

Dysphagia: difficulty swallowing

Dysphagia Type 1	Problem with delivering food or fluid to the esophagus	decreased ability to initiate the swallowing sequence*	person may cough to dislodge the solid/liquid or may aspirate when attempting to swallow	symptoms tend to worsen with liquid rather than solid
Dysphagia Type 2	Problem with transporting the bolus (food piece) down the throat	result of a disorder (structural or neuromuscular) in which the peristaltic movement of the throat is altered	persons may feel as though the food is stuck behind the sternum and if underlying pathology is not resolved can worsen to include liquids	disorders include esophageal diverticula: outpouchings of one or more layers of the esophagus achalasia: in which the smooth muscle is altered neoplasms or strictures
Dysphagia Type 3	Problem with bolus entering the stomach	Secondary to lower esophageal dysfunction or lesions that obstruct the pathway	categorized as a tightness or pain in the substernal area when swallowing	tumors in the mediastinum or below may reduce LES function and chronic inflammation of the lower esophagus can lead to further inability

Esophageal Pain

Heartburn/Pyrosis	caused by a reflux of the gastric fluids	a substernal burning sensation that radiates up to the neck	the acid irritates the esophageal mucosa and can also cause spasm of the throat muscles
Chest pain	esophageal distention (enlarging) or powerful muscle contractions	pain radiates to neck, jaw, shoulder and arm (similar to angina pectoris) and odynophagia	esophageal obstruction or diffuse esophageal spasm where there is a high magnitude spasm between normal peristalsis



Esophageal Pain (cont)

infection chest pain	infections in the esophagus in immunocompromised individuals	dull, aching chest pain	can worsen heartburn and regular chest pain
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Abdominal pain: first sign of GI tract disorder

visceral pain	due to stretching or inflammation	diffuse, poorly localized pain which can be knawing, burning or cramping
somatic pain	injury to abdominal pain, parietal peritoneum, mesentery or diaphragm	sharp intense pain at the area of injury, well localized
referred pain	pain in the same neurosegment felt in other part	sharp well localized pain that may be felt deep and at a location away from the injury
acute	instantaneous onset	
chronic	gradual onset	

usually accompanied by other signs/symptoms such as vomiting/emesis or bowel alteration

Diarrhea

acute diarrhea	due to acute infection, emotional stress, leakage of liquid stool around impacted stool
chronic	symptoms last longer than 4 weeks, due to chronic GI tract infection, alterations in motility or integrity, malabsorption, endocrine disorders
episodic diarrhea	probably related to food allergy or ingestion of irritants such as caffeine
osmotic	increased amount of poorly soluble nutrient intake such as carbs which pull water into the bowel lumen
secretory	caused by toxins that stimulate intestinal fluid secretion and lower absorption, ~ 1L of diarrhea
exudative (mucus, blood or protein)	blood, protein and mucus getting into the bowel lumen from the site of inflammation



By [jaydevine_23](https://cheatography.com/jaydevine-23/)
cheatography.com/jaydevine-23/

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Diarrhea (cont)

motility disturbances decreased time and contact of chyme with the absorptive enzymes

increase in fluidity and frequency of the stool, may be very liquidy

Disorders of the mouth

Stomatitis ulcerative inflammation of the mouth oral mucosa

causes: viral or bacterial infections, mechanical trauma, irritant exposure (alcohol/tobacco), medication, radiation therapy, autoimmune disorders and nutrient deficiency

most common type: HSV acquired most by children which causes tingling and itching and leave behind painful ulcers that have ruptures and HSV stays dormant in the dorsal ganglia reactivating years later

treatment: antiviral/biotics for infections, topical or systemic steroids for autoimmune causes, general treatment in oral hygiene and topical barriers or steroids

Hiatal hernia

the stomach pushes up into the chest cavity through the diaphragm -

risk increases with age, women more than men -

2 types: sliding hernia (most common), paraesophageal hernia or mixed sliding hernias when a portion of the stomach and gastroesophageal junction move up the diaphragm

