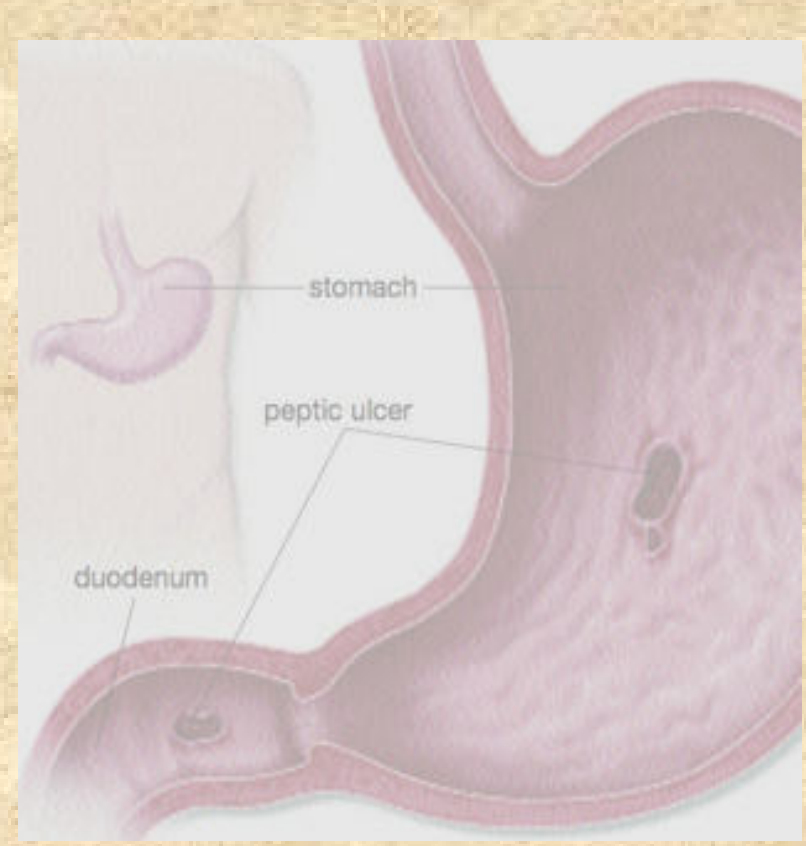
Peptic Ulcer Disease



Dept. of Surgery

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

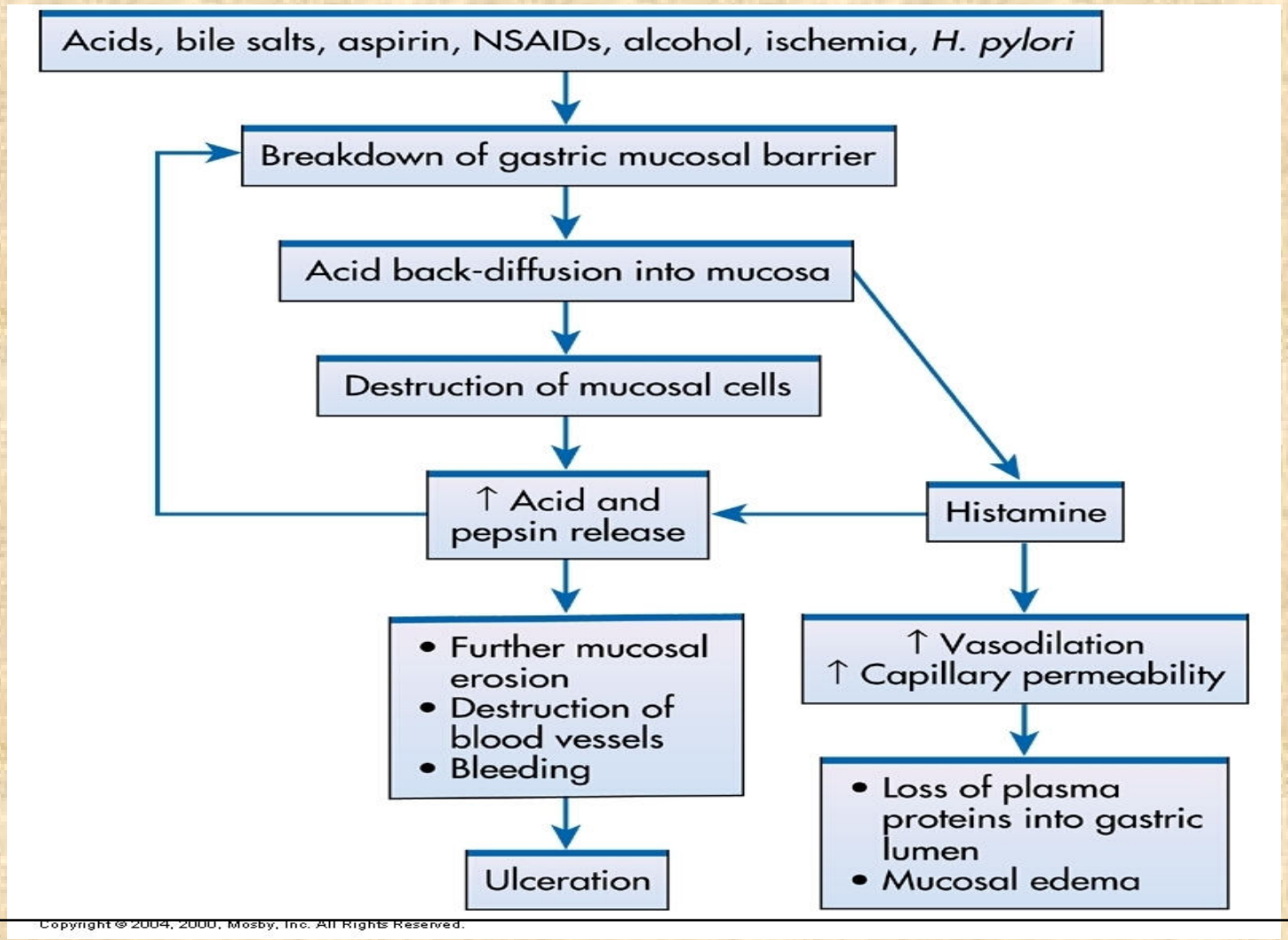
- Muscular wall erosion with formation of fibrous tissue
- Present continuously for many months or intermittently

Etiology and Pathophysiology

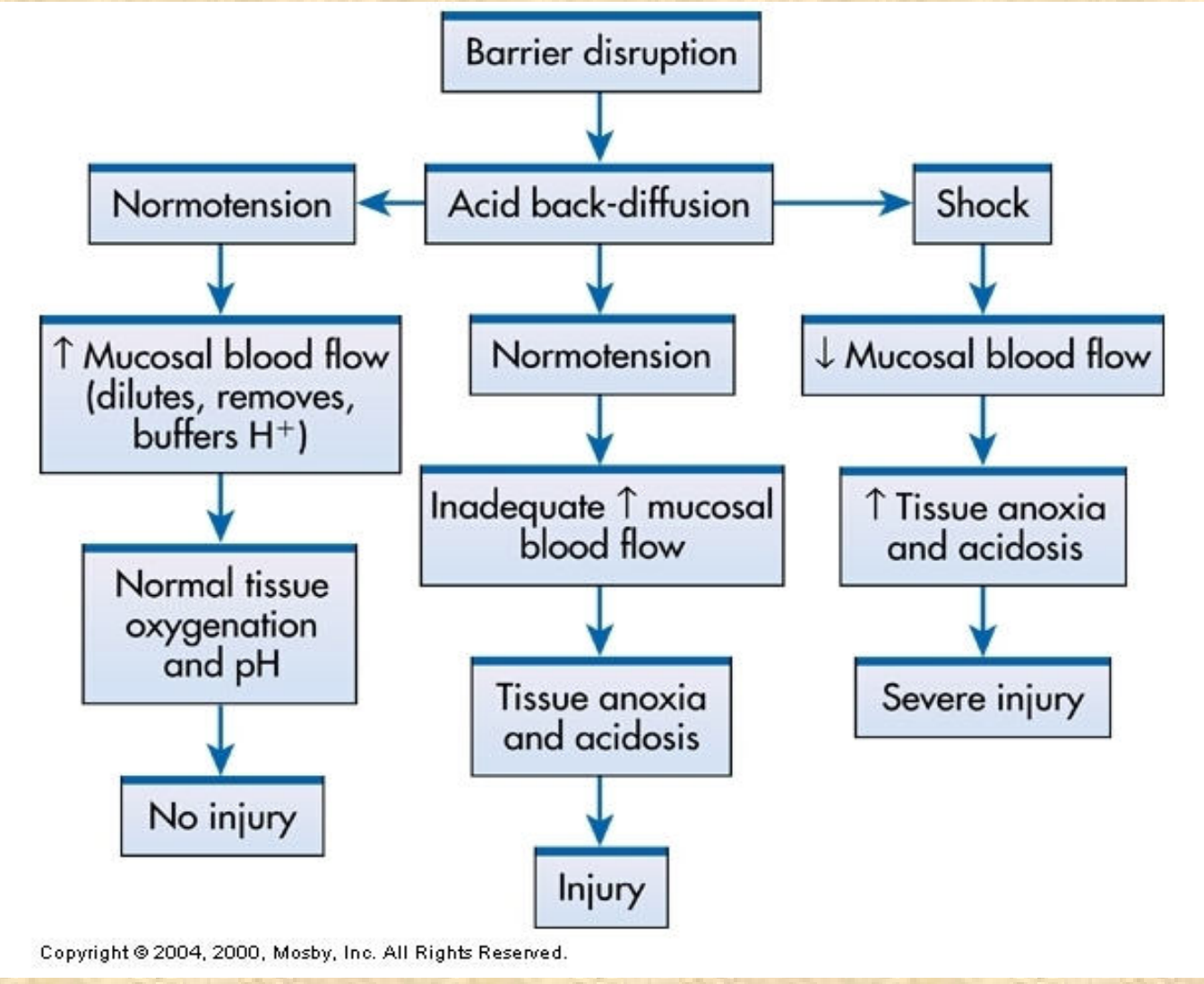
- Develop only in presence of acid environment
- Excess of gastric acid not necessary for ulcer development
- Person with a gastric ulcer has normal to less than normal gastric acidity compared with person with a duodenal ulcer
- Some intraluminal acid does seem to be essential for a gastric ulcer to 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, N SAIDs, reserpine, Chronic alcohol abuse, chronic gastritis

