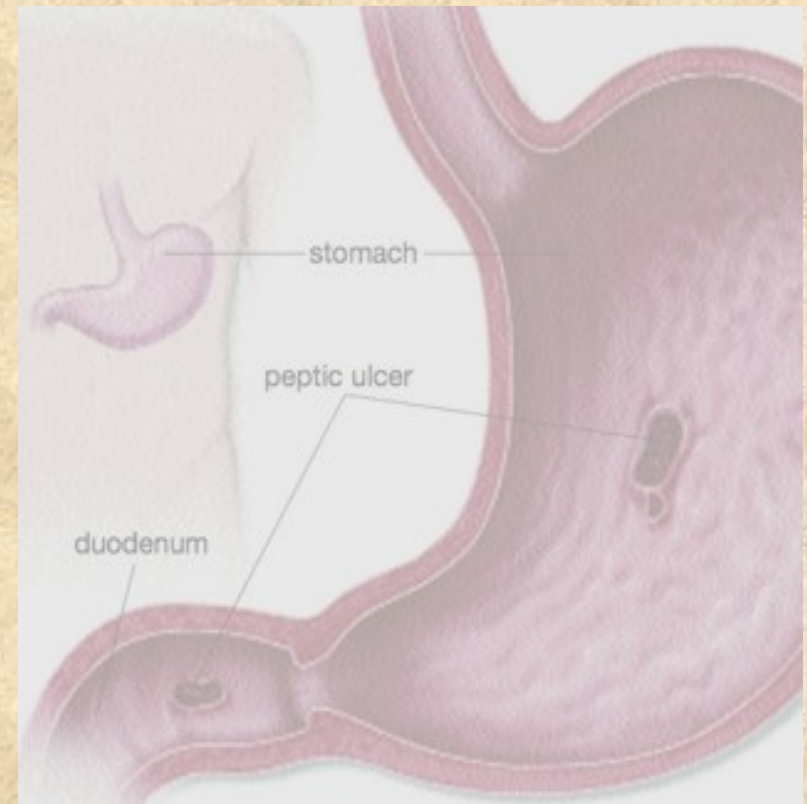


Peptic Ulcer Dis ease



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Introduction

Erosion of GI mucosa resulting from digestive action of HCl and pepsin

Site

- Lower esophagus
- Stomach
- Duodenum