- paraesophageal hernia is when the greater curvature of the stomach pushes up

risk factor anything that increase intraabdominal pressure such as pregnancy, obesity and chronic straining or coughing



Hiatal hernia (cont)

treatment similar to GERD with surgery for acute manifestations

life threatening in a large proportion of the stomach becomes incarcerated which is rare

GERD

pathogenesis any agent that alters the strength of the LES or increases intraabdominal pressure

risk factor ingesting fatty foods, smoking, alcohol, pregnancy, caffeine, anatomical features such as a hiatal hernia

clinical manifests: attributed to esophagitis (inflammation) heartburn, regurgitation, chest pain, dysphagia (difficulty swallowing)

treatment increase LES strength, esophageal clearance, improve gastric emptying, suppress acidity, avoid dietary risk factors

treatment (cont.) antacids, histamine blockers (sporadic GERDS), proton pump inhibitors have been known to reverse changes from chronic GERD, surgical intervention

Barrett esophagus complication of GERDS where the epithelium of the esophagus changes to another

leads to a higher risk of cancer progression can lead to ulceration, fibrotic scarring, esophageal strictures

LES: lower esophageal sphincter

GERDS: backflow of gastric contents into the esophagus

peptic ulcer disease

etiology disorder of the upper GI tract caused by acids and pepsin which causes injury to the mucosa of the throat, stomach or duodenum

- an increase in the factors that cause ulcers than those that do not

cause NSAIDS, smoking, genetics, h. pylori which is crucial to the formation of ulcers and thrives in acidic conditions



peptic ulcer disease (cont)

H. pylori	lowers healing and has a high rate of recurrence
gastric cause	breakdown of the mucosal barrier that usually prevents the acid from diffusing to the rest of the body (aspirin, NSAIDS, alcohol and bile)
duodenal cause	excess acid secretion and increased activity of the vagus nerve which stimulates the cells to release gastrin which targets cells to release HCl leading to high HCl
clinical manifests	epigastric burning pain relieved by food or dairy or antacids life threatening complication such as GI bleeding
diagnosis	Upper GI barium contrast radiography, endoscopy to visualize ulcers, testing for h. pylori
treatment	encourage healing of mucosa by reducing acid, prevent recurrence, h. pylori antibiotics, proton pump inhibitors and sucralfate which forms a protective barrier, no smoking, reduce stress

ulcerative colitis

etio	chronic inflammation of rectal and colon mucus, large ulcers forming
clinical manifest and complication	increased risk of cancer after 7 - 10 years, exacerbations and remission, bloody diarrhea and lower abdominal pain
treatment	corticosteroid, broad spectrum antibiotics, salicylate analog, immunomodulating drugs, IV followed by oral cyclosporine for refractory

inflammation - abscess formation - abscesses begin to combine - large ulcers form

Colon cancer

risk factor	age 40+, high fiber and fat diet, polyps, chronic irritation/inflammation, hereditary
diagnosis	recommend colonoscopy every 10 years after reaching risk age



By **jaydevine_23**
cheatography.com/jaydevine-23/

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Colon cancer (cont)

familial adenomatous polyposis:	where three or more family members have colorectal cancer, two generations of colorectal cancer or one or more cases of colorectal cancer before age 50
clinical manifest based on location	right: tarry, black stool left side: intermittent cramping with stringy stool with mucus or blood rectum: change in bowel habits, urgent need to defecate upon waking, rectal fullness, alternating diarrhea/constipation, rectal ache
prognosis	the earlier the prognosis the better, based on tumor size, location, invasion, if it metastasized, and uses the TMN classification
treatment	colostomy (opening colon via abdomen), surgical removal, chemo or radiation

second only to lung cancer in the US

Intestinal obstruction

partial or complete blockage of small/large bowel	mechanical: tumors, hernia, volvulus	functional: inhibition of peristalsis	ogilvie: recurrent bout of ileus
clinical manifest	increase bowel sound, pain, nausea, vomit	absence of bowel sounds	upper jejunal area: vomit, dehydration and electrolyte depletion
risk factors	surgery for adhesions	congenital abnormalities of the bowel	metastatic cancer esp from female reproductive or intestinal tract
treatment	remove mechanical block	surgical intervention or decompression with tube	fluid/electrolyte replacement
other info	if left untreated can lead to perforation/ischemia, and necrosis	necrosis leads to bowel gangrene, sepsis and peritonitis	fluid, gas, water, electrolyte accumulate in bowel