Duodenal Ulcers

- Between ages of 35 to 45 years
- Account for 8 0% of all peptic ulcers
- Associated with ↑HCl acid secretion
- H.pylori associated in 9 0- 9 5 % of cases
- Diseases with ↑risk of duodenal ulcers

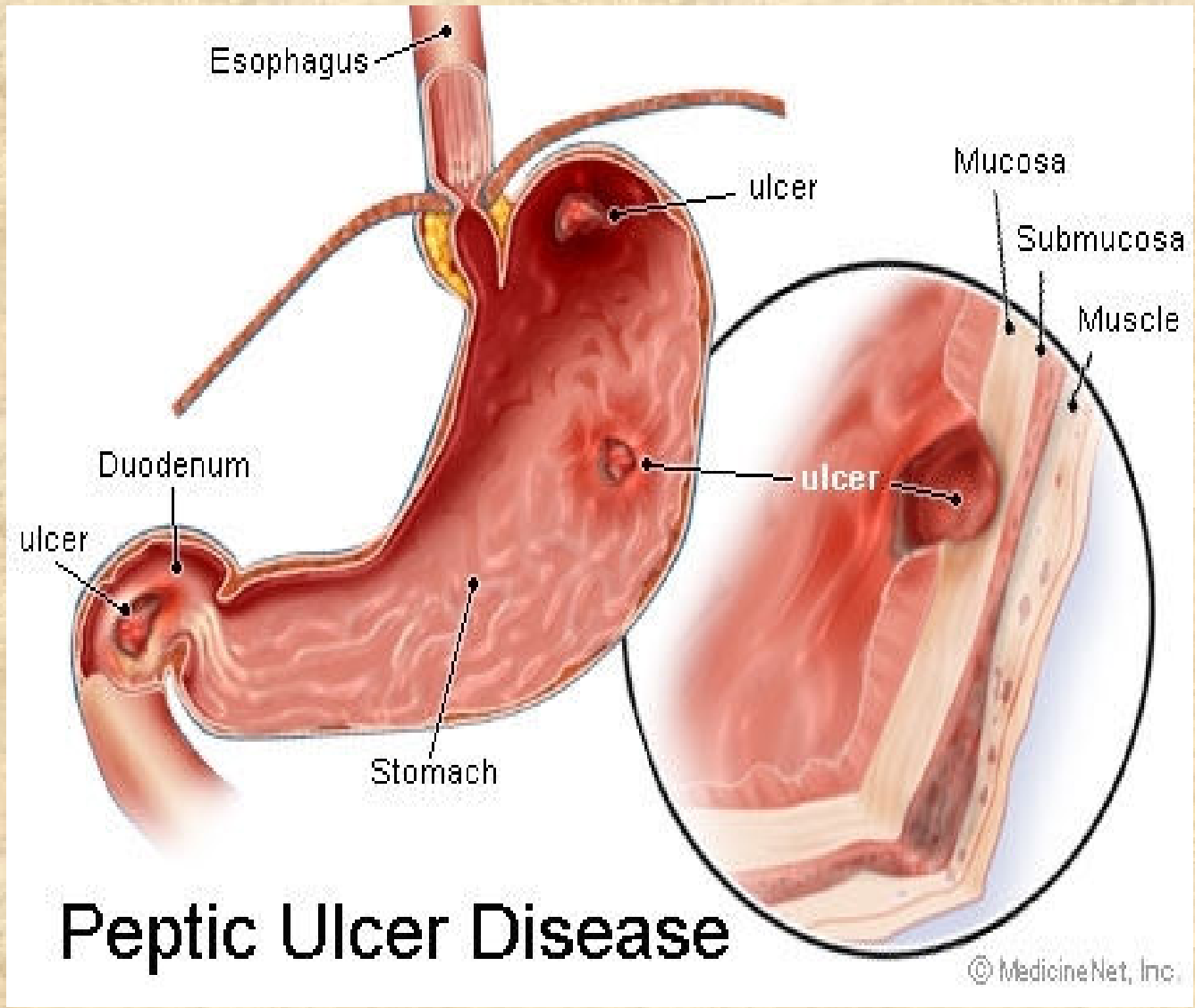
  COPD, cirrhosis of liver, chronic pancreatitis, hyperparathyroidism, chronic renal failure

