
PEPTIC ULCER DISEASE

PEPTIC ULCER DISEASE

Burning epigastric pain exacerbated by fasting and improved with meals is a symptom complex associated with peptic ulcer disease (PUD).

Seen as disruption of the mucosal integrity of the stomach and/or duodenum leading to a local defect with active inflammation.

Pathogenesis of peptic ulcer disease

Three-level barrier –

Pre-epithelial

(mucus-bicarbonate-phospholipid layer)

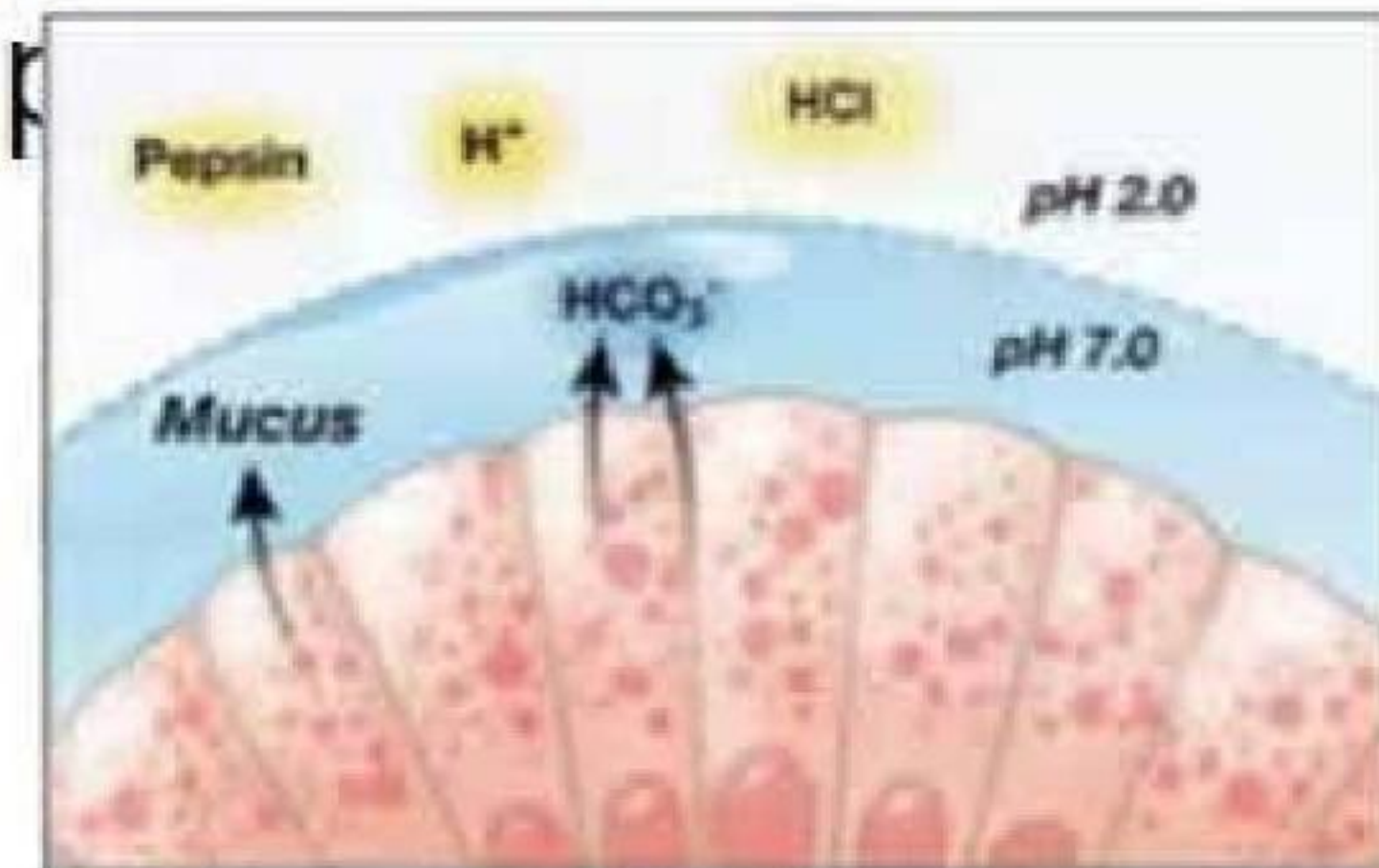
Surface epithelium

Subepithelial elements

(microvascular system within the gastric

MUCUS LAYER

- Forms a gel like layer that impedes diffusion of ions and molecules such as p



- Bicarbonate is secreted by surface epithelial cells of the gastroduodenal mucosa into the mucous gel and neutralizes the acidic pH.

