GASTROINTESTINAL DYSFUNCTION (CHAPTER 42)

What is the normal number of defecations (bowel movements) in a day? How do we determine if a patient is constipated?

Defecation "schedule" is **highly individual** from person to person, but in general 2-3 times a day is the **upper limit** of normal.

Constipation is considered to be when bowel movements are **infrequent** (from personal baseline) or **hard to pass**.

What conditions would lead to functional or mechanical constipation?

Functional constipation refers to the failure of the intestines to move material through the bowels at an appropriate rate.

One classic example is an **ileus**, a complication of **anesthesia** in which a patient's bowel peristalsis is slow to return to a normal level after a procedure.

Mechanical constipation, on the other hand, results from a **physical blockage** of the GI tract.

This blockage can be from something like a **tumor**, or from structural abnormalities of the GI tract such as a **rectocele** or **diverticulum**.

How do we determine if a patient is suffering from diarrhea?

The primary indicator is **soft stools**, however in many cases (**large-volume diarrhea**) the overall volume of stool produced is also increased.

Consistency is the main factor, rather than frequency or volume; this can range from soft and mushy to fully liquid, depending on the cause and severity.

Describe osmotic diarrhea.

Osmotic diarrhea occurs when the large intestine fails to absorb solutes from the intestinal lumen, causing a concentration gradient that pulls fluid from the surrounding tissue by osmosis.

One example of this would be diarrhea caused by lactose intolerance: the lack of lactase inhibits the absorption of lactose, causing it to remain in the intestinal lumen and attract water.

Compare to the MoA of an **osmotic laxative** such as **polyethylene glycol** (MiraLAX/Golytely.)

Describe secretory diarrhea.

Secretory diarrhea occurs most commonly when an **inflammatory process** (such as response to **infection**) causes electrolytes to be lost into the intestinal lumen from the body fluid.

This carries water along with it by **osmosis**, resulting in loose stools.

Note that, because water is being **added** to the stool, this is considered a form of **large-volume** diarrhea.

Compare the different types of diarrhea.

osmotic – colloids inside intestines pull water into lumen

secretory – intestines **lose colloids** into intestinal lumen

motility-related – not due to fluid balance; intestine simply doesn't have time to absorb water due to increased motility.

Osmotic and secretory diarrhea are classified as large-volume diarrhea due to the added fluid; motility-related diarrhea is also called small-volume diarrhea.

How is diarrhea treated?

As with any medical condition, try to **treat the cause** if you can. For example, bacterial gut infections (cholera, etc.) are treatable with antibiotics.

The priority is to **maintain fluid volume** by increasing fluid intake or administering IV fluids.

Also, make sure to monitor serum **pH** and **electrolyte levels**! Patients will lose bicarbonate and risk entering **metabolic acidosis**!

Electrolyte replacement (KCl, KH₂PO₄) may be added to IVF to balance out losses.

Sodium bicarbonate (NaHCO₃) may be given IV to correct for pH imbalance.

Compare parietal and visceral abdominal pain.

Parietal abdominal pain comes from the superficial (shallow) areas of the abdomen and is transmitted by "fast" Aδ (A-delta) fibers.

It tends to be a **sharp**, **stabbing** pain that resolves quickly.

Visceral abdominal pain comes from the actual organs, as more of a **dull**, **aching** sensation that **lingers** for a longer period.

List some sources of upper GI bleeding.

The upper GI tract consists of the mouth, esophagus, stomach, and duodenum (the beginning of the small intestine.)

As such, upper GI bleeding refers to blood passing the epithelium into any of these cavities. What might cause this?

peptic ulcers – either gastric **OR** duodenal; remember the duodenum is still upper GI!

gastroesophageal varices – a sign of severe liver failure (we'll talk about this in the next unit)

stomach or esophageal cancer

List some sources of lower GI bleeding.

The **lower** GI tract is everything else: the **ileum** and **jejunum** of the small intestine, as well as the **colon** and **rectum** of the large intestine.

