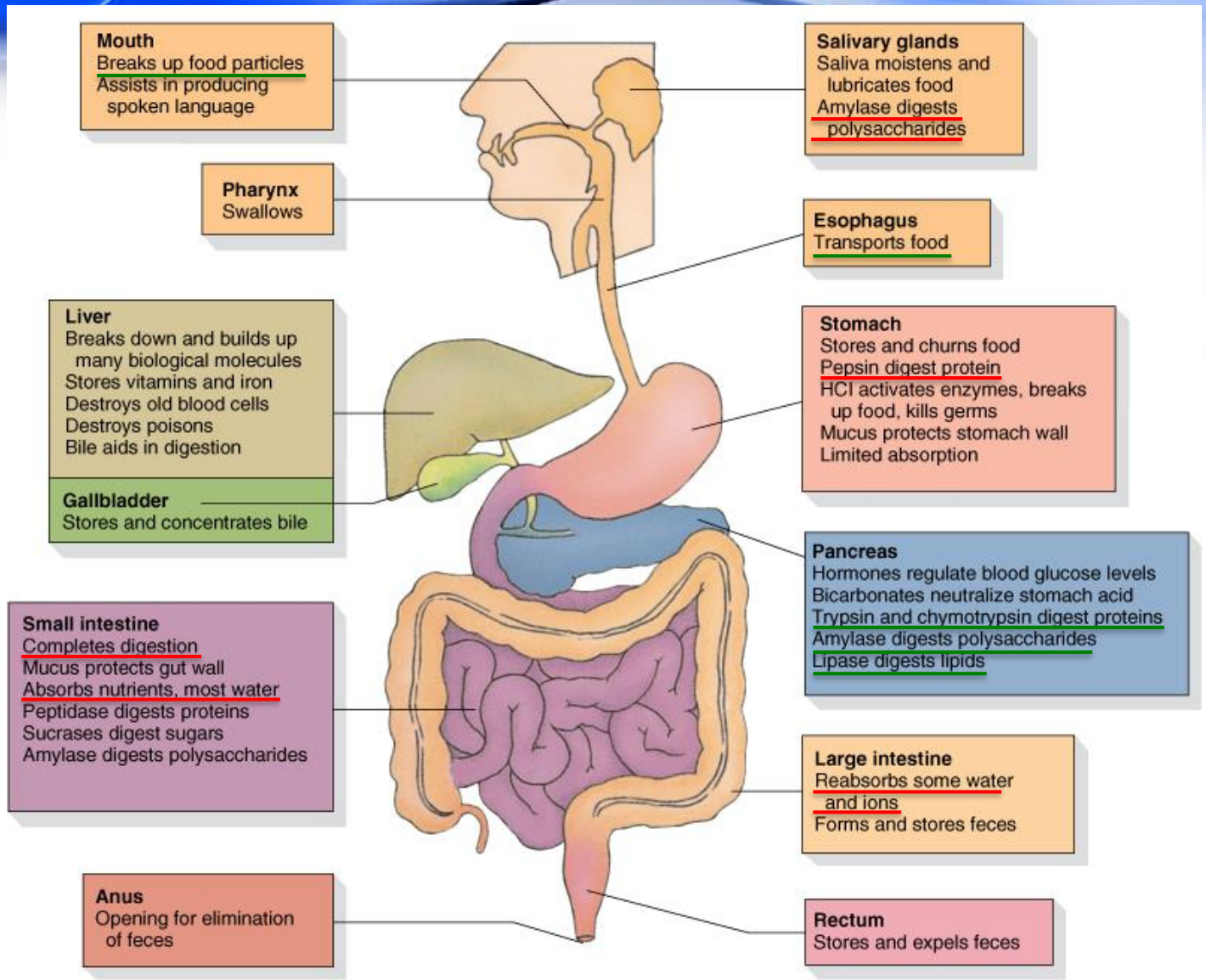
THE GASTROINTESTINAL TRACT - function

- ✓ *Ingestion of food*
- ✓ *Digestion*
  - *mechanical digestion of food particles*
  - breaks up food particles
- ✓ *Motility*
  - movements of organs and food
  - *mechanical digestion of food particles*
- ✓ *Secretion*
  - secretion of digestive juices
  - chemical digestion of food particles
- ✓ *Absorption*
  - absorption of digestion products to blood or lymphatic vessels
- ✓ *Storage and Elimination*
  - non-digested food particles

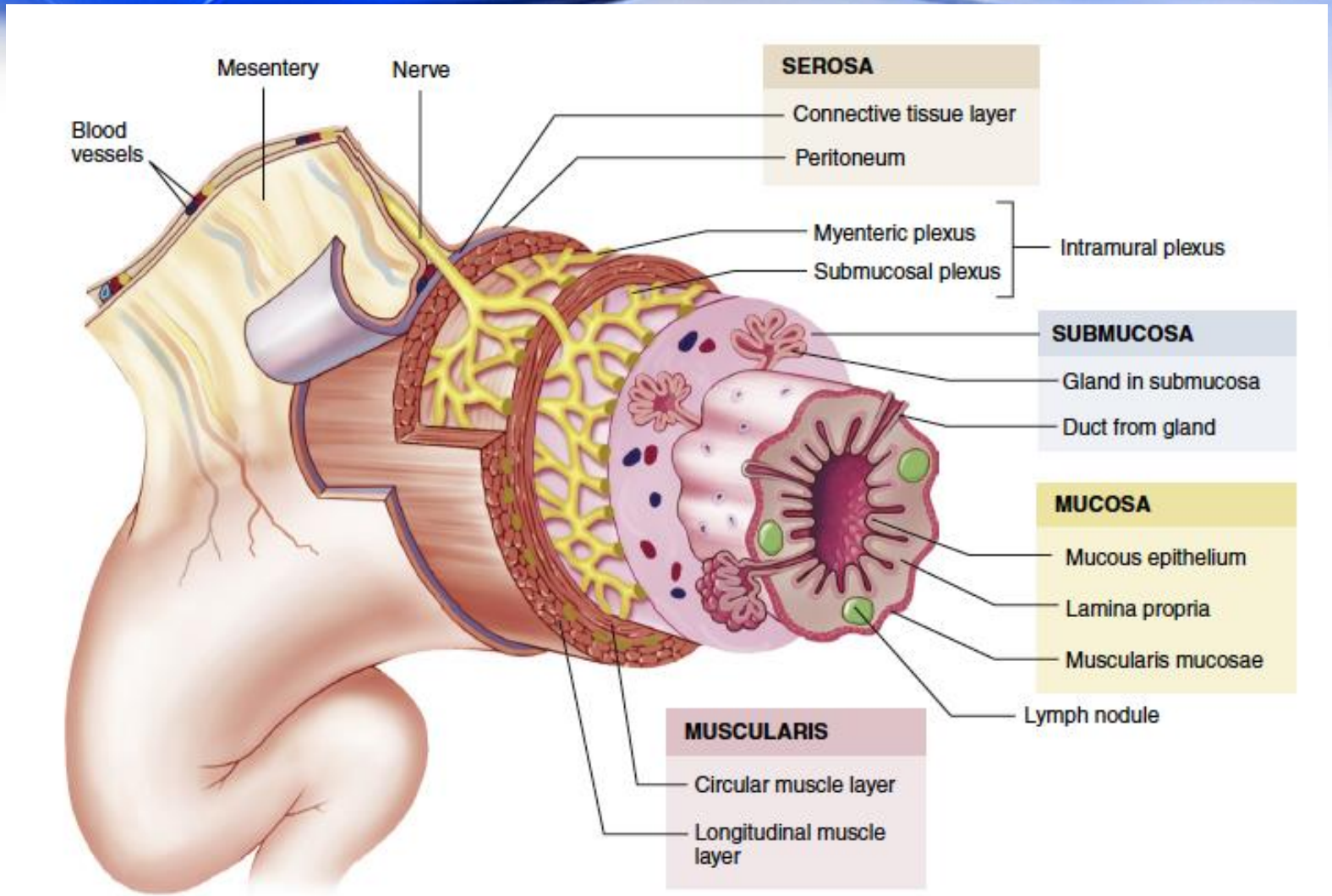


- ✓ *Protective function – mechanical, chemical, immunological*
  - not only GIT organs but also the body as a whole, against the potential harmful food components