SURFACE EPITHELIUM

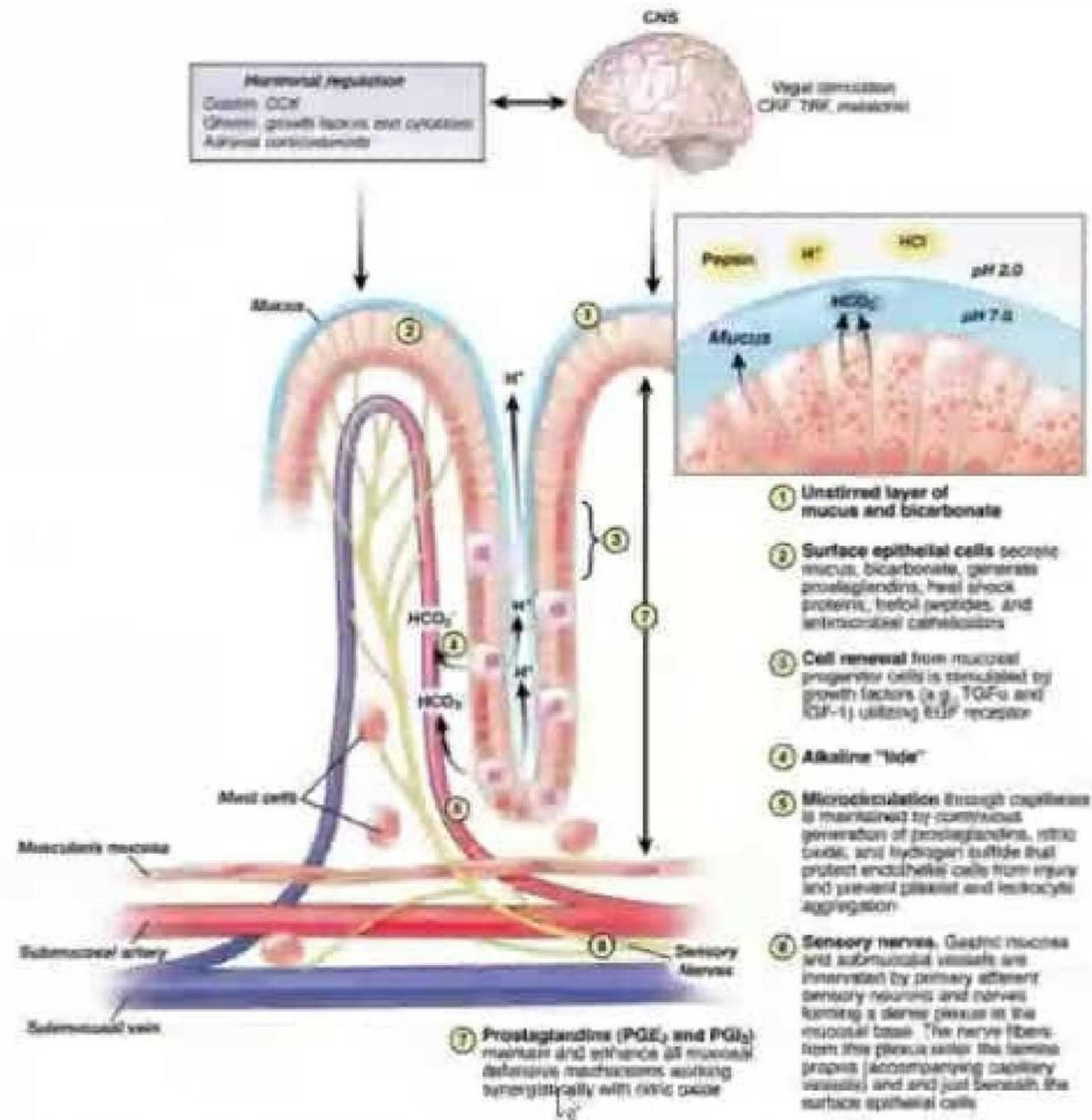
- Involved in **mucus** production
 - Epithelial cell ionic transporters that maintain intracellular **pH** and bicarbonate production
 - Intracellular **tight junctions**
 - Generates **heat shock proteins** that prevent protein denaturation and protect cells from increased temperature, cytotoxic agents, or oxidative stress.
 - Cell **regeneration**:
 - Restitution
 - Regeneration
- } EGFR, TGF-alpha, FGF