Why might we have bleeding in the intestines?

diverticulosis or diverticulitis

inflammatory bowel disease (IBD) – especially ulcerative colitis (UC!)

colorectal cancer – often starts as occult bleeding

internal **hemorrhoids**

Define hematemesis, hematochezia, melena, and occult bleeding.

hematemesis – bloody vomit (emesis)

hematochezia – bright red, bloody stool

melena – dark, "tarry" blood in stool

occult blood – invisible trace amounts of blood in stool

One common cause of **hematemesis** is **gastroesophageal varices**, which are related to liver failure—we'll talk about this in the next unit.

Can also be caused by **peptic ulcers** in the stomach or damage to the esophageal mucosa.

Hematochezia is caused by "late" bleeding in the lower GI tract, primarily in the large intestine.

The blood is still fresh when it exits the body, thus retaining its **bright red** appearance.

Melena is caused by "early" bleeding in the upper GI tract, such as the stomach, esophagus, or duodenum.

Partial digestion of hemoglobin in the blood results in its dark, "tarry" appearance.

Occult blood can be from anywhere in the digestive tract, and occurs in small amounts only detectable by testing.

There are a wide range of explanations, from peptic ulcers or hemorrhoids to colorectal cancer.

Define dysphagia. Compare mechanical obstruction with functional dysphagia.

dus- (bad, difficult)

-phagía (eating)

DYSPHAGIA

"difficulty swallowing"

(Don't confuse with **dysphasia**—difficulty speaking or understanding language)

Also: note "difficult," not "painful"—painful swallowing is odynophagia

Mechanical obstruction refers to the **physical blockage** of the upper GI tract, e.g. due to esophageal cancer.

Functional dysphagia results from neuromuscular issues that halt or slow **peristalsis**, or interfere with voluntary swallowing.

Define achalasia. Compare the three types of achalasia.

a- (not)khálasis (relaxation)

ACHALASIA

"an inability of the gastroesophageal sphincter to relax, causing food to become stuck in the esophagus"

Achalasia has three primary causes:

type 1 – decreased contractility due to atrophy or nerve damage

type 2 – contraction of the entire esophagus simultaneously

type 3 – uncontrolled spasm of the distal (lower) esophagus

Describe the clinical manifestations of dysphagia.

Inability to swallow effectively may result in regurgitation of food, as well as chest pain or discomfort when attempting to eat (odynophagia.)

If dysphagia persists, there may be **changes in dietary habits** resulting in **weight loss** over time.

What is gastroesophageal reflux disease (GERD?)

Describe its pathophysiology and treatment.

GERD, commonly referred to as "heartburn," is a common cause of "burning" mid-epigastric abdominal pain, especially shortly after eating.

It results from acidic stomach contents spilling up through the gastroesophageal sphincter, burning the esophageal mucosa.

Avoid lying down shortly after eating. Can be treated with medications that reduce acid (antacids, proton pump inhibitors.)

What is a hiatal hernia? Compare sliding and paraesophageal hernias.

Normally, the esophagus enters the abdominal cavity through the "esophageal perforation," a hole in the muscle of the diaphragm.

The stomach can get pulled up through this hole, resulting in herniation.

Gets worse with pressure on abdomen, e.g. lying supine or wearing tight clothing.

In a **sliding** hernia, the topmost part of the stomach simply gets **pulled** directly out through the esophageal perforation.

In a **paraesophageal** hernia, a pouch of the stomach pokes out through the hole, leaving a chance that the tisue will become **incarcerated** and not return to the proper position.

Describe pyloric obstruction. What are its consequences?

The **pylorum** is the lower part of the stomach, just prior to the start of the **duodenum** of the small intestine.

Pyloric obstruction (or **gastric outlet obstruction** refers to the blockage of the **pyloric sphincter**, which is the "exit" of the stomach into the intestines.

The primary symptoms are epigastric **abominal pain** and postprandial (after eating) **nausea/vomiting** due to the stomach not being able to empty properly.

Over time, **malnutrition** or **dehydration** can result from this blockage of the GI tract.