THE GASTROINTESTINAL TRACT - function



THE GASTROINTESTINAL TRACT - structure



DISORDERS OF THE DIGESTIVE SYSTEM

- ***Disorders of the digestive system have serious consequences for the activity of the organism as a whole***

  - ✓ congenital malformations
  - ✓ inflammatory processes
  - ✓ infectious processes
  - ✓ traumatic processes
  - ✓ neoplastic processes

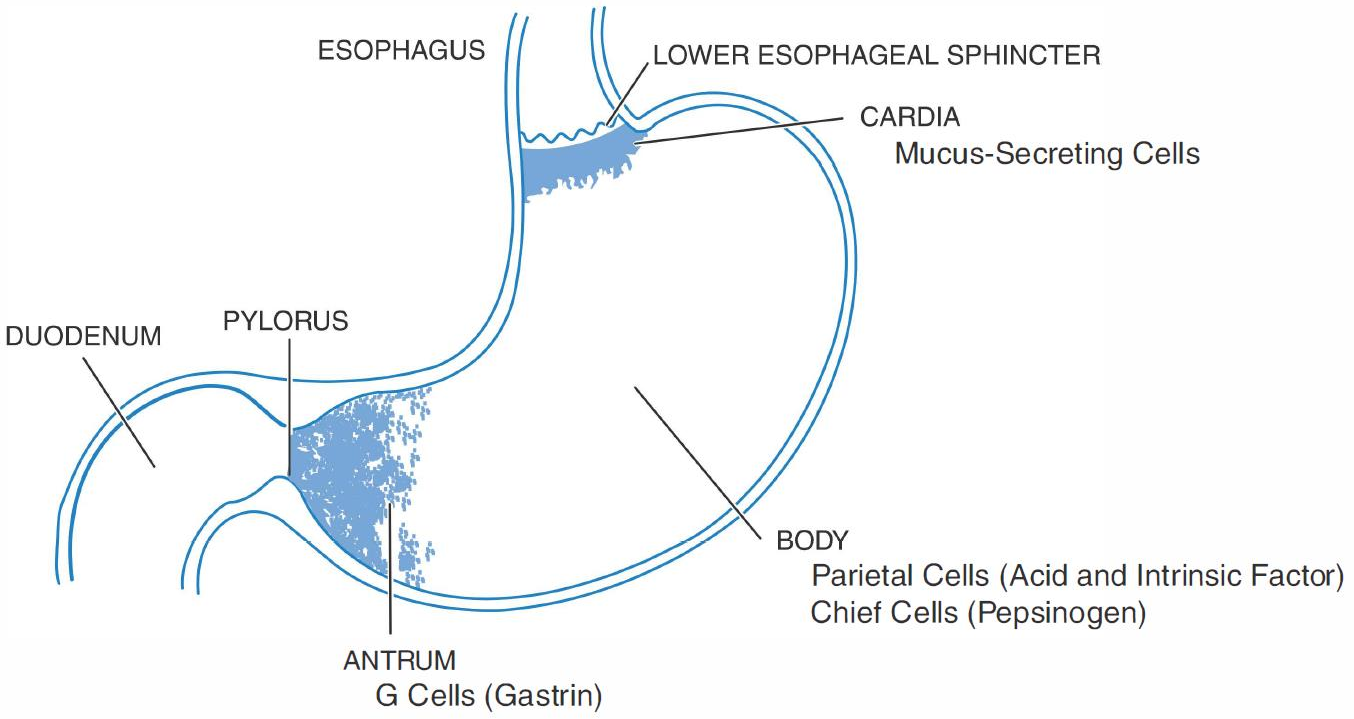
- ***Digestive system communicates with the external environment through the intake of fluids and food***

  - ✓ Toxic substances in food and fluids
  - ✓ GIT itself contains toxic substances
    - secretion components - enzymes, HCl
    - waste products of digestion of food, bacterial flora

THE MOST COMMON DISORDERS of the digestive system

- ✓ ***Motor dysfunction of smooth muscle*** *of the individual parts of the digestive system*
- ✓ ***Indigestion*** *of food and* ***absorption*** *of nutrients - malabsorption syndrome*
- ✓ ***Bleeding*** *into the individual parts of the digestive tract*
- ✓ ***Perforation of the wall of the digestive system*** *with subsequent leakage of the contents to the peritoneal cavity*
- ✓ ***Obstruction*** *in moving of the contents of one part of the digestive system to the next section*
- ✓ ***Circulation*** *disorders in the wall of the individual parts of the digestive system*

PHYSIOLOGY OF THE UPPER GASTROINTESTINAL TRACT



Clinical manifestations of GI dysfunction - DYSPEPSIA (malfunction of digestion)

Symptoms	*Malfunction*	*Disease*
✓ abdominal pain	✓ esophagus	✓ peptic ulcer
✓ feeling of imperfect digestion	✓ stomach	✓ long-lasting reflux of stomach contents into the esophagus
✓ bloating	✓ duodenum	✓ gastritis
✓ nausea		

- frequently it is functional (non-ulcer) dyspepsia
- ***dyspepsia similar to ulcer symptomatology:*** pain predominates
- ***dyspepsia similar to dysmotility symptomatology:*** nausea, vomiting, bloating
- For individual diseases of the upper GI, these ***symptoms can be combined in various ways***