SUBEPITHELIAL MICROVASCULATURE

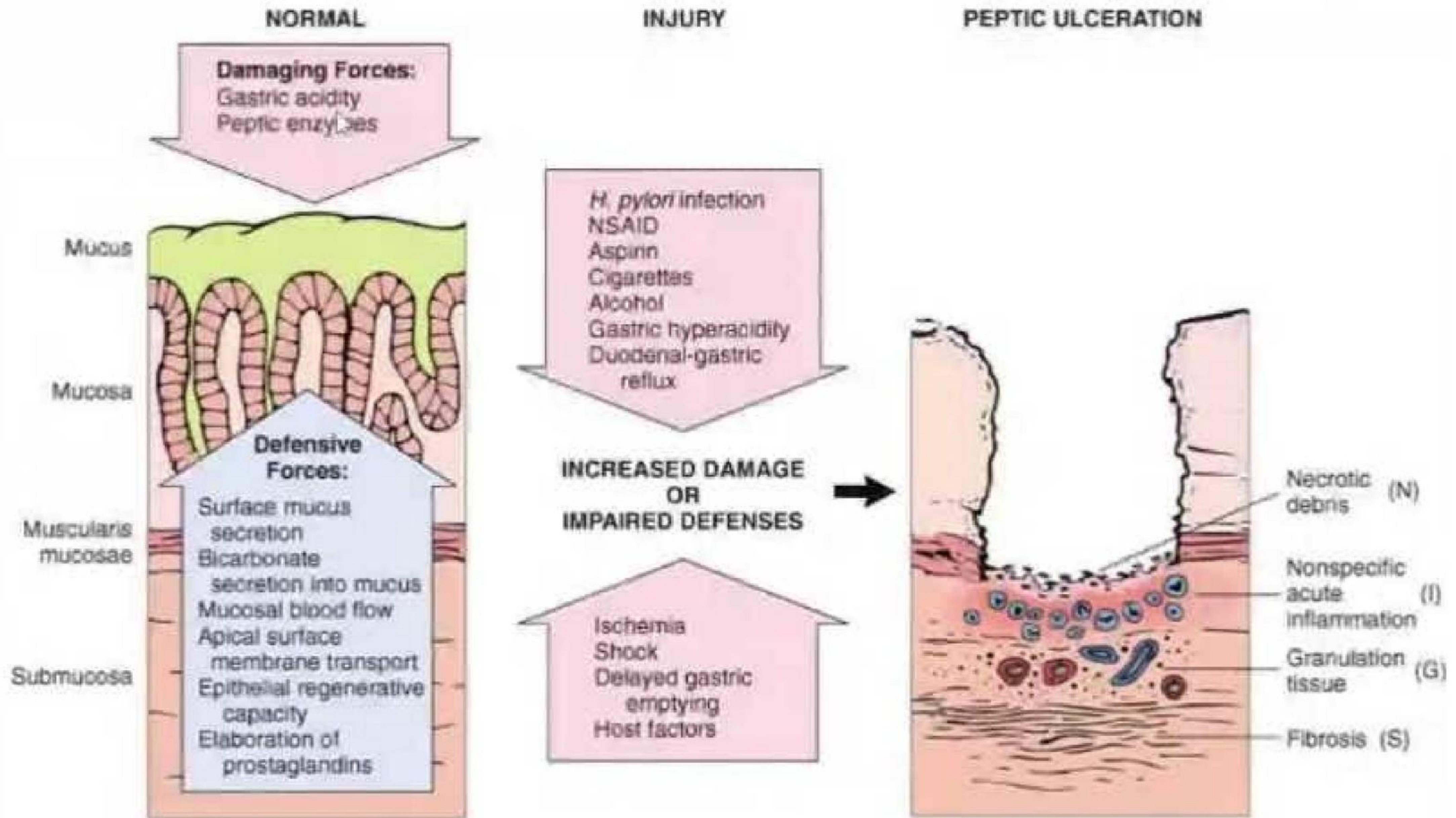
- Provides HCO_3^- which neutralizes the acid generated by the parietal cell.
- Provides an adequate supply of micronutrients and oxygen.
- Removes toxic metabolic by-products.