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 therapy



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
      - Immunoglobin G (I g G)
    - 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 pro g ram

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

Drug Therapy

- Antacids
- H_2 receptor blockers
- PPIs
- Antibiotics
- Anticholinergics
- Cytoproctective therapy

Histamine receptor blocks (H_2 R blockers)

- Used to manage peptic ulcer disease
- Block action of histamine on H_2 receptors
  - **↓ HCl acid secretion**
  - **↓ conversion of pepsinogen to pepsin**
  - **↑ ulcer healing**

Proton pump inhibitors

- Block ATPase en zyme 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

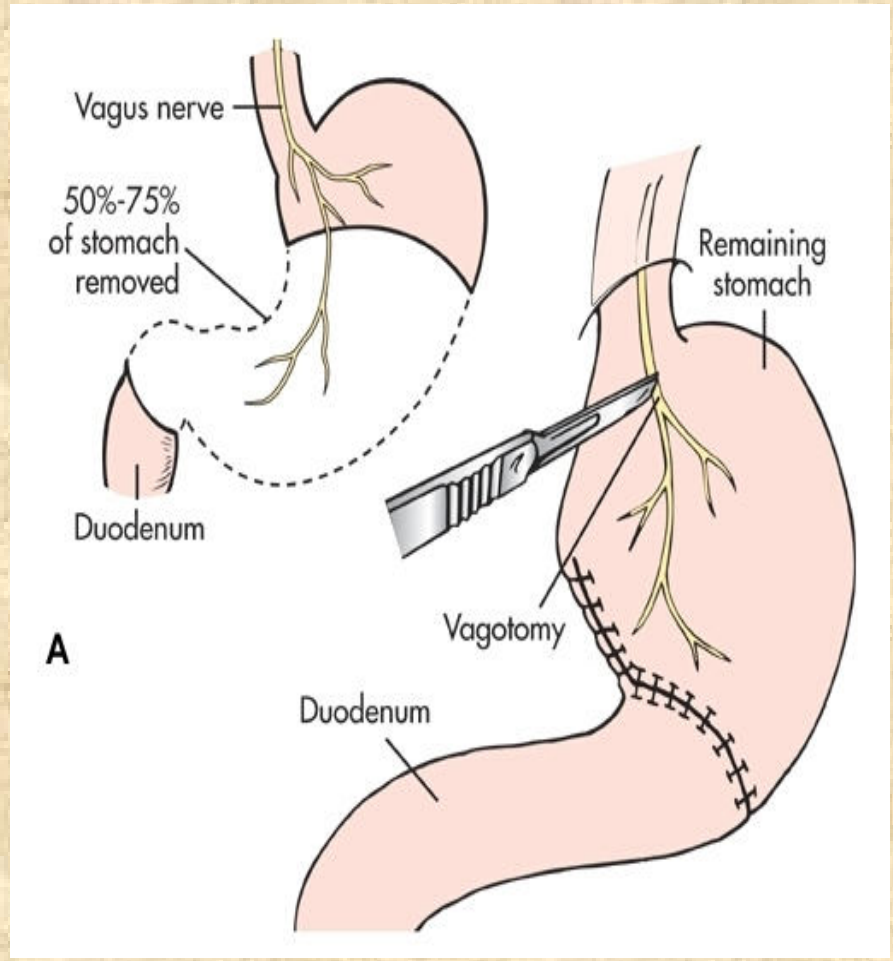
- Dietary modifications may be necessary so that foods and beverages irritating to patient can be avoided or eliminated
- Nonirritating or bland diet consisting of 6 small meals a day during symptomatic phase
- Protein considered best neutralizing food
  - Stimulates gastric secretions
- Carbohydrates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