Intestinal gas

altered motility or lack of digestive enzyme caused by swallowing air, normal bacterial/enzyme activity, or neutralization of bicarb in the upper GI

belching normal expelling of swallowed air

abdominal distention failure to digest nutrients or defect in intestinal motility

excessive flatus bacterial digestion of certain foods that are gas causing (legumes, vegetables)

MOTILITY DISORDERS

Irritable Bowel Syndrome IBS

alternating diarrhea and constipation accompanied by cramping with no pathology of the GI tract etiology also unclear

clinical manifest diarrhea, constipation, cramping, mucus in stool, nausea

treatment antidiarrheal agents, antispasmodic agents, high fiber diet

Volvulus

- twisting of the bowel itself resulting in bowel obstruction and blood vessel constriction

- results from 180 twist, ingested foreign body, or adhesion and cannot always be determined

- usually in cecum or sigmoid colon

Intussusception

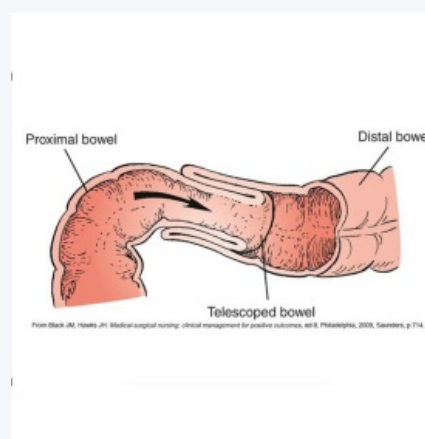
etiology telescoping of a portion of the bowel into adjacent portion resulting in obstruction (bowel pushes itself into the other.)

risk infants and males

factors

treatment surgical

Intussusception



Megacolon

- Congenital or acquired
- Massive dilation of colon
- Cause: prolonged constipation
- Pseudomembranous colitis may result in acute megacolon: surgical emergency.

Hirschsprung Disease

a congenital disease in which the autonomic ganglia are reduced or absent. more common in males than females and in children. Causes difficulty in passing of stool 48 hours after birth in children

- Clinical manifestations
 - Profuse diarrhea, hypovolemic shock, intestinal perforation
 - Stasis of stool and megacolon may occur.
 - Fecal stagnation; enterocolitis with bacterial overgrowth

- Treatment
 - Colonic lavage, surgical intervention

NEOPLASM OF GI TRACT

Esophageal cancer: 1-2% of all

risk factor men are more likely than women, genetic, diet high in nitrosamine, smoking, barrett esophagus, alcohol

prognosis/diagnosis poor prognosis, can quickly metastasize

treatment stent placement, tumor ablation/removal via heat and laser, surgery, radiation and chemotherapy

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By [jaydevine_23](#)
cheatography.com/jaydevine-23/

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small intestinal neoplasms

benign or malignant, accounts for less than 5 %

clinical manifest depends on the type and extent of obstruction, can lead to biliary stasis (stopping of bile) jaundice, bleeding and ulcers

treatment surgical removal of tumor and parts of the intestine - chemotherapy

Gastric carcinoma

risk factor more prevalent in Japan, men older than 30 years, h. pylori infection, Epstein Barr, genetic/dietary factors, smoking

stages are determined by penetration into a major muscle of the stomach and looking at the lymphatic system

treatment aspirin has protective benefits, surgical removal

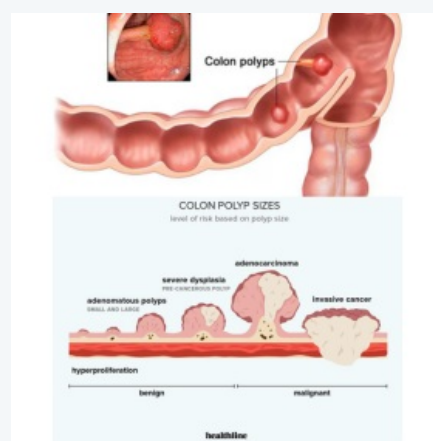
clinical manifest early - asymptomatic | advanced: anorexia, weight loss and bleeding