PROSTAGLANDINS

- Derived from membrane phospholipids by the action of phospholipase enzyme.
- Release of mucosal bicarbonate and mucus
- Inhibit parietal cell secretion
- Maintains mucosal blood flow
- Epithelial cell restitution.



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: Harrison's Principles of Internal Medicine, 18th Edition; www.accessmedicine.com
 Copyright © The McGraw-Hill Companies, Inc. All rights reserved.



CLASSIFICATION:

Acute

Chronic

- gastric ulcer
- duodenal ulcer

ACUTE PEPTIC (STRESS) ULCER

Multiple small mucosal erosions

m/c stomach occasionally involves the duodenum

Etiology:

- ✓ Shock
- ✓ Severe trauma
- ✓ Septicemia
- ✓ Burns – 1st part duodenum ; Curling's ulcer
- ✓ Intracranial lesions- Cushing's ulcer
- ✓ Drugs- aspirin, steroids, indomethacin, etc
- ✓ Local irritants- alcohol, smoking, etc

Hypothesis to pathogenesis:

Ischemic damage to the mucosa

Depletion of mucus barrier

Morphology:

Multiple ulcers

Oval / circular ulcers < 1cm in dia
m/c on the stomach

Gastric rugal folds are essentially normal,
margins and base of the ulcers are not indurated.

Microscopy:

Shallow ulcers limited to the mucosa / submucosa.
Do not invade the muscularis propria.

Mild acute inflammatory reaction noted.

CHRONIC PEPTIC ULCER

Etiology:

- Imbalance of mucosal defence mechanism
- Helicobacter pylori
- NSAIDs : epithelial and endothelial damage
- Local irritants- drugs, alcohol, smoking
pyloric antrum & lesser curvature
- Nutritional deficiencies