- 10% of men, 4% of women

Types

Acute

- Superficial erosion
- Minimal erosion

Chronic

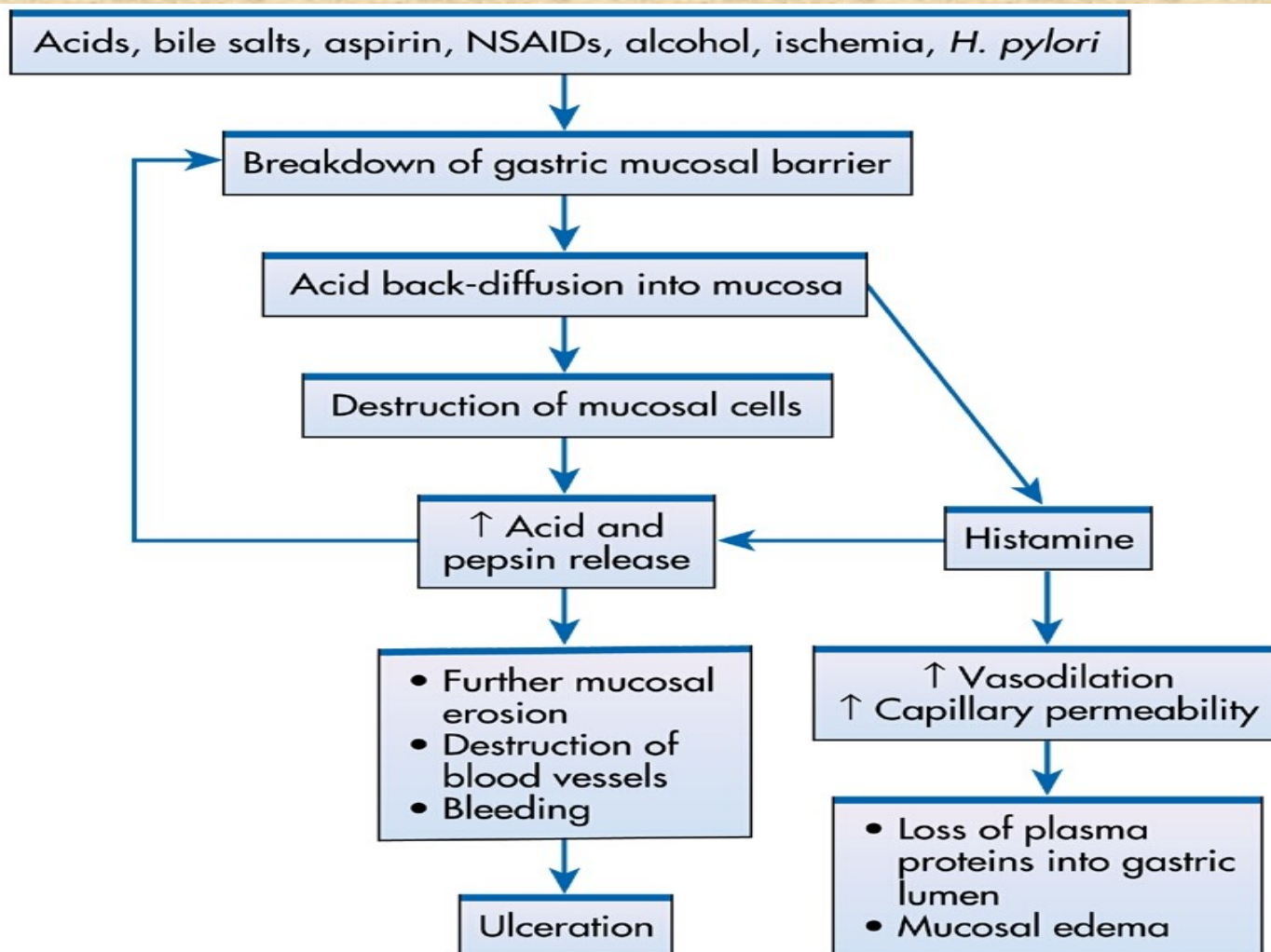
- Muscular wall erosion with formation of fibrous tissue
- Present continuously for many months or intermittently

