Respiratory stuff

Ventilation = ability of air to reach all parts of the lungs
Perfusion = how well the blood circulates within the lungs

General principle to know: A deficiency state is a fnx of a gap between what is available and what is needed…more so than an “absolute” amount.

A ventilation perfusion mismatch = alveoli are not getting the amount of perfusion they need. The gap between what is needed and what is available can be lethal. When there is a ventilation perfusion mismatch – “VQ mismatch” in med speak – 

Pulmonary embolism. Blood clot originating from a thrombus from the venous side of circulation which works its’ way up to the right side of the heart, thru the atrium/ventricle, then goes from pulmonary artery to the lungs. While it’s in large vessels all is swell. When it gets to the lungs, hits the small vessels and will eventually plug it. Cuts off perfusion to everything downstream. The alveoli will tend to collapse and blood vessels around it will constrict. This puts the patient into a cascade of Very Bad Things….mismatch between what is needed and what is available. Some people can survive these just fine because their needs are lower. Some people are dead as a result because their needs are higher.

Disorders of Gastrointestinal Function

Chapter 39 – Disorders of Gastrointestinal Function, slide 50.

Digestion….and why the hell are we talking aout this…no preamble from perfusion to this, but here we go…

One of the digestive organs = gb. Bile produced in liver, stored in gb. Bile needed to brk down fats
Pancreas produces enzymes = lipase, protease, amylase, etc. these too are needed for digestion.
Mechanical functions of the stomach trigger the release of many digestive enzymes. Amounts of enzymes released depend upon how easy to digest and mash the food is in the stomach. If you chew well and eat easily digestible foods, far fewer enzymes and stomach acids are needed. Higher levels of acid in the chyme (beat up food bits), require more enzymatic activity and levels.

The autonomic nervous system regulates all of this. Releases bile for instance in a wave or bolus rather than in a trickle. Why? It’s caustic stuff. You don’t want a trickle going thru the biliary tract all the time! Cholelithiasis or GB stones is a common problem with the GB. Pain is associated = pain in RUQ radiating to midline or back. Also called “barrel stave pain” or pain that wraps the body. Can also just be central to the gastric area that feels intense, pressure, boring thru the body type of pain. This kind of pain can be st, gb, pancreas. Don’t assume it’s stomach.

Risk factors for gallbladder disease:
  High cholesterol. The stones themselves are cholesterol and bilirubin + calcium
Obesity and/or rapid massive weight loss
High estrogen
5 F’s: fat, fair, female, forty, fertile
That said, everyone is susceptible to gallbladder disease.

Caused by:
- Change in the composition of the bile
- Bile stasis
- Inflammation of the gallbladder

**Acute Cholecystitis**
Can develop a biliary obstruction without a stone! Rare, but it happens. Even comes with the fun jaundice package on occasion. Blockage can be complete or partial. The stone is mobilized following a fatty meal

**Symptoms:**
- Biliary colic – right upper quadrant and/or epigastric pain. Often on the back or right shoulder – referred pain. (Pancreatitis can express as right shoulder pain too.)
- Often follows a fatty meal.
- Indigestion, vomiting, about ¼ have jaundice.
- Intensifying pain with fever as it progresses
- Elevation of liver function tests: bilirubin, AST, ALT, alkaline phosphatase

**Chronic Cholecystitis**
Comes from chronic irritation by stones or repeated bouts of acute cholecystitis. This is associated with acute exacerbations of inflammation, common duct stones, and pancreatitis.

Cholecystitis is diagnosed through imaging.

**Acute Pancreatitis**
Slide 58. This is the escape of pancreatic enzymes into the pancreas and surrounding tissues. This cause the “autodigestion” of the pancreas.

**Causes:**
- Gallstones with obstruction
- Alcohol – causes rupture of cells that contain the digestive enzymes – autodigestion of pancreas
- Hyperparathyroidism and associated hypercalcemia
- Hyperlipidemia (blood looks creamy and thick – greyish in color)
- Infections and drugs – i.e., thiazide diuretics and steroids. Oral hypoglycemic medications can cause it too.

Presents as epigastric pain, radiating to the back. Bowel sounds will be hypoactive – bowel shuts down, so no sounds. Also look for tachycardia (usually above 120), which happens fairly quickly, then hypotension, and fever as the disease progresses. **Cardinal sign: epigastric pain with vomiting** (pretty violent vomiting at that).
Lab abnormalities include elevated serum amylase and lipase (only produced in pancreas). Others: hypocalcemia, hypoglycemia, hyperbilirubinemia, leukocytosis, fall in the hematocrit.