CHRONIC PEPTIC ULCER

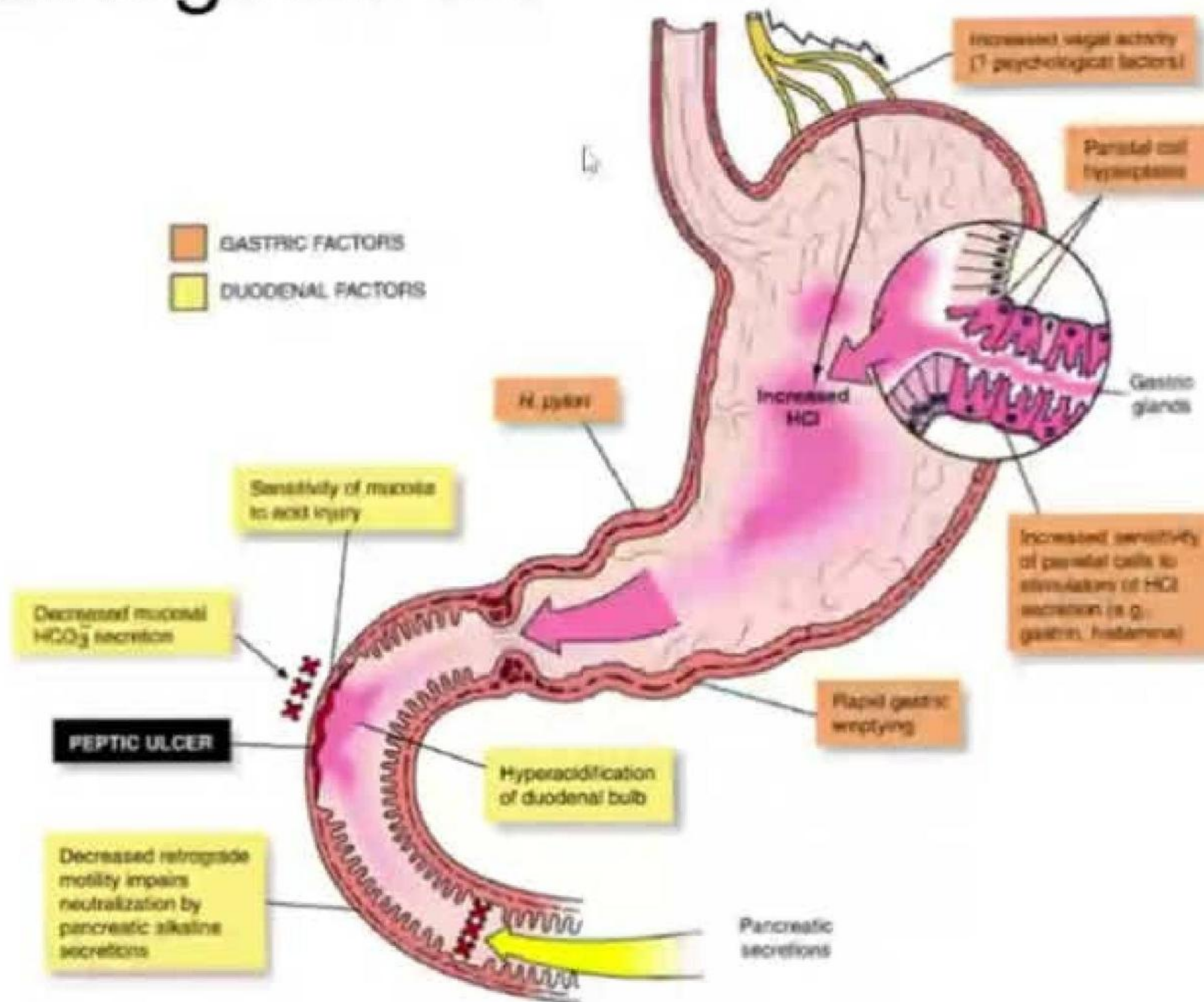
- Psychological factors
- Genetic factors – O blood group; a/w HLA-B5
- Hormonal factors:

eg:

gastrin hypersecretion in ZES syndrome

2 forms: - Gastric ulcer
 - Duodenal ulcer

Pathogenesis:



morphology

Gastric ulcer – lesser curvature
pyloric antrum
post > ant wall of stomach

Duodenal ulcer- 1st part of duodenum
ant > post wall of duodenum

Gross:

1. Solitary ulcers
2. Small oval /round
3. Puched out
4. Flat margins
5. In level of mucosa
6. Mucosal folds converge



Microscopy

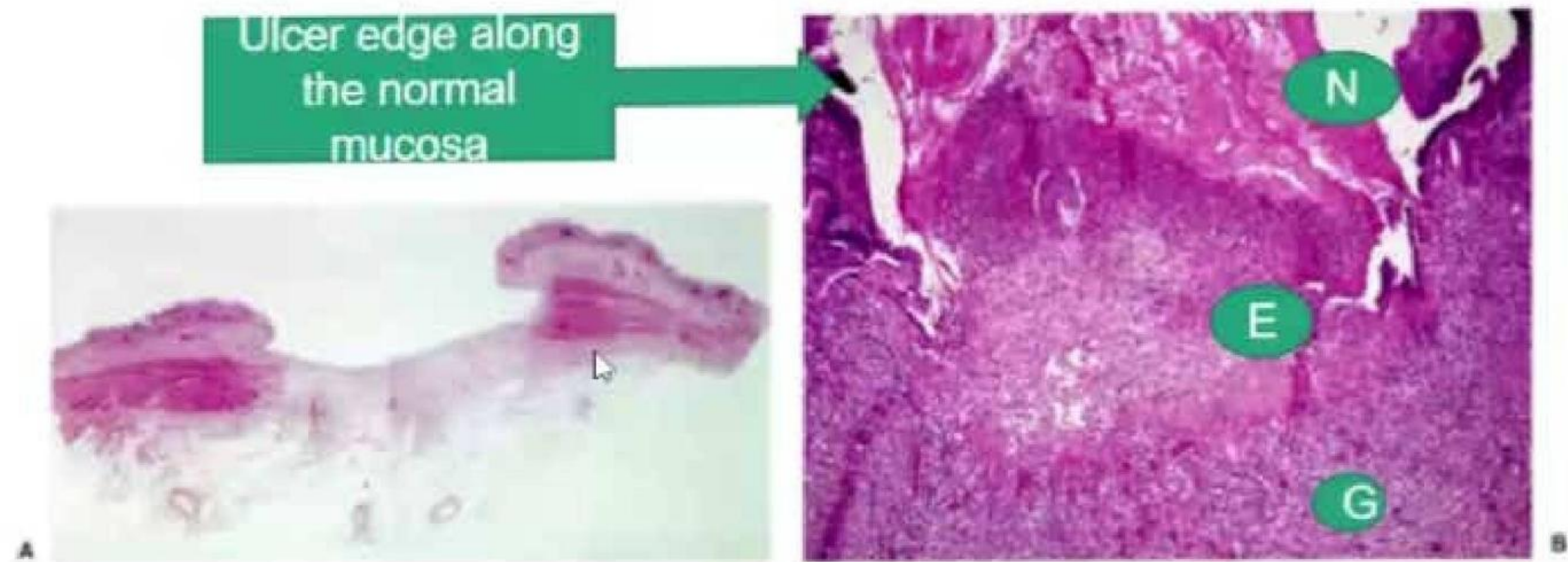
4 histological zones:

Necrotic zone- *floor of the ulcer*
necrotic debris+ leucocytes+ fibrin

Exudative zone
coagulative necrosis

Granulation tissue zone

Zone of cicatrisation-
fibrocollagenous scar tissue
Thrombosed & sclerotic arteries- hemorrhage



Complications

Obstruction

Hour glass deformity- LC

Pyloric stenosis

Duodenal stenosis

Haemorrhage

Perforation

Malignant transformation- in gastric ulcer.

'duodenal ulcers never turns malignant'

GASTRIC ULCER v/s DUODENAL ULCER

<i>Features</i>	<i>Gastric ulcer</i>	<i>Duodenal ulcer</i>
Commonest site	Lesser curvature	First part of duodenum
Incidence	Less common	More common
Age	Beyond 6th decade	25 to 50 years
<i>A/w H. pylori association</i>	<i>Less common</i>	<i>Strong</i>
Acid level	normal/ high	markedly high

GASTRIC ULCER v/s DUODENAL ULCER

<i>Features</i>	<i>Gastric ulcer</i>	<i>Duodenal ulcer</i>
Clinical features		
Pain with antacids	not consistent	Prompt relief
Pain with food	Aggravates	Relieves
Night pain	Not observed	Common
Heart burn	Not common	Common

GASTRIC ULCER v/s DUODENAL ULCER

<i>Features</i>	<i>Gastric ulcer</i>	<i>Duodenal ulcer</i>
	Hematemesis m/c	Melena m/c
Vomiting	Common	No vomiting
Weight loss	Present	Absent
Complication	+/-	No malignant change

ZOLLINGER-ELLISON SYNDROME (ZES)

- Hypergastrinemia caused by gastrin-secreting tumors (gastrinomas)
- Increased acid production
- Severe peptic ulcer disease

Questions

- What are the main causes of peptic ulcer?
- Mention the pathogenesis of PUD related to gastric factors
- Describe the morphology of peptic ulcer
- Mention the common sites of peptic ulcer
- What are the complications of peptic ulcer
- Mention the tests for diagnosis of H-pylori