Etiology and Pathophysiology

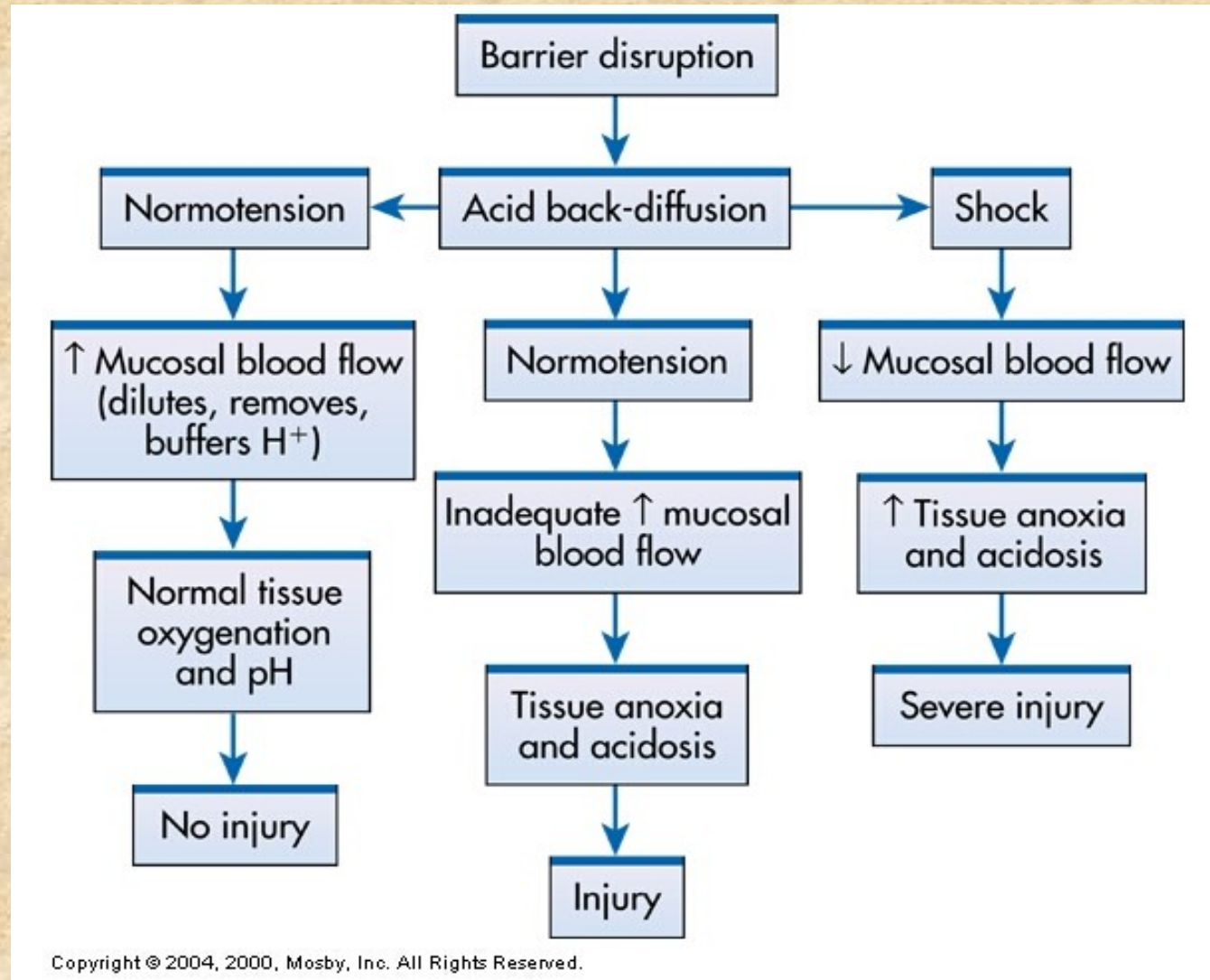
- Develop only in presence of acid environment
- Excess of gastric acid not necessary for ulcer development
- Person with a gastric ulcer has normal to less than normal gastric acidity compared with person with a duodenal ulcer
- Some intraluminal acid does seem to be essential for a gastric ulcer to occur
- Pepsinogen is activated to pepsin in presence of HCl
- Secretion of HCl by parietal cells has a pH of 0.8
- pH reaches 2 to 3 after mixing with stomach contents

- At pH level 3.5 or more, stomach acid is neutralized
- Surface mucosa of stomach is renewed about every 3 days
- Mucosa can continually repair itself except in extreme instances
- Mucosal barrier prevents back diffusion of acid from gastric lumen through mucosal layers to underlying tissue
- Mucosal barrier can be impaired and back diffusion can occur

Diffusion of Acid



Disruption of Gastric Mucosal Barrier



Protective Mechanism

- Mucus forms a layer that entraps or slows diffusion of hydrogen ions across mucosal barrier
- Bicarbonate secreted Neutralizes HCl acid in lumen of GI tract

Gastric Ulcers

Characterized by

- A normal to low secretion of gastric acid
- Back diffusion of acid is greater (chronic)
- Critical pathologic process is amount of acid able to penetrate mucosal barrier
- H pylori is present in 50% to 70%
- Drugs --- Aspirin, corticosteroids, NSAIDs, reserpine, Chronic alcohol abuse, chronic gastritis