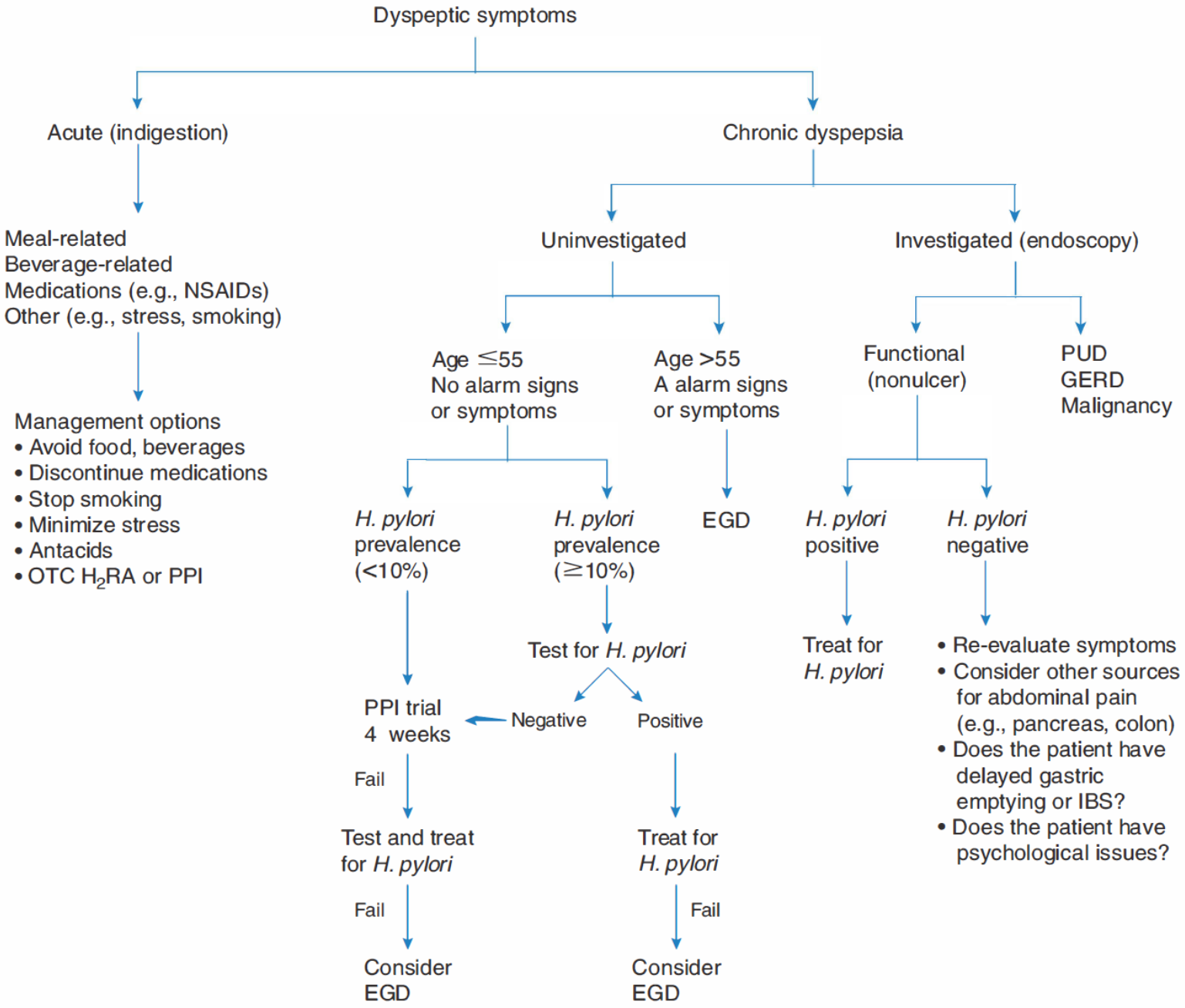
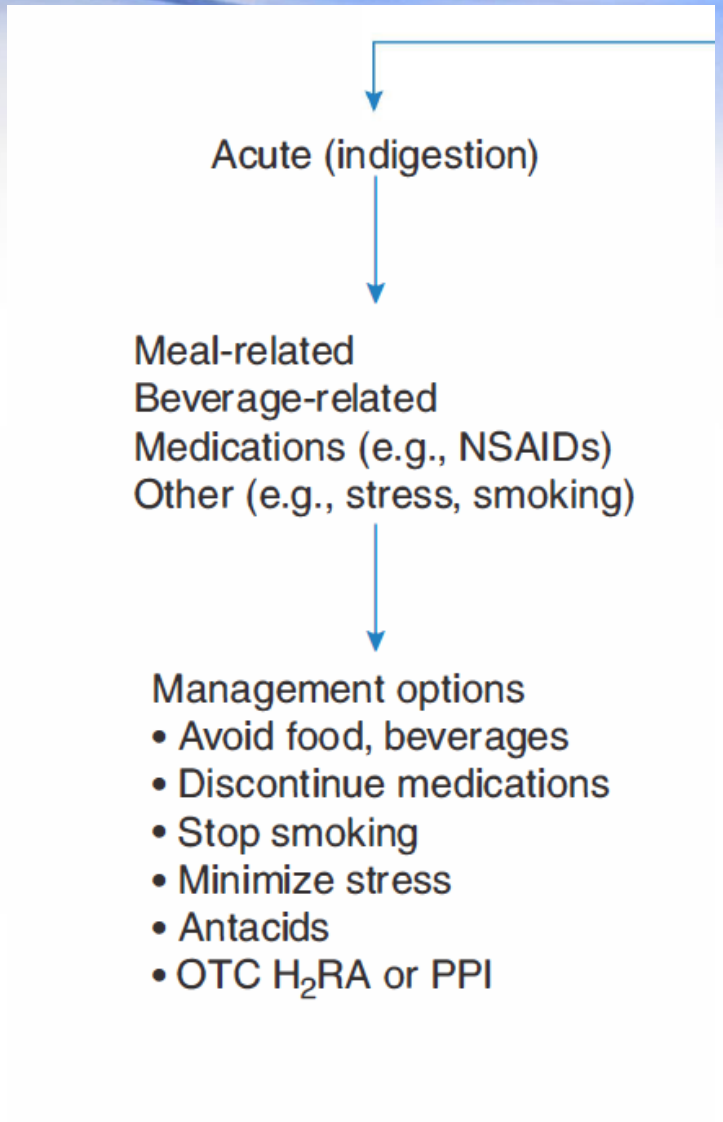

Dyspeptic symptoms
Acute (indigestion)
Chronic dyspepsia
Meal-related
Beverage-related
Medications (e.g., NSAIDs)
Other (e.g., stress, smoking)
Management options
• Avoid food, beverages
• Discontinue medications
• Stop smoking
• Minimize stress
• Antacids
• OTC H2RA or PPI
Uninvestigated
Investigated (endoscopy)
Age ≤55
No alarm signs
or symptoms
Age >55
A alarm signs
or symptoms
Functional
(nonulcer)
PUD
GERD
Malignancy
H. pylori
prevalence
(<10%)
H. pylori
prevalence
(≥10%)
EGD
H. pylori
positive
H. pylori
negative
Test for H. pylori
PPI trial
4 weeks
Negative
Positive
Treat for
H. pylori
• Re-evaluate symptoms
• Consider other sources
for abdominal pain
(e.g., pancreas, colon)
• Does the patient have
delayed gastric
emptying or IBS?
• Does the patient have
psychological issues?
Fail
Test and treat
for H. pylori
Treat for
H. pylori
Fail
Fail
Consider
EGD
Consider
EGD

Table 23-2

Indications for Testing and Treating *Helicobacter pylori* Infection

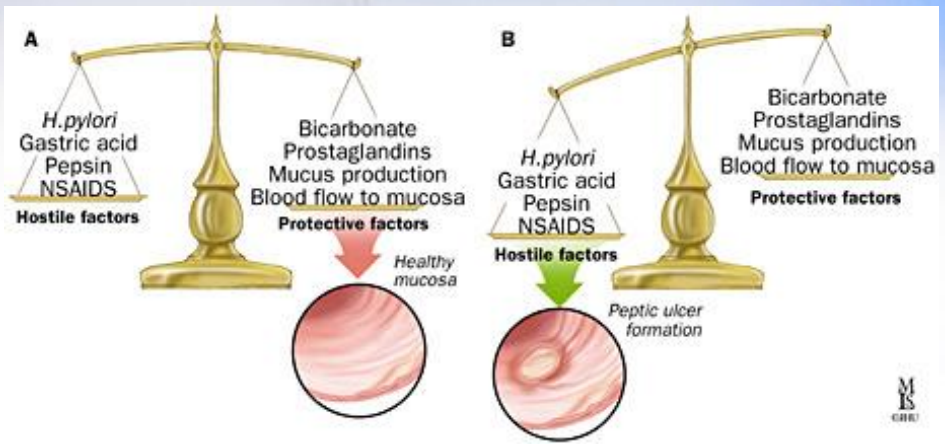
Recommended (Evidence Established)
■ Uninvestigated dyspepsia (depending on *H. pylori* prevalence)
■ PUD (active gastric or duodenal ulcer)
■ History of PUD (confirmed ulcer not previously treated for *H. pylori*)
■ Gastric MALT lymphoma
■ After resection of early gastric cancer
■ Reduce the risk of recurrent bleeding from gastroduodenal ulcer
Controversial (Evidence Not Well Established)
■ NUD
■ Individuals using NSAIDs (no signs/symptoms of peptic ulcer)
■ GERD
■ Individuals at risk for gastric cancer
■ Individuals with unexplained iron deficiency anemia

Treatment

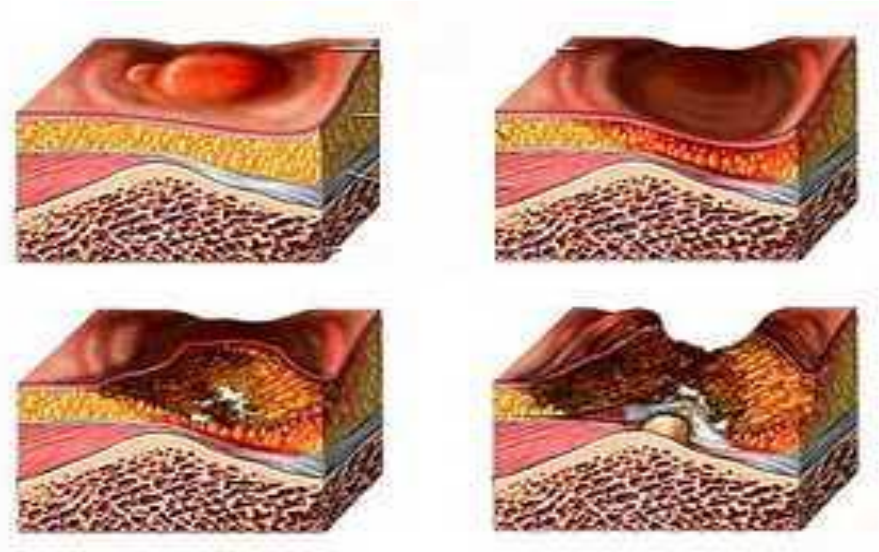

Acute (indigestion)
Meal-related
Beverage-related
Medications (e.g., NSAIDs)
Other (e.g., stress, smoking)
Management options
• Avoid food, beverages
• Discontinue medications
• Stop smoking
• Minimize stress
• Antacids
• OTC H_2RA or PPI