Often dx’d with clinical presentation, lab work, CTs and ultrasound. Tx is to keep off of food/liquids/pain control for 72 hours. Resume feeding very slowly.

**Chronic pancreatitis**

Types:

1. Chronic calcifying pancreatitis – though don’t necessarily have the hyperparathyroidism. Could be result of many episodes of acute pancreatitis. It is the deposition of calcified protein plugs in the pancreatic ducts. Often seen in alcoholics.

2. Chronic obstructive pancreatitis – keeps passing gallstones or may have small calcified stone in pancreatic duct. Anything obstx’d pancreatic duct can cause pancreatitis. Cystic fibrosis. This is an obstx of the pancreatic duct by stenosis of the Sphincter of Oddi.

Sx:

- Rather like acute version, less severe
- Progressive loss of exocrine and endocrine fnx of the pancreas: diabetes, malabsorption

A lot of folks with acid reflux disease = over production of acid, excessive amts of digestive enzyme secretion. More commonly, ppl produce insufficient amounts when needed, but production is not stopped when it is *not* needed. Due to autonomic nervous system, parasympathetic division? That’s what Dr. Stewart thinks.

**Cancer of the pancreas – slide 55**

4th leading cause of death…*most have metastasized by the time of diagnosis*. Causes include smoking, a diet high in fat, meat, salt, dehydrated/fried foods, refined sugar, soy beans, nitrosamines. You are protected by a high fiber diet, vitamin C, fruits/vegs, no preservatives. Pancreatic cancer goes to stomach, bowel and brain.

Symptoms: pain, jaundice, weightloss.

Most pancreatic cancers are in the head of the pancreas, leading to obstx of bile duct and jaundice. Diagnosed by ultrasound and a CT.

**IBS** – slide 3

Irritable Bowel Syndrome. The mechanical fx of the digestive tract as well as the secretory functions are mediated thru the autonomic nervous system. Many of the IBS type problems come from the nervous system input not working consistently. A lot of bowel disorders are termed “motility disorders.” Small and large bowel are designed to contract from superior to inferior down the chain. This is missing in ppl with IBS.

IBS is a fnx disorder of the GI characterized by a variable combo of chronic and recurrent intestinal symptoms not explained by structureal or biochemical abnormalities.

Sx, which wax and wain in intensity:
- Ab pain
- Altered bowel fnx
- Flatulence
- Bloating
- Belching
- Anorexia – don’t want to eat when it is hitting them hard because food makes it worse.
- Nausea
- Constipation and diarrhea

Symptoms can be relieved by defecation. Are assoc’d with a change in consistency and frequency of stools. Though the slide says that the cramps, intermittent, and lower ab pain recede at night and don’t interfere with sleep, that’s bullshit. It does affect patients at night and does interfere with sleep. This old school belief is part of the “it’s psychosomatic” assertion. A lot of Western docs believe if an illness is considered psychosomatic, that you just need therapy and that you’re generating the problem somehow.

That said, it does have strong psychogenic associations – mood and emotions can indeed affect it. There’s also a pattern of familial association.

The cause of IBS is not clear – but it is a disregulation of intestinal motor and sensory funx modulated by the CNS.

IBS is diagnosed by exclusion – it is considered an organic disease with acute onset, weight loss, anemia, occult blood, symptoms at night, and evidence of malabsorption. IBS also predisposes you to motility disorders.

<<See the Kidney Energetics lecture for the adrenal treatment that can help relieve IBS type symptoms. Kidney 1 and spleen treatments can help too. >>

**Inflammatory Bowel Disease**

Encompasses both Crohn’s Disease and Ulcerative Colitis. Small intestine is more likely to have a motility problem than the large intestine. The LI is more likely to get involved in a widespread inflammatory rx. Inflammatory Bowel Disease is more likely to be LI. Crohn’s and UC are distinctive disorders but share these things: inflammation of the bowel, no proven causative agent, familial occurrence. Both are a result of activation of inflammatory cells which cause nonspecific tissue damage

Both are characterized by remission and activation of diarrhea, fecal urgency and weight loss. Systemic manifestation can include skin lesions, arthritis, and anemia.

Causes are largely unknown, but there seems to be a genetic susceptibility triggering autoimmune rx. Can be triggered and furthered by a dietary antigen or perhaps a microorg.