Duodenal Ulcers

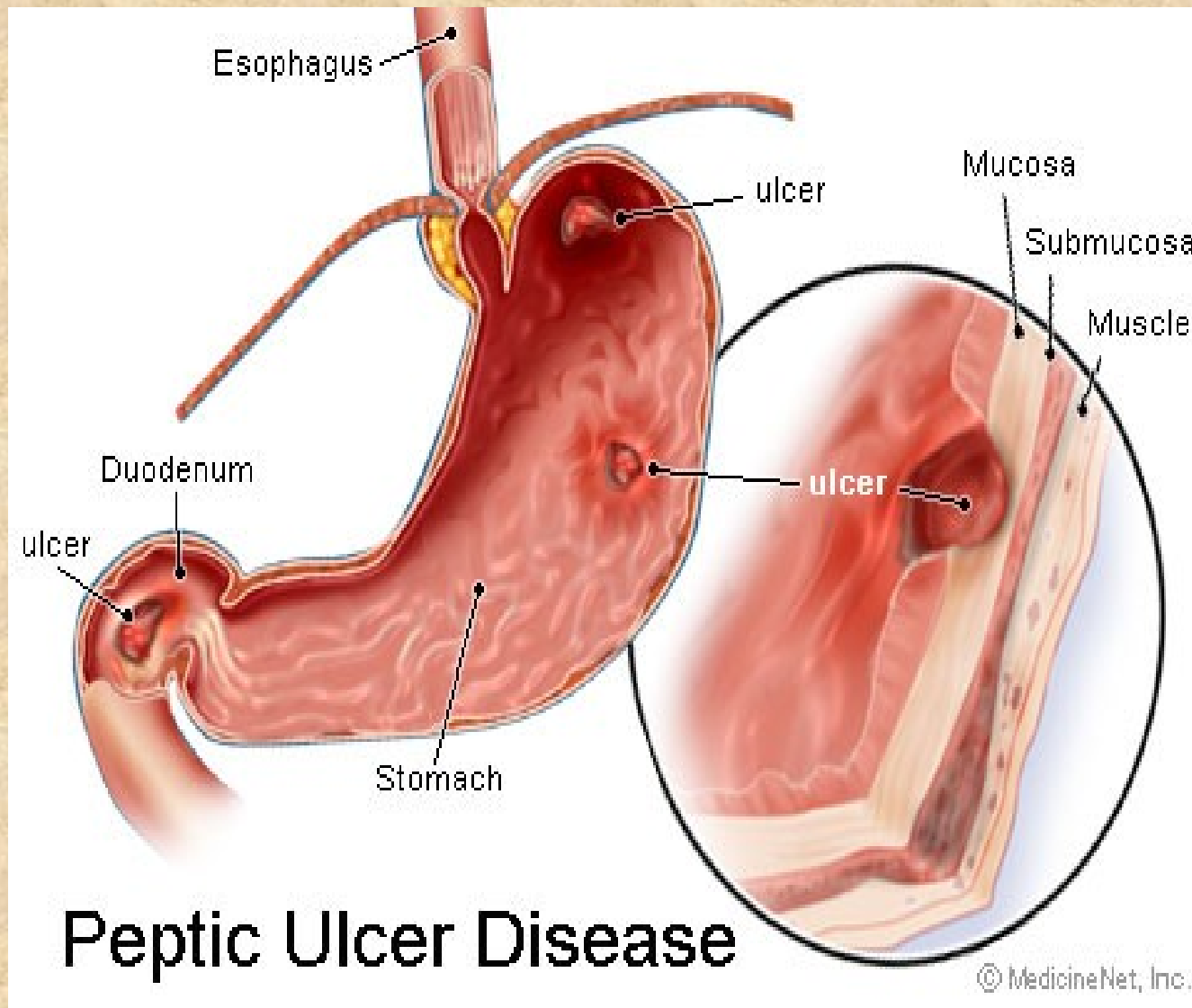
- Between ages of 35 to 45 years
- Account for 80% of all peptic ulcers
- Associated with ↑HCl acid secretion
- H.pylori associated in 90-95% of cases
- Diseases with ↑risk of duodenal ulcers
 - COPD, cirrhosis of liver, chronic pancreatitis, hyperparathyroidism, chronic renal failure

Clinical Features

- Common to have no pain or other symptoms
 - Gastric and duodenal mucosa not rich in sensory pain fibers
 - Duodenal ulcer pain
 - Burning, cramplike
 - Gastric ulcer pain
 - Burning, gaseous

Complications

- 3 major complications
 - ❖ Hemorrhage
 - ❖ Perforation
 - ❖ Gastric outlet obstruction
- Initially treated conservatively
- May require surgery at any time during course of the rapy



Diagnostic Studies

- **Endoscopy procedure**
 - Determines degree of ulcer healing after treatment
 - Tissue specimens can be obtained to identify *H. pylori* and to rule out gastric cancer
- **Tests for *H. pylori***
 - Noninvasive tests
 - Serum or whole blood antibody tests
 - Immunoglobulin G (IgG)
 - Urea breath test
 - C 14 breath test
 - Invasive tests
 - Biopsy of stomach
 - Rapid urease test