Colonic polyps

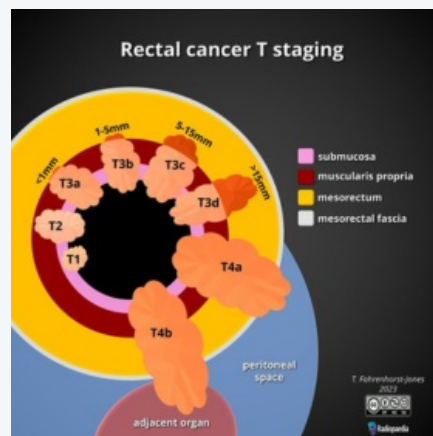
clinical manifest usually none, but may cause gross bleeding and abdominal pain

treatment varies in size, type and location and removed using a scope

Colonic polyps image



Colon cancer staging



Constipation

small, infrequent or difficult bowel movement fewer than 3 stools per week

low fiber, low exercise, slower peristalsis due to aging or pathological disorders fecal impaction can occur where the stool starts blocking the GI tract

MALABSORPTION DISORDERS

DISORDERS REGARDING THE SMALL INTESTINE AND INTAKE OF NUTRIENTS

Celiac disease

a familial intolerance to gluten will lead to inflammation and atrophy of intestinal villi

causes: impaired nutrient absorption due to reduced surface area

diagnosis intestinal biopsy, anti tissue transglutaminase antibody and immunoglobulin A endomysial antibody

treatment gluten free diet, corticosteroids, supplemental folate, b12, and fat soluble vitamin

Tropical sprue/enteropathy

etiology	of unknown cause but usually causes bacterial overgrowth in the large intestine where the mucus membrane is damaged due to fermentation
small intestine	the mucus lining atrophies leading to malabsorption and folate and b12 acid deficiency
risk factor	those who live or visit countries along the equator and in adults more than children
clinical manifestation	bloody diarrhea, abdominal distention and fat in stool steatorrhea
treatment	antimicrobials, antidiarrheals and vitamin/electrolyte supplement

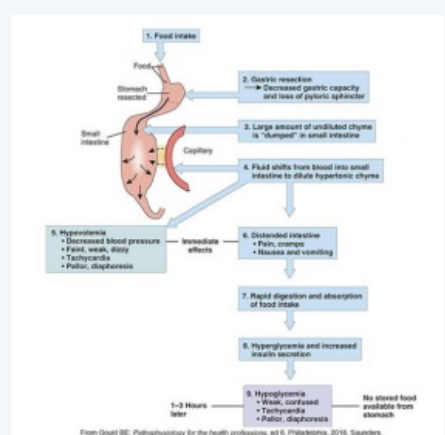
Dumping syndrome

dumping of stomach contents into the small intestine after a short amount of time	most likely due to pyloric sphincter regulation loss - the sphincter is what allows chyme movement from stomach to intestine
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Dumping syndrome (cont)

risk factor/cause	common after gastrectomy, gastric surgery for obesity, cancer or ulcers
due to the large amount of partially digested food entering the intestine it can cause a shift in osmotic causing diarrhea	there is also rapid absorption of glucose in the blood leading to very high plasma insulin but results in low energy a few hours later due to no food storage in the stomach
clinical manifestation	diarrhea, abdominal pain, rapid fall in blood glucose/hypoglycemia
treatment	eating small but more meals about 6 -8 throughout the day

Dumping syndrome image



Short bowel syndrome

due to removal of majority of the intestines	causes severe diarrhea and malabsorption
reduced ability to absorb due to short area to allow for absorption	(esp if ileocecal valve is removed)
clinical manifest	diarrhea
treatment	supportive to nutrient intake