Compare small and large intestinal obstruction.

Small bowel obstruction refers to a **blockage** of the small intestine, usually due to tumors, herniation, or post-surgical scarring.

This can be concerning as it prevents **water** from reaching the large intestine for absorption, potentially causing **fluid imbalance**.

Large bowel obstruction refers to a blockage of the large intestine, again often caused by tumors or scarring, or potentially fecal impaction.

These obstructions can result in **constipation** and abdominal **distension** (bloating,) as well as **pain** or **cramps**.

Compare acute and chronic gastritis.

Acute gastritis is typically associated with infection by the bacterium **Heliobacter pylori** (H. pylori)

Inflammation can also be brought on by excess consumption of **alcohol** or **spicy foods**, but it is **usually** infectious.

Chronic gastritis, likewise, can be caused by chronic H. pylori infection, or by long-term alcohol abuse.

It may be more common in the elderly due to weakening of the stomach lining, and typically has the same risk factors as acute gastritis and peptic ulcer disease (PUD.)

Compare the types of peptic ulcer.

Peptic ulcers can occur in either the stomach (gastric ulcers) or the first part of the small intestine (duodenal ulcers.)

They can occur due to an infection, such as H. pylori, or sometimes as an adverse effect of chronic NSAID use.

Symptoms include a burning, aching epigastric pain, which may get better or worse after eating, as well as vomiting and changes in appetite.

Typically, **gastric ulcers** tend to produce pain which worsens immediately after eating, whereas **duodenal ulcers** show **improved pain when eating**, only to worsen 2-3 hours later when the food has passed into the duodenum.

What factors are implicated in the development of ulcers?

Gastritis, particularly if caused by H. pylori, can weaken the stomach lining and make it easier for ulcers to form.

Smoking and **alcohol abuse** are also a significant risk factor, as is **chronic use of NSAIDs or aspirin**.

Contrary to popular belief, **stress** is believed **not** to lead to the **formation** of peptic ulcers, although it can contribute to **delayed healing** of ulcers that already exist.

Describe dumping syndrome. How does it develop?

Gastric dumping syndrome, a potential complication of gastric bypass surgery, occurs when food passes through the stomach and into the small intestine **too quickly**.

Common symptoms are nausea, abdominal cramping, tachycardia, lightheadedness, and diarrhea.

Extreme diarrhea can even result in hypovolemia (low blood pressure due to fluid loss.)

Describe alkaline reflux gastritis. How does it develop?

In **alkaline reflux gastritis** (biliary reflux,) contents from the small intestine reflux through the pyloric sphincter back into the stomach.

Damage to the parietal cells results in decreased intrinsic factor production, leading to **pernicious** anemia.

(No longer able to absorb folate/iron as effectively, impairing hemoglobin prouction.)

What are the consequences of gastrectomy?

Two potential complications of gastrectomy are the ones that we just covered: **biliary reflux** and **dumping syndrome**.

Others include **diarrhea** and **weight loss** due to impaired digestion, as well as **pernicious anemia** due to decreased production of intrinsic factor.

Compare malabsorption with maldigestion.

malabsorption – failure to absorb nutrients from chyme (digested food) in the intestines

maldigestion – failure to digest (break down) food in the upper GI tract

Remember that digestion occurs **before** absorption; these are two **different** processes that must occur **in order**!

Digestion is **breaking down** the food to allow absorption.

Absorption is actually taking the nutrients **into** the capillaries from the intestines.

Describe some malabsorption syndromes.

Celiac disease is a fairly common autoimmune condition in which a hypersensitivity to gluten (a protein found in wheat and some other grains) causes the immune system to attack and damage the intestinal lining, impairing absorption.

Other diseases of malabsorption involve deficiencies of **enzymes** or **other chemicals** needed to process food in the intestines.

This includes **pancreatic insufficiency** (lack of pancreatic enzymes) which interferes with the absorption of carbs (**amylase**,) proteins (**trypsin**,) and fats (**lipase**.)

Bile salt deficiency or **cholestasis** (bile blockage, e.g. by gallstones) can also interfere with **fat absorption**.