- **Barium contrast studies**
 - Widely used
- **X- ray studies**
 - Ineffective in differentiating a peptic ulcer from a malignant tumor
- **Gastric analysis**
- **Lab analysis**

Treatment

Medical regimen consists of

- Adequate rest
- Dietary modification
- Drug therapy
- Elimination of smoking
- Long-term follow-up care

Aim of treatment program

- ↓ degree of gastric acidity
- Enhance mucosal defense mechanisms
- Minimize harmful effects on mucosa

Drug Therapy

- Antacids
- H₂ receptor blockers
- PPIs
- Antibiotics
- Anticholinergics
- Cytoprotective therapy

Histamine receptor blocks (H₂R blockers)

- Used to manage peptic ulcer disease
- Block action of histamine on H₂ receptors
 - ↓ HCl acid secretion
 - ↓ conversion of pepsinogen to pepsin
 - ↑ ulcer healing

Proton pump inhibitors

- Block ATPase enzyme that is important for secretion of HCl acid

Antibiotic therapy

- Eradicate H. pylori infection
- No single agents have been effective in eliminating H. pylori

- **Antacids**
 - Used as adjunct therapy for peptic ulcer disease
 - ↑ gastric pH by neutralizing acid
- **Anticholinergic drugs**
 - Occasionally ordered for treatment
 - ↓ cholinergic stimulation of HCl acid
- **Cytoprotective drug therapy**
- **Serotonin reuptake inhibitors**

Nutritional therapy

- Dietary modifications may be necessary so that foods and beverages irritating to patient can be avoided or eliminated
- Nonirritating or bland diet consisting of 6 small meals a day during symptomatic phase
- Protein considered best neutralizing food
 - Stimulates gastric secretions
- Carbohydrates and fats are least stimulating to HCl acid secretion
 - Do not neutralize well

Surgical Treatment

- < 20% of patients with ulcers need surgical intervention
- Indications for surgical interventions
 - ❖ Intractability
 - ❖ History of hemorrhage, ↑ risk of bleeding
 - ❖ Prepyloric or pyloric ulcers
 - ❖ Multiple ulcer sites
 - ❖ Drug-induced ulcers
 - ❖ Possible existence of a malignant ulcer
 - ❖ Obstruction