Disorders of the GIT – PEPTIC ULCER

- is a result of ***imbalance*** between the ***mucosal defense mechanisms*** in the esophagus, stomach and duodenum, and ***gastric mucosa-damaging mechanisms***



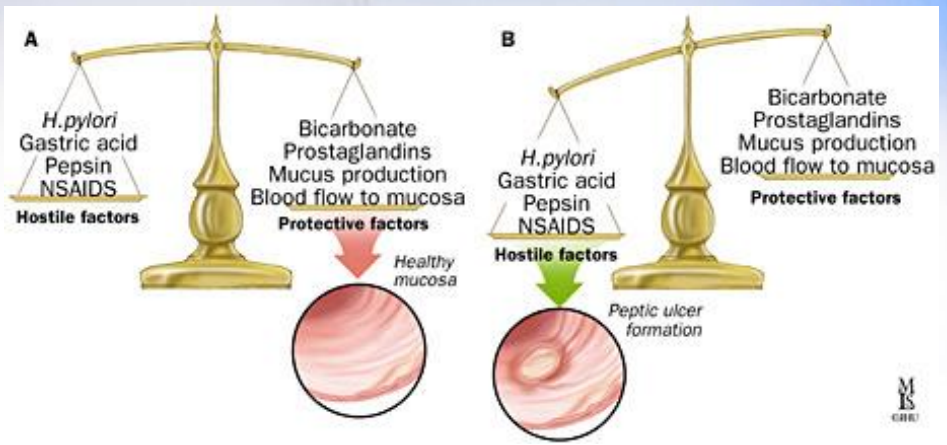
- relates to digestion of *mucous membrane and lower parts of the stomach, duodenum, and lower esophagus* by HCl and pepsin

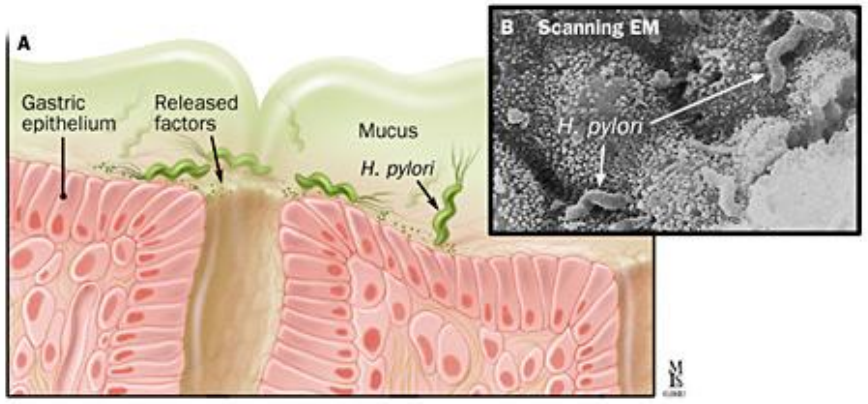


PEPTIC ULCER

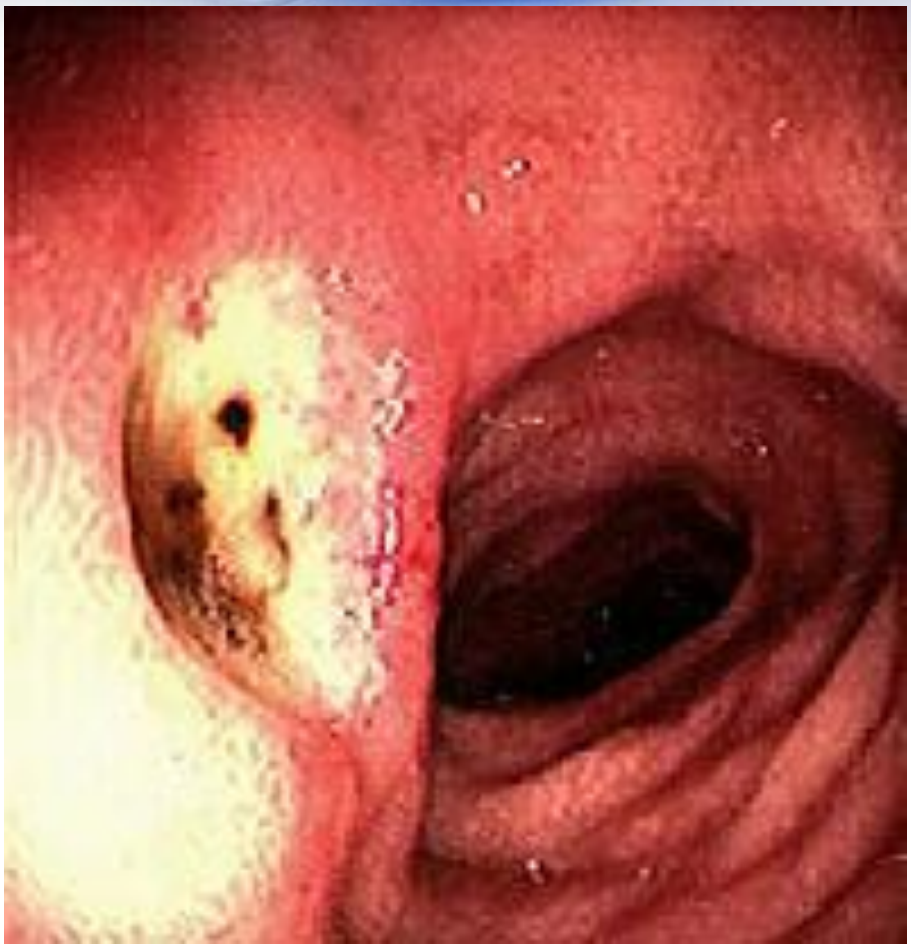
Risk factors for peptic ulcer disease:

- genetic predisposition
- H.pylori infection of the gastric mucosa
- age greater than 65 years
- psychologic stress (mechanism unknown)
- excessive use of alcohol
- smoking
- acute pancreatitis
- chronic obstructive pulmonary disease
- obesity
- cirrhosis





PEPTIC ULCER



PEPTIC ULCER

CHARACTERISTICS	GASTRIC ULCER	DUODENAL ULCER
Incidence		
Age at onset	50-70 years	20-50 years
Family history	Usually negative	Positive
Gender (prevalence)	Equal in women and men	Equal in women and men
Stress factors	Increased	Average
Ulcerogenic drugs	Normal use	Increased use
Cancer risk	Increased	Not increased
Pathophysiology		
Helicobacter pylori infection	Often present (60-80%)	Often present (95-100%)
Abnormal mucus	May be present	May be present
Parietal cell mass	Normal or decreased	Increased
Acid production	Normal or decreased	Increased
Serum gastrin	Increased	Normal
Serum pepsinogen	Normal	Increased
Associated gastritis	More common	Usually not present
Clinical Manifestations		
Pain	Located in upper abdomen	Located in upper abdomen
	Intermittent	Intermittent
	Pain-antacid-relief pattern	Pain-antacid or food-relief pattern
	Food-pain pattern	Nocturnal pain common
Clinical course	Chronic ulcer without pattern of remission and exacerbation	Pattern of remissions and exacerbations for years

PEPTIC ULCER

- ***Types of Peptic Ulcers:***

acute - quickly heal by the mucosa regeneration

chronic - penetrate deeper into the tissue, healing takes several weeks or months