Describe inflammatory bowel disease (IBD.)

The two types of IBD are ulcerative colitis (UC) and Crohn's disease.

ulcerative colitis – inflammation of mucosa of large intestine (colitis,) especially the rectum

Results in abcesses and hemorrhage of the colon, presenting as hematochezia

Crohn's disease – inflammation of mucosa anywhere in the GI tract, but often "rectal-sparing"

Unlike UC, usually does not involve blood in the stool

Note that **inflammatory bowel disease** (IBD) is **not** the same as **irritable bowel syndrome** (IBS!)

Describe diverticulosis and diverticulitis.

Diverticula are "pouches" formed in the intestinal mucosa (usually the sigmoid colon) by the mucosa bulging outward.

Diverticulosis refers to the **presence** of diverticula, which are not a normal occurrence.

When diverticula become **infected**, the condition is knows as **diverticulitis**.

Describe some explanations for the development of appendicitis.

The exact cause that precipitates the development of appenditicis is **poorly understood**, but most theories point to the **occlusion** (blockage) of the entrance of the appendix (e.g. with stool,) allowing an infection to develop inside.

The appendix becomes **inflamed** and **bloated**, and ultimately needs to be removed via **appendectomy** in many cases.

What is vascular insufficiency? Why is this problem serious?

The GI tract is supplied by three major arteries that split off from the abdominal aorta:

celiac artery – supplies the stomach, liver, and spleen

inferior and superior **mesenteric arteries** – supply the intestines and pancreas

When **both** mesenteric arteries (inferior and superior) are blocked or compromised, this results in **mesenteric ischemia**.

Acute mesenteric ischemia (AMI) can result in bowel necrosis within 6 hours, followed by the intrusion of gut flora into the bloodstream and ultimately **sepsis** or even death.

What is the importance of studying obesity?

Obesity, particularly abdominal obesity, is a major risk factor for a number of chronic health problems.

The obesity rate in the U.S. is now **over 40%**, so understanding these health risks has a huge impact on evidence-based patient care.

Compare anorexia nervosa and bulimia nervosa.

Anorexia nervosa is a psychological disorder that causes disturbed body image and a sharp decline in appetite, usually resulting in severe weight loss (or failure to gain appropriate weight in children.)

Those who suffer from anorexia nervosa exhibit **self-starvation**, or failure/refusal to eat enough to maintain the body's needs.

Bulimia nervosa is a similar psychological disorder characterized by disturbed body image, however it manifests in a cycle of binge eating followed by purging.

Purging may take any number of forms, from simply fasting to "make up" for the added intake, forced vomiting, or abuse of pharmaceutical laxatives.

How does the body deal with short-term starvation or long-term starvation?

In the presence of a **short-term** carbohydrate defecit, the body (particularly the **liver**) begins to produce glucose from existing glycogen stores (**glycogenolysis**.)

The body also begins to reappropriate fats and proteins (through **gluconeogenesis**) to make up for the temporary energy shortage.

Remember that gluconeogenesis produces acidic **ketones**! Although, in the short term, the ketone levels remain low enough that the body can **compensate** for the added acidity.

If the caloric deficit becomes a **long-term** problem, the body begins to "cannibalize" existing tissue, breaking down fats stored in the **adipocytes** as well as protein from **muscle** tissue.

As **gluconeogenesis** from lack of carb intake continues, **ketones** in the body can continue to build up, leading to **starvation ketoacidosis** which is lifethreatening!

What condition results from absolute deprivation of food?

Severe deficit of caloric intake results in a condition known as **marasmus**.

Marasmus presents as a shriveled, bony appearance with prominent ribs and thin, atrophied extremities.

What condition results from protein deprivation?

Severe protein deprivation with **adequate** caloric intake results in a condition known as **kwashiorkor**.

The major presenting symptom is **ascites**, or abdominal edema, which occurs due to the **underproduction of plasma proteins** and accompanying decrease in **oncotic pressure** (remember **fluid balance**?)