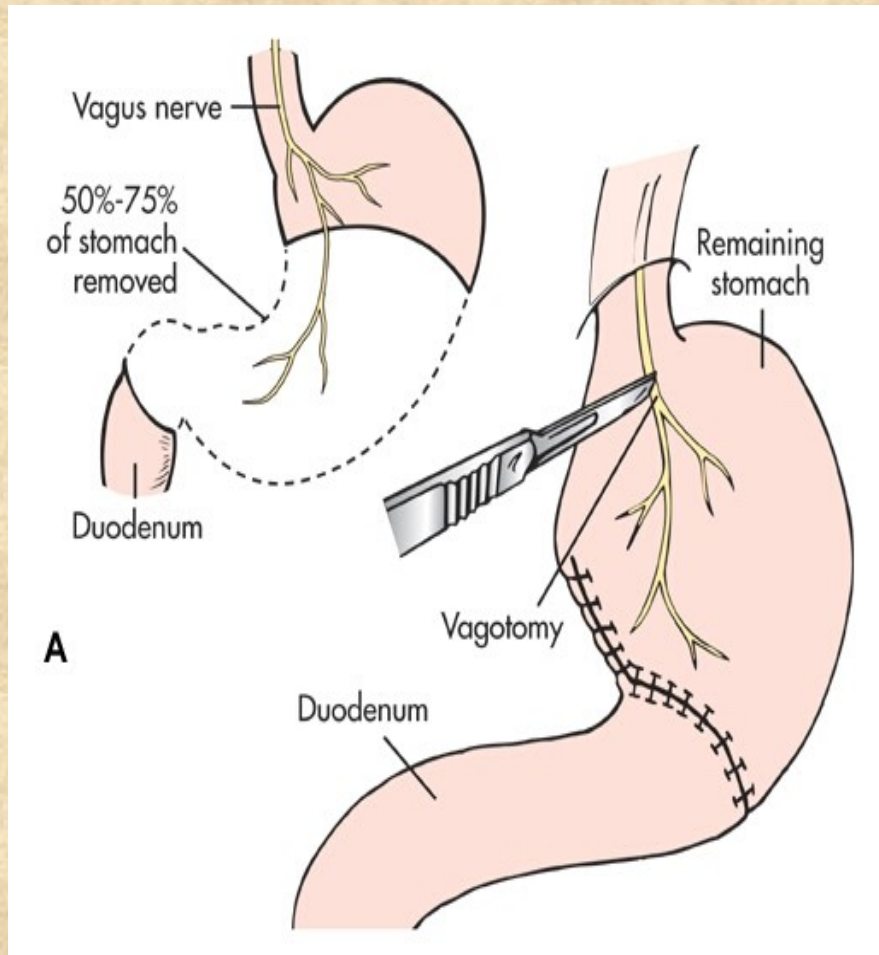
Surgical procedures

- ❖ Gastroduodenostomy

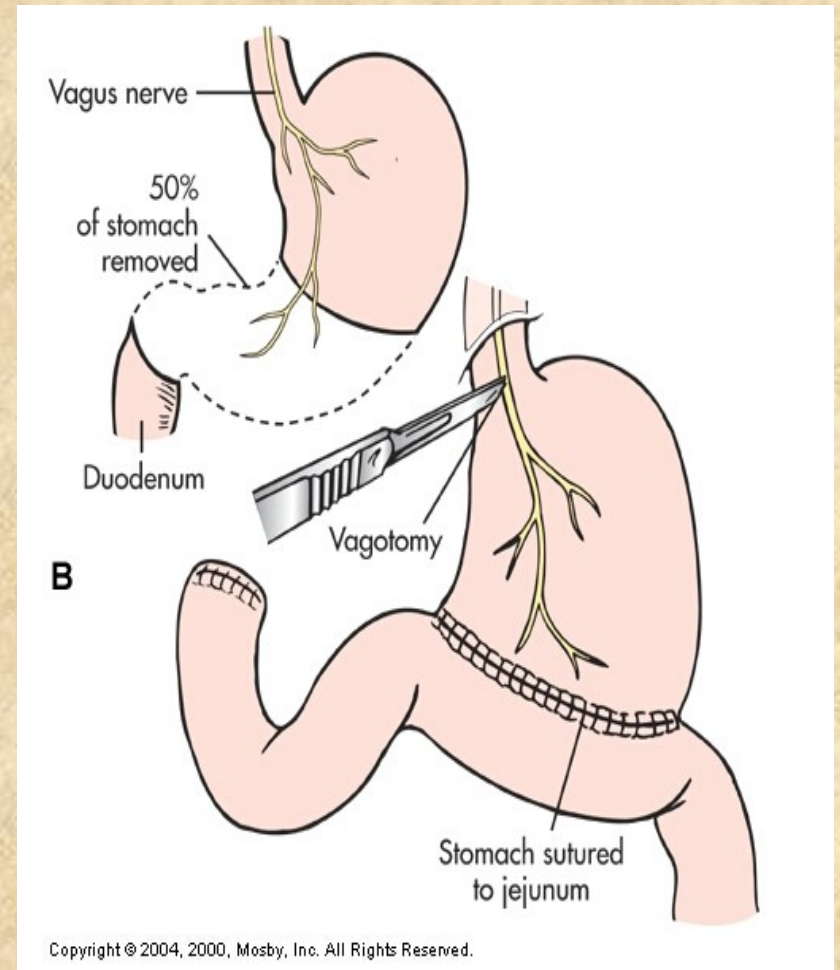
- ❖ Gastrojejunostomy

- ❖ Vagotomy

- ❖ Pyloroplasty



A. Billroth I Procedure



B. Billroth II Procedure

Goals

- ❖ Comply with prescribed therapeutic regimen
- ❖ Experience a reduction or absence of discomfort related to peptic ulcer disease
- ❖ Exhibits no signs of GI complications
- ❖ Have complete healing
- ❖ Lifestyle changes to prevent recurrence