- ***Special types of ulcers:***

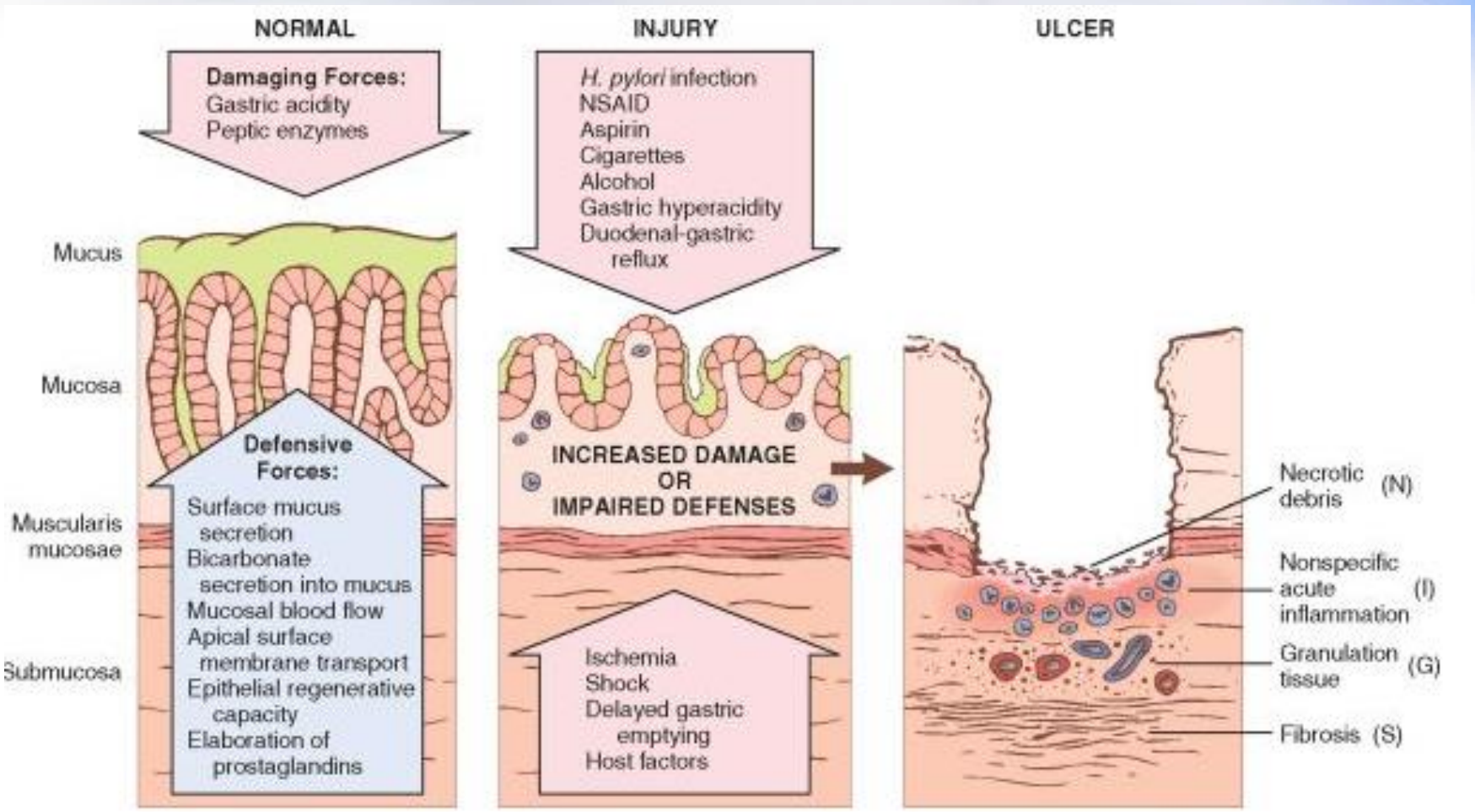
Cushing - traumatic origin, or after surgery CNS (irritation of n. Vagus -> hypersecretion HCl)

Curling - traumatic origin, after burns (↑ levels of histamin -> hypersecretion HCl)

Zollinger - Ellison Syndrome - ↑ production of gastrin -> stimulates the secretion of HCl)

Stress ulcers - mucosal perfusion defect

PEPTIC ULCER



EPITHELIAL GASTRODUODENAL BARRIER

- ***Mucus-bicarbonate barrier***
  - smooth adhesive mucus layer
  - pH gradient (lumen – epithelial surfice)
  - bicarbonate secretion by epithelial cells

- ***H^+ disposal in gastric wall***
  - mucoid barrier damage
  - back diffusion of H^+ into the wall
  - *mucosal blood flow*

- ***Proliferation and epithelial repair***
  - mitosis and cell migration along the basal membrane
  - mucoid cap after epithelial damage

Table 23-3

Risk Factors for Nonsteroidal Anti-Inflammatory Drug–Induced Ulcer and Ulcer-Related Upper Gastrointestinal Complications

Established
■ Confirmed prior ulcer or ulcer-related complication
■ Age >65 years
■ Multiple or high-dose NSAID use
■ Concomitant use of aspirin (including low cardioprotective dosages, e.g., 81 mg)
■ Concomitant use of an anticoagulant, corticosteroid, bisphosphonate, clopidogrel, or SSRI
■ Selection of NSAID (selectivity of COX-1 vs. COX-2)
Controversial
■ *H. pylori*
■ Alcohol consumption
■ Cigarette smoking

PEPTIC ULCER DISEASE

- ✓ Clinical presentation
- ✓ Complication

Table 23-4

Selected Nonsteroidal Anti-Inflammatory Drugs

Salicylates
Acetylated: aspirin
Nonacetylated: trisalicylate, salsalate
Nonsalicylates[a]
Nonselective (traditional) NSAIDs: ibuprofen, naproxen, tolmetin, fenoprofen, sulindac, indomethacin, ketoprofen, ketorolac, flurbiprofen, piroxicam
Partially selective NSAIDs: etodolac, diclofenac, meloxicam, nabumetone
Selective COX-2 inhibitors: celecoxib[b], rofecoxib[c], valdecoxib[c]

[a]Based on COX-1/COX-2 selectivity ratio in vitro

Table 23-5

Diagnostic Tests for *Helicobacter pylori* Infection

Tests Using Gastric Mucosal Biopsy in Patients Undergoing Endoscopy
Rapid Urease Test
■ Tests for active *H. pylori* infection; >90% sensitivity and specificity. ■ In the presence of *H. pylori* urease, urea is metabolized to ammonia and bicarbonate resulting in an increase in pH, which changes the color of a pH-sensitive indicator. ■ Results are rapid (within 24 hours), and test is less expensive than histology or culture. ■ Withhold H_2RAs and PPIs 1–2 weeks before testing and antibiotics and bismuth salts 4 weeks before testing to reduce the risk of false negatives.
Histology
■ "Gold standard" for detection of active *H. pylori* infection; >95% sensitive and specific. ■ Permits further histologic analysis and evaluation of infected tissue (e.g., gastritis, ulceration, adenocarcinoma); tests for active *H. pylori* infection. ■ Results are not immediate; not recommended for initial diagnosis; more expensive than rapid urease test.
Culture
■ Permits sensitivity testing to determine antibiotic choice or resistance; 100% specific; tests for active *H. pylori* infection. ■ Use usually limited to patients who fail initial course of eradication therapy.
Polymerase Chain Reaction
■ Detects *H. pylori* DNA in gastric tissue; highly specific and sensitive. ■ High rate of false positives and false negatives; positive DNA does not correlate directly with presence of the organism; used primarily for research.

Table 23-5

Diagnostic Tests for Helicobacter pylori Infection (*continued*)

Nonendoscopic Tests That Do Not Use Gastric Mucosal Biopsy

Urea Breath Test

- Tests for active *H. pylori* infection; >95% sensitive and specific.
- Radiolabeled urea with either C^{13} or C^{14} is given orally; urease secreted by *H. pylori* in the stomach (if present) hydrolyzes radiolabeled urea to produce radiolabeled CO_2, which is exhaled and then quantified from the expired breath; radiation exposure is minimal.
- Withhold H_2RAs and PPIs 1–2 weeks before testing and antibiotics and bismuth salts 4 weeks before testing to reduce the risk of false negatives.
- Used to detect *H. pylori* before treatment and to document posttreatment eradication.
- Results usually take about 2 days; less expensive than tests that utilize gastric mucosal biopsy but more expensive than serologic tests; availability and reimbursement is inconsistent.

Antibody Detection (In-Office or Near Patient)

- Qualitative test; detects IgG antibodies to *H. pylori* in whole blood or fingerstick.
- Effective for primary diagnosis, but not of benefit in confirming eradication because antibodies to *H. pylori* remain positive for years after successful eradication of the infection.
- Results obtained quickly (usually within 15 minutes) but reduced sensitivity and specificity compared with laboratory-based tests; widely available and inexpensive.
- Results not affected by H_2RAs, PPIs, or bismuth; antibiotics given for other indications may result in a positive antibody test.