Surgical Treatment

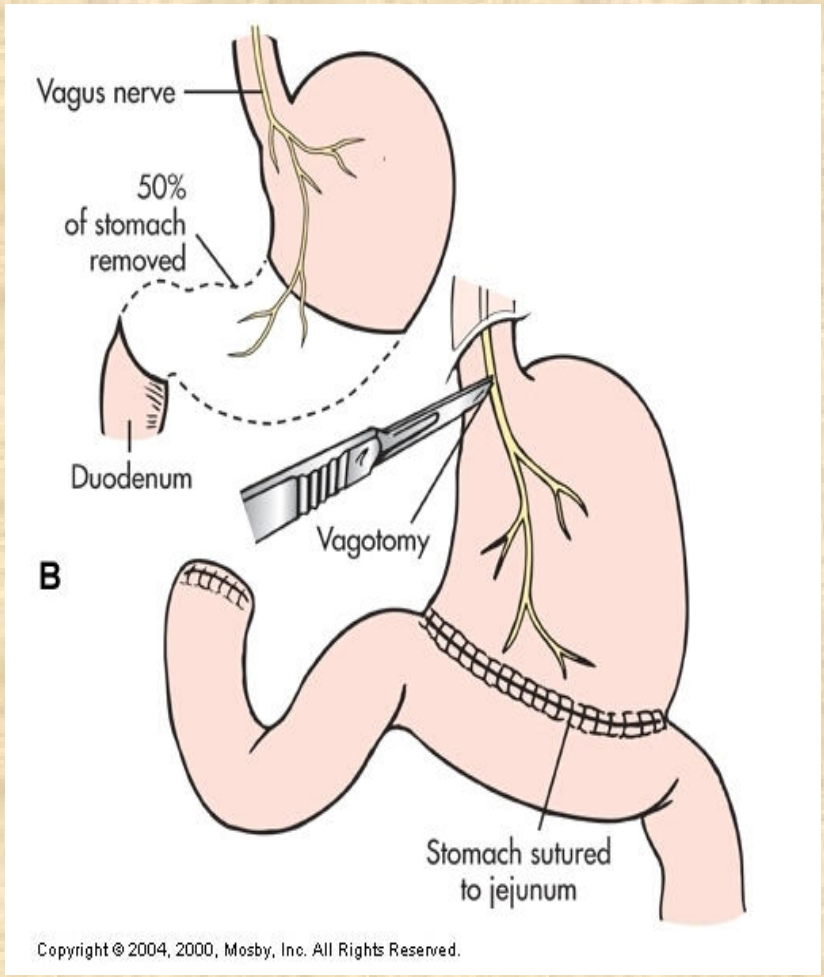
- < 20% of patients with ulcers need surgical intervention
- Indications for surgical interventions
  - ❖ Intractability
  - ❖ History of hemorrhage, ↑ risk of bleeding
  - ❖ Prepyloric or pyloric ulcers
  - ❖ Multiple ulcer sites
  - ❖ Drug-induced ulcers
  - ❖ Possible existence of a malignant ulcer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