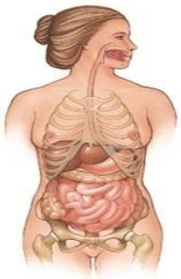
GIT infections

Examination of clinical specimens

Outlines

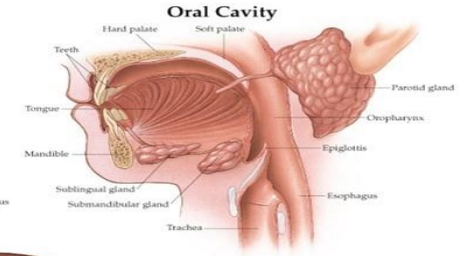
- Introduction
- Anatomy
- Resident Microflora
- Pathogenesis
- Clinical presentation
- Etiology
- Epidemiology
- Laboratory Diagnosis

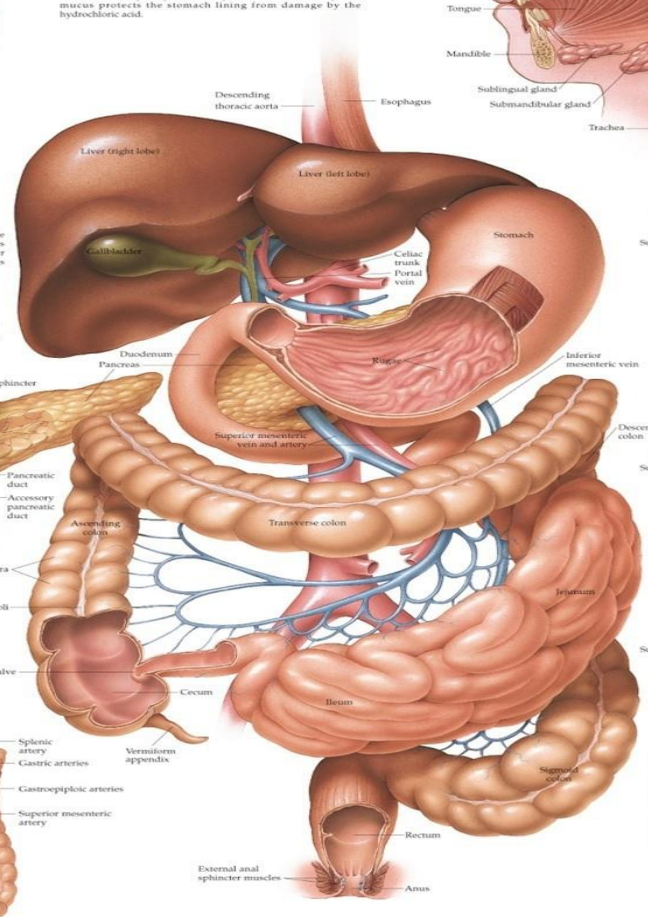
THE DIGESTIVE SYSTEM



The Oral Cavity, Salivary Glands and Stomach

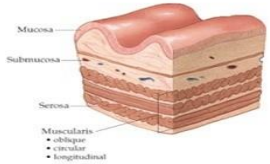
Digestion begins in the mouth as food is mixed with saliva. Saliva breaks down the starch in food into smaller sugars. After moving to the stomach through the esophagus, food is further broken down by enzymes and hydrochloric acid. A layer of mucus protects the stomach lining from damage by the hydrochloric acid.

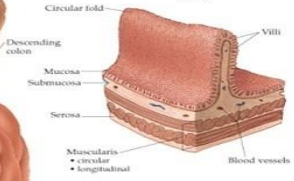


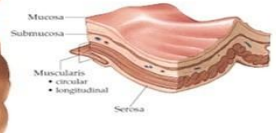


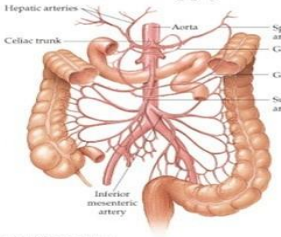
The Liver, Pancreas and Duodenum

Partially digested food, or chyme, passes from the stomach to the duodenum. Here bile and enzymes from the pancreas enter the duodenum and further break down fat, protein and carbohydrates. Bile is produced by the liver and stored in the gallbladder.









The Small and Large Intestines

Chyme moves to the last parts of the small intestine, the jejunum and ileum, where nutrients are absorbed into the bloodstream. The nutrients travel to the liver, via the hepatic portal venous system, for further metabolism and storage. Undigested material enters the colon, where water and electrolytes are absorbed. The remaining waste is stored until eliminated.

INTESTINAL MICROFLORA

10^{14} micro-organisms, >500 differentes species

Stomach 10^2 to 10^3

Lactobacilli

Duodenum

$<10^{4-5}$

Streptococci
Lactobacilli

Jejunum

Enterobacteria
Enterococcus
Faecalis
Bacteroides
Bifidobacteria
Peptococcus
Peptostreptococc
Ruminococcus
Clostridia
Lactobacilli

Ileum 10^3 to 10^7

Colon with appendix 10^9 to 10^{12}

and….

Pathogenesis

- **Host factors**
  - Stomach acidity
  - Normal peristalsis
  - The mucous layer
  - The normal flora
  - Peyer's patches
  - Personal hygiene
  - Age
  - Medications for ulcers
  - Antimicrobial or antimetabolite treatments
    - PMC

Pathogenesis

- **Microbial factors** Table 75-1
  - Enterotoxin or bacterial adherance/invasion
  - Cytotoxin or bacterial invasion
  - Penetrations the mucosa and invading the reticuloendothelial system

Clinical manifestations

TABLE 1 Types of bacterial gastroenteritis[a]

Parameter	Secretory gastroenteritis	Inflammatory gastroenteritis	Invasive gastroenteritis
Location	Proximal small intestine	Colon	Distal small intestine
Type of illness	Watery diarrhea	Dysentery	Enteric fever
Stool examination	No fecal leukocytes	Fecal polymorphonuclear leukocytes	Fecal mononuclear leukocytes (if patient has diarrhea)
Mechanism	Enterotoxin or bacterial adherence/invasion causes a shift in water and electrolyte excretion/adsorption	Bacterial invasion or cytotoxins cause mucosal damage that leads to inflammation	Bacteria penetrate the mucosa and invade the reticuloendothelial system
Classic pathogens	*Vibrio cholerae*, ETEC, *Clostridium perfringens*, *Bacillus cereus*, *Staphylococcus aureus*	*Shigella*, STEC, *Salmonella* (not *Salmonella* Typhi/Paratyphi), *Vibrio parahaemolyticus*, *Clostridium difficile*, *Campylobacter*	*Salmonella* Typhi/Paratyphi, *Yersinia enterocolitica*

[a] Adapted from reference 321 with permission of the publisher.

Etiologic agents

- Table 75-4

Epidemiology

- Institutional settings
- traveler's diarrhea
- Food- and water-born outbreaks
- Immunocompromised hosts

Other infections of GIT

- **Esophagitis**
- **Gastritis**
  - *H. pylori*
- **Proctitis**
  - *C. trachomatis*
  - *N. gonorrhoeae*
  - *Herpes simplex*
- **Miscellaneous**

Laboratory Diagnosis

- **Specimen collection and transport:**
  - Stool specimens for bacterial culture
  - Miscellaneous specimen types

Laboratory Diagnosis

A. Direct detection in stool

- Wet mounts
- Stains
- Antigen detection
- Molecular biological techniques

Laboratory Diagnosis

B. Culture of fecal material

- **Bacteria**
  - Routine culture
- ***C. difficile*-associated diarrhea**

C. Serology