Antibody Detection (Laboratory)

- Quantitative test; detects IgG antibodies to *H. pylori* in serum using laboratory-based ELISA tests and latex agglutination techniques.
- More accurate than in-office tests; similar sensitivity and specificity to rapid urease biopsy and urea breath tests.
- Unable to determine if antibody is related to active or cured infection; antibody titers vary between individuals and take up to 6 months to 1 year to return to the uninfected state.
- Results not affected by H_2RAs, PPIs, or bismuth; antibiotics given for other indications may result in a positive antibody test.

Fecal Antigen Test

- An enzymatic immunoassay test that identifies *H. pylori* antigen in stool; sensitivity and specificity comparable to the UBT for initial diagnosis.
- H_2RAs, PPIs, antibiotics, and bismuth may cause false-negative results but to a lesser extent than the UBT.
- Considered an alternative to detecting *H. pylori* before treatment and documenting posttreatment eradication; patients may have a reluctance to obtain stool samples.

Table 23-6

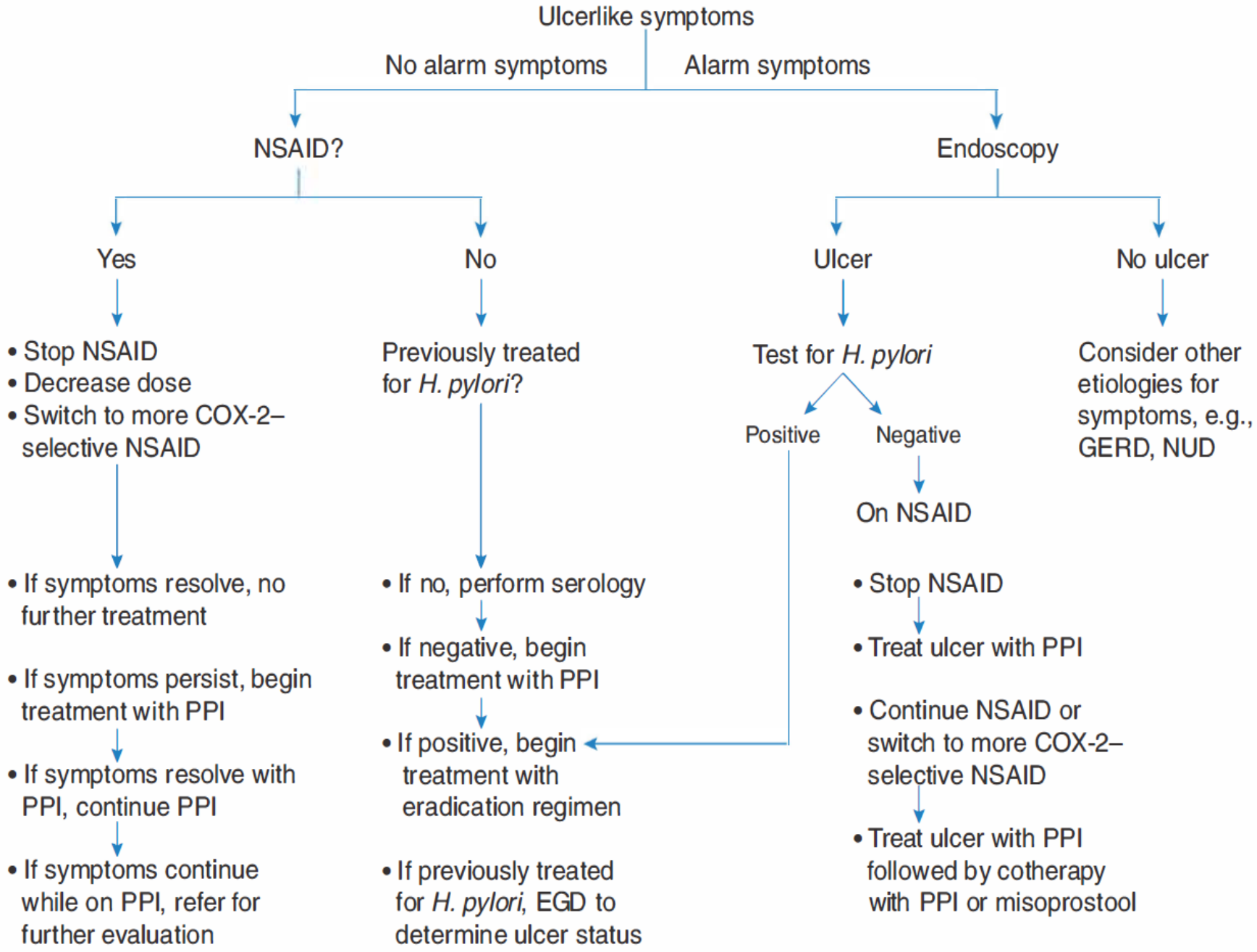
Oral Drug Regimens Used to Eradicate *Helicobacter pylori* Infection

Drug Regimen	Dose	Frequency	Duration
Proton-Pump Inhibitor–Based Three-Drug Regimens			
PPI	Standard dose[a]	BID[a]	14 days[b]
Clarithromycin	500 mg	BID	14 days[b]
Amoxicillin[c]	1 g	BID	14 days[b]
Or			
PPI	Standard dose[a]	BID[a]	14 days[b]
Clarithromycin	500 mg	BID	14 days[b]
Metronidazole[c]	500 mg	BID	14 days[b]
Bismuth-Based Four-Drug Regimens			
Bismuth subsalicylate[d]	525 mg	QID	10–14 days
Metronidazole	250–500 mg	QID	10–14 days
Tetracycline plus	500 mg	QID	10–14 days
PPI	Standard dose[a]	Daily or BID[a]	10–14 days
Or			
H_2RA[e]	Standard dose[e]	BID[e]	4–6 weeks
Sequential Therapy[f]			
PPI	Standard dose[a]	BID[a]	Days 1–10
Amoxicillin	1 g	BID	Days 1–5