INFLAMMATORY BOWEL DISEASE

Crohn disease

regional enteritis or granulomatous	affects proximal portion of the colon or terminal ileum
etio	chronic inflammation of all layers of the intestinal wall due to obstruction and inflammation of the lymph vessels
diagnostic findings	ulcerations, strictures, fibrosis, fistulas
clinical manifest	fever, diarrhea, right lower quad pain, RLQ mass, tenderness

Crohn disease (cont)

treatment alleviating and reduce inflammation, stop smoking, drugs similar to ulcerative colitis, no definitive care mostly supportive

Crohns v. ulcerative



ESOPHAGEAL DISORDERS

Mallory-Weiss Syndrome

etiology bleeding caused by tear in the mucosa due to excessive vomiting

clinical manifest vomiting of blood (hematemesis) and passing or large amounts of blood rectally

diagnosis endoscopic examination

Mallory-Weiss Syndrome (cont)

treatment blood transfusion thought bleeding may stop on its own, controlling active bleeding with coagulation techniques, epinephrine injection etc.

esophageal varices

etiology portal hypertension from alcoholism or viral hepatitis

causes in tropical areas a species of liver fluke

a high mortality rate and it affects more than half of patients with cirrhosis

Portal hypertension



Vomiting/emesis

forceful expulsion of the gastric contents through the mouth

coordinated sequence of abdominal muscles and reverse esophageal peristalsis

also caused by alterations in the integrity of the GI tract wall and motility (obstruction)

ENTEROCOLITIS

Antibiotic associated colitis

etiology inflammation and necrosis of the large intestine due to clostridium difficile or antibiotics and mediated by bacterial toxins

clinical manifests diarrhea, abdominal pain, leukocytosis, sepsis or perforation

treatment stop antibiotics if possible, treat ischemia or any contributing factors, fecal transplant if severe

Necrotizing enterocolitis

occurs in infants <34 week or low weight infants <5 lbs

etiology diffuse or patch necrosis

etiology bowel ischemia, perinatal oxygen deficiency, use of hypertonic formula

clinical manifest distended abdomen and stomach and perforation (hole in stomach)

treatment fluids, antibiotics, surgery for ischemia or perforation

typhilitis specialized necrosis in adult cancer patient with poor prognosis

Appendicitis

etiology	inflammation of the appendix due to fecalith or stone made of feces
clinical manifest	periumbilical pain, RLQ pain, nausea, vomit, fever, diarrhea and systemic inflammation
treatment	immediate surgical removal, antibiotics, fluids, any localized abscesses may be drained with a tube

untreated appendicitis can lead to rupture and peritonitis

Diverticular disease

etiology	presence of diverticula or herniations on the colon - diverticulosis
cause	low intake of dietary fiber causing high intraluminal pressure
clinical manifest	diverticulosis - asymptomatic diverticulitis: inflammation of the diverticula has fever, acute lower abdomen pain and leukocytosis
treatment	antibiotics and surgery for abscesses

INFLAMMATORY STOMACH DISORDERS

Gastritis: stomach lining

acute	precipitated by ingestion of irritating substances such as alcohol, aspirin, non-steroidal anti-inflammatory drugs (NSAIDs), viral bacteria
clinical manifest	maybe asymptomatic, anorexia, postprandial discomfort, hematemesis
treatment	remove the cause

chronic helicobacter pylori which is transmitted person to person, fecal to oral route or water borne

complication peptic ulcer, atrophic gastritis (thinning of the lining), gastric adenocarcinoma, mucos associated lymphoid tissue and decreased acid + intrinsic factor

Gastroenteritis

etiology	inflammation of stomach and small intestine
chronic	secondary to another GI disorder
acute	direct infection by pathogenic bacteria or toxin
-	may be caused by imbalance in the normal flora

Gastroenteritis (cont)

clinical manifests	diarrhea secretory, abdominal discomfort, nausea, vomit, fever and malaise
treatment	replace fluids and electrolytes



By jaydevine_23

cheatography.com/jaydevine-23/

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