**Crohn’s Disease:**

Also a form of cholitis. A recurrent granulomatous inflammation that can be anywhere in the digestive tract. Mostly small and large bowel, perhaps both. When chronic, fibrosis and thickening of the bowel wall. Tissue should be soft and elastic, gets thicker and more sclerotic. Get functional changes in the bowel as a result. The submucosa is strongly affected. Often see “skip lesions” which are sharply demarcated lesions.
Usually seen in ppl in 20’s-30’s+, females slightly more than males. Rare onset in adolescence. Can lead to abscess – body trying to wall it off.

**Dx:**
Sigmoidoscopy, upper GI with small bowel, barium enema to eval extent of disease and the presence of fistulae. CT scan will detect abscess formation.

**Ulcerative Colitis**
Nonspecific inflammations, confined to colon and rectum. Begins in rectum, spreads proximally. Usually in the sigmoid and rectum—if gets to transverse, m that’s BAD!. Mucosa is primarily involved. Lesions are continuous, not skip lesions.

Diarrhea, often blood and with mucus. Abdominal cramping is mild, fecal incontinence is common, anorexia, weakness, and fatigue are clinical manifestations. Can be mild to fulminant – rip roaring.

UC increases the risk of colon cancer.

Dx’d by a proctosigmoidoscopy.

Both UC and Crohn’s are very inflammatory of the intestinal wall for reasons currently non-understood.

**Diverticular Disease**
Can be diverticulosis and diverticulitis. A diverticulum is an “outpouch” of gut lumen, probably caused by pressure changes in the gut. More likely to occur when the colon is working very hard to move things thru. Dehydration is causative as well. Major causes:
- Lack of dietary fiber – fiber tends to be hydrophilic, makes stools wetter and easier to pass – fiber also gives the colon something to push against, assisting in movement. Colon only absorbs water you need. If you are dehydrated and eat a lot of fiber, you constipate yourself!
  Our ancestors evolved to handle 120g of fiber per day – largely indigestible bug shells, barks, wild grains, etc. Had 3-4 large bulky stools per day.
- Decreased physical activity – assists colonic activity
- Poor bowel habits – chronic constipation for instance.
- Aging –
Diverticulitis is an inflammation of the diverticuli. Can also become infected. Needs antibio’s.

**Appendicitis**
Common. Most frequently in 5-30 y.o. males. Usually due to obstruction of the appendix, often by a “fecalith” or poop stone. Appendix perforates when not treated.

Presentation rules:
- Uncommon presentation of a common disease is more likely than a common presentation of an unusual disease.
- Abrupt onset, brief is the rule for appendicitis.
- Usual presentation is RLQ. Less common is periumbilical or epigastric pain…but this usually shifts to RLQ.
• N/V, anorexia, diarrhea
• Leukocytosis, neutrophilia (neutrophils, PMNs, polys)
• Tenderness to palpation in the RLQ (McBurney’s Point) with or without rebound pain.

Don’t assume all symptoms will present!

**Diarrhea**
Causes can include infection, food intolerance, drugs, intestinal disease. Intestinal disease can include IBD, IBS, malabsorption, endocrine disorders, radiation colitis.

Also, diabetic neuropathy – autonomic nerves dysfunction. Far more common however, diabetic neuropathy will cause constipation.

Acute diarrhea often caused from infection. Lasts less than 4 days, self-limiting.
Chronic diarrhea, lasting longer than 3-4 weeks, usually caused by intestinal disorders.

Large volume diarrhea results from an increase in water content in the stool. 2 types: secretory, osmotic.
Small volume diarrhea results from an increase in peristalsis. See pg 904.

**Fecal impaction**
Note that some patients present with watery diarrhea – the rectum is full of stool, becoming distended.
Once it reaches a certain level of distention it won’t contract any longer. The watery diarrhea is the stuff that will fit around the impaction. See slide 24

**Malabsorption Syndrome**
Hepatobiliary disease is the most common cause. 2\textsuperscript{nd} on the hit parade: pancreatic insufficiency. Also, intraluminal bacterial growth.

Symptoms include steatorrhea (fatty stools, very very stinky), diarrhea, cramping, distention, flatulence, bloating. Tend to become very vitamin K deficiency, causing very thin blood and increased risk of bleed out. Vitamin D deficient too.

**Celiac Disease**

**Colorectal cancer**