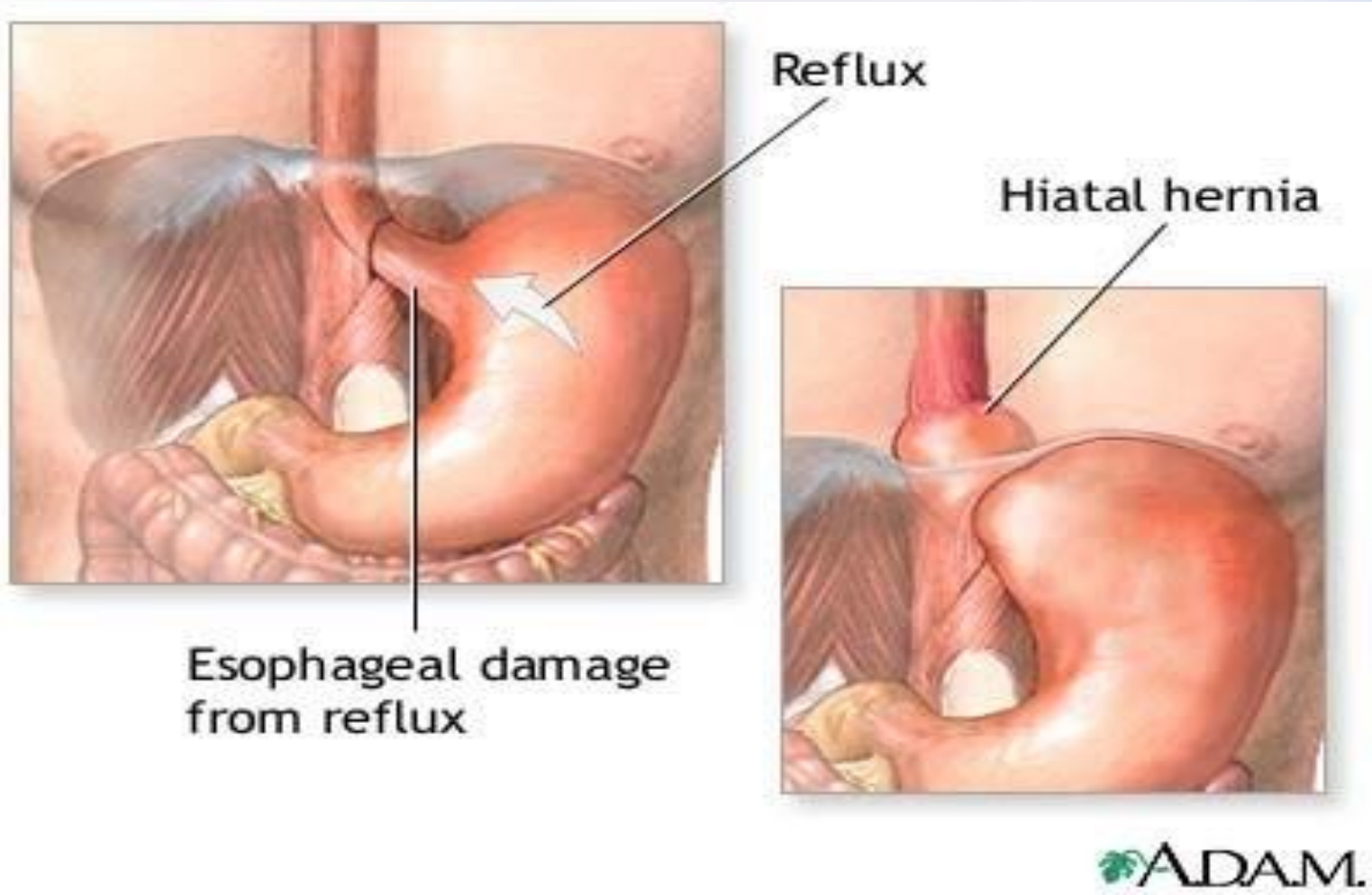
Table 23-6

Oral Drug Regimens Used to Eradicate *Helicobacter pylori* Infection (*continued*)

Drug Regimen	Dose	Frequency	Duration
Clarithromycin	250–500 mg	BID	Days 6–10
Metronidazole	250–500 mg	BID	Days 6–10
Secondary or Rescue Therapy			
Bismuth subsalicylate[d]	525 mg	QID	10–14 days
Metronidazole	500 mg	QID	10–14 days
Tetracycline	500 mg	QID	10–14 days
PPI	Standard dose[a]	Daily or BID[a]	10–14 days
Or			
PPI	Standard dose[a]	BID[a]	10–14 days
Amoxicillin	1 g	BID	10–14 days
Levofloxacin	500 mg	Daily	10–14 days


Ulcerlike symptoms
No alarm symptoms
Alarm symptoms
NSAID?
Endoscopy
Yes
No
Ulcer
No ulcer
• Stop NSAID
• Decrease dose
• Switch to more COX-2–selective NSAID
Previously treated for H. pylori?
Test for H. pylori
Positive
Negative
Consider other etiologies for symptoms, e.g., GERD, NUD
On NSAID
• If symptoms resolve, no further treatment
• If symptoms persist, begin treatment with PPI
• If symptoms resolve with PPI, continue PPI
• If symptoms continue while on PPI, refer for further evaluation
• If no, perform serology
• If negative, begin treatment with PPI
• If positive, begin treatment with eradication regimen
• If previously treated for H. pylori, EGD to determine ulcer status
• Stop NSAID
• Treat ulcer with PPI
• Continue NSAID or switch to more COX-2–selective NSAID
• Treat ulcer with PPI followed by cotherapy with PPI or misoprostool

GASTROESOPHAGEAL REFLUX DISEASE



- GERD is a common acid-related GI disorder associated with a wide array of symptoms, the most frequent of which is heartburn and acid regurgitation.

Etiology and risk factor

Table 23-7

Risk Factors Associated With Gastroesophageal Reflux Disease[6,8,153–155,166,167]

Drugs	Dietary
α-Adrenergic agonists	Foods high in fat
Anticholinergics	Spicy foods
Aspirin	Carminatives (peppermint, spearmint)
Barbiturates	Chocolate
Benzodiazepines	Caffeine (coffee, tea, colas)
β_2-Adrenergic agonists	Garlic or onions
Bisphosphonates	Citrus fruits and juices
Calcium-channel blockers	Tomatoes and juice
Dopamine	Carbonated beverages
Estrogen	
Isoproterenol	**Lifestyle**
Iron	Cigarette/cigar smoke
Narcotics	Obesity
Nitrates	Supine body position
NSAIDs	Tight-fitting clothing
Progesterone	Heavy exercise
Potassium	
Prostaglandins	**Medical/Surgical Conditions**
Quinidine	Pregnancy
Tetracycline	Scleroderma
Theophylline	ZES
Tricyclic antidepressants	Gastroparesis
Zidovudine	Nasogastric tube intubation

NSAID, nonsteroidal anti-inflammatory drug; ZES, Zollinger–Ellison syndrome.

Pathophysiology

- TRANSIENT RELAXATIONS OF THE LOWER ESOPHAGEAL SPHINCTER
- ESOPHAGEAL ACID CLEARANCE AND BUFFERING CAPABILITIES
- ANATOMIC ABNORMALITIES
- GASTRIC EMPTYING

Treatment

Table 23-8

Dietary and Lifestyle Modifications Used to Manage Gastroesophageal Reflux Symptoms[8,65,167,178]

Dietary	Medication	Lifestyle
Avoid foods listed in Table 23-7	Avoid medications with a potential to relax the lower esophageal sphincter or that have a direct irritant effect on the esophageal mucosa (Table 23-7)	Stop or decrease smoking/tobacco
Avoid eating large meals	Medications with the potential to irritate the esophagus should be taken with a full glass of water	Avoid alcohol
Avoid eating within 3 hours of bedtime		Lose weight[a]
		Elevate the head of bed 6–8 inches or use a foam wedge[a]
		Sleep in the left lateral decubitus position[a]

[a]Sufficient evidence exists to support lifestyle modification

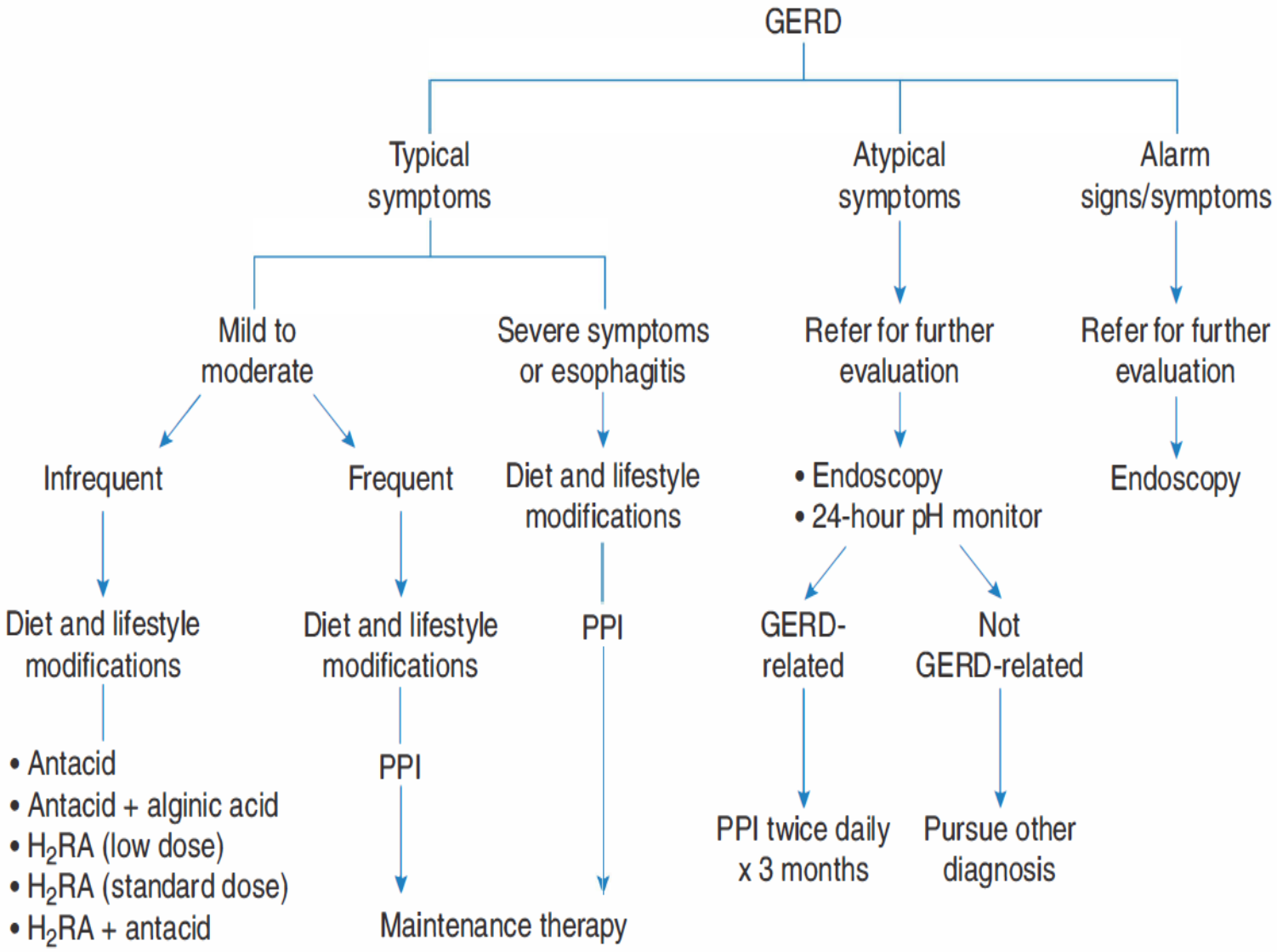


Figure 23-5 Management of GERD. GERD, gastroesophageal reflux disease; H_2RA, H_2 receptor antagonist; PPI, proton-pump inhibitor.

Table 23-10
Atypical Manifestations of Gastroesophageal Reflux Disease[6,205]

Noncardiac Chest Pain	Pulmonary
Ear, Nose, and Throat	Chronic cough
Laryngitis/pharyngitis	Nonallergic, nonseasonal asthma
Hoarseness	Aspiration
Globus sensation	Bronchiectasis/bronchitis
Laryngeal cancer	Sleep apnea
Sinusitis	Idiopathic pulmonary fibrosis
Otitis	Pneumonia
Other	
Hypersalivation	
Dental erosions	

Pharmacotherapy

- Antacids
- Histamine blockers
- PPI
- Combination therapy
- Prokinetic agents
- sucralfate

Stress-Related Mucosal Bleeding

- Acute SRMB is a type of erosive gastritis that occurs in critically ill patients with severe physiologic stress (e.g. , surgery, trauma, organ failure, sepsis, severe burns, and neurologic injuries).

Table 23-11

Risk Factors for Stress-Related Mucosal Bleeding[236–240]

- Respiratory failure
- Coagulopathy
- Hypotension
- Sepsis
- Hepatic failure
- Acute renal failure
- Enteral feeding
- High-dose corticosteroids[a]
- Organ transplant
- Anticoagulants
- Severe burns (>35% of body surface area)
- Head injury
- Intensive care unit stay >7 days
- History of previous GI hemorrhage

Table 23-12

Stress-Related Mucosal Bleeding Prevention: Regimens and Doses

Agent	Dose and Frequency of Administration	FDA Approval[a]
Antacid	30 mL PO/NG every 1–2 hours	No
Cimetidine	300 mg IV loading dose, then 50 mg/hour continuous IV infusion[b]	Yes
Famotidine	20 mg IV every 12 hours or	No
	1.7 mg/hour continuous infusion	No
Ranitidine	50 mg IV every 6–8 hours or	No
	6.25 mg/hour continuous infusion	No
Sucralfate	1 g PO/NG every 6 hours	No
Omeprazole	20–40 mg PO/NG[c] every 12–24 hours	No
Omeprazole/sodium bicarbonate powder for oral suspension	40 mg PO/NG initially, followed by 40 mg 6–8 hours later, then 40 mg PO/NG every 24 hours	Yes
Lansoprazole	30 mg PO/NG[c,d] every 12–24 hours	No
Pantoprazole	40 mg IV/PO/NG[c] every12–24 hours	No
Esomeprazole	40 mg IV/PO/NG every 12–24 hours	No

ZOLLINGER-ELLISON SYNDROME

- ZES is an uncommon gastric acid hypersecretory disease characterized by severe recurrent peptic ulcers that result from a gastrin-producing tumor (gastrinoma).

- Diagnosis

- Treatment

Nausea and vomiting

What you need to know
Age
Infant, child, adult, elderly
Pregnancy
Duration
Associated symptoms
Has vomiting started?
Abdominal pain
Diarrhoea
Constipation
Fever
Alcohol intake
Medication
Prescribed
OTC
Previous history
Dizziness/vertigo

Constipation

- Constipation is a condition that is difficult to define and is often selfdiagnosed by patients. Generally it is characterised by the passage of hard, dry stools less frequently than the person’s normal pattern.

Constipation

Table 3 Drugs that may cause constipation

Drug group	Drug
Analgesics and opiates	*Dihydrocodeine, codeine*
Antacids	*Aluminium salts*
Anticholinergics	*Hyoscine*
Anticonvulsants	*Phenytoin*
Antidepressants	*Tricyclics, SSRIs*
Antihistamines	*Chlorpheniramine, promethazine*
Antihypertensives	*Clonidine, methyldopa*
Anti-Parkinson agents	*Levodopa*
Beta-blockers	*Propranolol*
Diuretics	*Bendrofluazide*
Irono	
Laxative abuse	
Monoamine oxidase inhibitors	
Antipsychotics	*Chlorpromazine*

Constipation

- Treatment:
- Osmotic laxatives (e.g. lactulose, Epsom salts, Glauber’s salts)
- Lubricant laxatives (e.g. liquid paraffin)
- Bulk laxatives (e.g. ispaghula, methylcellulose, sterculia)
- Stimulant laxatives (e.g. senna, bisacodyl)

constipation

When to refer
Change in bowel habit of 2 weeks or longer
Presence of abdominal pain, vomiting, bloating
Blood in stools
Prescribed medication suspected of causing symptoms
Failure of OTC medication

Diarrhoea

What you need to know
Age
Infant, child, adult, elderly
Duration
Severity
Symptoms, associated symptoms
Nausea/vomiting
Fever
Abdominal cramps
Flatulence
Other family members affected?
Previous history
Recent travel abroad?
Causative factors
Medication
Medicines already tried
Other medicines being taken

Diarrhoea

Table 4 Features of some infections causing diarrhoea

Infection	Incubation	Duration	Symptoms
Staphylococcus	2–6 h	6–24 h	Severe, short-lived; especially vomiting
Salmonella	12–24 h	1–7 days	Mainly diarrhoea
Campylobacter	2–7 days	2–7 days	Diarrhoea with abdominal colic
Bacillus cereus	1–5 h	6–24 h	Vomiting
Bacillus cereus (two types of infection)	8–16 h	12–24 h	Diarrhoea

Diarrhoea

When to refer
Diarrhoea of greater than 1 day's duration in children younger than 1 year; 2 days in children under 3 years and elderly patients; 3 days in older children and adults
Association with severe vomiting and fever
Recent travel abroad
Suspected drug-induced reaction to prescribed medicine
History of change in bowel habit
Presence of blood or mucus in the stools

Management

Table 6 Amount of rehydration solution to be offered to patients

Age	Quantity of solution (per watery stool)
Under 1	50 ml (quarter of a glass)
1–5	100 ml (half a glass)
6–12	200 ml (one glass)
Adult	400 ml (two glasses)

Haemorrhoids

Haemorrhoids (commonly known as piles) can produce symptoms of itching, burning, pain, swelling and discomfort in the perianal area and anal canal and rectal bleeding. Haemorrhoids are swollen veins, rather like varicose veins, which protrude into the anal canal (internal piles).

Haemorrhoids

What you need to know
Duration and previous history
Symptoms
Itching, burning
Soreness
Swelling
Pain
Blood in stools
Constipation
Bowel habit
Pregnancy
Other symptoms
Abdominal pain/vomiting
Weight loss
Medication

Haemorrhoids

When to refer
Duration of longer than 3 weeks
Presence of blood in the stools
Change in bowel habit (persisting alteration from normal bowel habit)
Suspected drug-induced constipation
Associated abdominal pain/vomiting

Haemorrhoids

- Management:
- Local anaesthetics (e.g. benzocaine, lidocaine (lignocaine))
- Skin protectors
- Topical steroids
- Antiseptics
- Laxatives


Thank you for your
attention