

GI System

- Most important thing when GI complain
- If can't reproduce the pain = referred pain
- Two categories
 - Portion of GI that sends pain to the body surface
 - Can localize
 - Can poke on it and make it hurt
 - Retroperitoneal structures-
 - Referred pain pattern ONLY
 - Can't reproduce pain by palpation
 - Ascending colon and descending colon-refers to front and back and lateral
 - Colitis issues
 - ◆ UC (L-sided)
 - ◆ Chrohn's (R-sided)
 - Pancreas
 - Belt line pain (right below the ribcage and crosses multiple rib spaces)
 - Duodenum - bowl of
 - Largely retroperitoneal except right at the beginning (near the pylorus)
 - Good bit of the stomach
 - Ileocecal valve

-esophagus: retrosternal (or epigastric pain)
 GERD: also retrosternal
 -MI: sternal pressure
 -GI pain patterns can sometimes imitate cardiogenic pain

Imaging Tools

- Plain Film- KUB
 - Can see dome of bladder
 - Can't see top of kidney
- Contrast based tools
 - Esophogram
 - Simplest form is oral contrast agent: Barium sulfate- suspended metal, visualize lining
 - Goes through oropharynx and then watch distal esophagus (2 1/2 peristaltic waves is normal), most solids and liquids will clear in this way
 - Upper GI- most common of anterogram
 - Give CO₂ tablets to blow stomach up, stretch rugal folds and have them roll over fully coating and then we watch
 - Starts in the typical location by watching mouth shape bolus and we keep watching until goes through first 10 cm of duodenum
 - Barium Enema
 - Introduce agent through a feed system
 - Work contrast all the way to ileocecal valve- want a little leakage into ileocecal valve
 - Add a negative density by pumping air into colon (the most uncomfortable part)
 - ★ The people that hate this the most tend to be the ones who need it the most
 - Upper GI with small bowel follow through
 - We don't do this often because low yield- most abnormalities in first 10 cm- costs more money
 - If frequency of bowel movements is low enough to be concerning- you want to tract *transit time*
- Diagnostic Ultrasound
 - Pancreatitis can be seen on US
 - Very hard to read and use
- Helical CT
 - Absolute requirement to be helical
- MRI
 - Also carved out a spot for itself in the abdomne
 - Big fixed organs- kidney, liver, hepatic flexures, aortic, vena cava
 - Not good with duodenum

★ *Esophagus starts at C5*

Factors and Conditions Associated with Esophageal Cancer

- Smoking
- Alcohol
- Head and neck cancer
- Barrett esophagus- adenocarcinoma
- Corrosive esophagitis
- Achalasia
- Tylosis (palmar and plantar)

- Plummer-Vinson syndrome

★ **History in the GI patient is important** - lost of different sx and locations of the problem

Location of Pain

- Sternum is a common location (fist on sternum and pressure -->MI until proven otherwise)- "gorilla on chest" (*cardiogenic vs. non-cardiogenic*)
 - Only hurts when you swallow = non-cardio (but not always)
 - Pressure on the chest- think MI
 - Clinched fish over the heart is likely heart
 - Finger pads over sternum is esophageal pain
- Retrosternal Complaint = think esophagus
 - 50% have pain as they rub right over the sternum- "burning"
- Shoulder pain
 - Diaphragm
 - Gallbladder- right side mostly (sometimes left)
- Epigastric pain
 - Stomach, duodenum, gallbladder
 - C sweep of the duodenum will also cause this
 - Liver
 - Hepatic Ducts
- Mid back pain - "Designer belt"
 - Pancreas
 - More narrow but still in same area = aorta
 - ◆ Arch is the most common site of dilation but the descending portion is the most deadly
 - Testicular and vulvar pain may present here

Onset of Pain

- Rapid Onset
 - High grade obstruction fills up quick and causes trouble quick
 - Rupture
- Gradual Onset
 - Cancer is most dangerous
 - Inflammation- lg imbedded in tissue
 - Irritation (gastritis for ex.) --> often event-related (like from eating spicy foods) - only hyperemic ; insulting agent

How Long Does Pain Last

- Short Bouts of Pain
 - High Grade Obstruction
 - Nothing gets past - stretch receptors constantly stimulated - once contraction wave stops it recoils and goes back/pain relieves
 - Colicky pain- waves of pain
 - Unsatisfied contraction
 - Distention will produce transient pain pattern
- Long Bouts of Pain

Character of the Pain

- Cramping pain is motor based- limited to muscles that can contract
- Burning pain is pH butting up against tissues that can't tolerate it
- Deep pain = cancer

Board Review- Key GIGU points

- Most common location for Diverticulum: Sigmoid Colon
- Appendicitis- mostly has roots in obstruction- something plugged up opening - blind sac
 - Meckel's diverticulitis imitates appendicitis - iliem thing- used to be part of maternal/fetal circulation- aka. Vitiline duct and omophalomenteric duct
 - Periumbilical pain, diarrhea followed by constipation, moderate spike in WBC
 - Rebound tenderness, low grade fever, peritonitis leading to rupture is our concern
- Peptic Ulcer (H. Pylori) - bacteria - tx. Is antibiotic tx
- Dysphagia- difficulty swallowing
- Bowel disease that can produce that can cause joint pathology
 - #1 is Chrones
 - #2 is Ulcerative Colitis
 - #3 is Whipplis - old white guy overly protective of his toilet paper because of his diarrhea - an inflammatory bowel disease

- Gallbladder disease
- Most common constituent to make up gallstone- cholesterol
- Oxalate gallstones, oxalate renal stones should make us think of Chrones disease
- Eating fatty foods causes a cholecystitis attack

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Burning Pain Differential

- Associate with change in pH
- If when they are upright we think maybe pyloric reflux
- Upper pain that is bias laterally think duodenal bulb

Dysphagia

- Difficulty swallowing - difficulty clearing the pharynx or esophagus
- Vs. Odynophagia which is painful swallowing
- Sometimes neurological in nature
 - Post stroke is classical example
- In scleroderma the myo-neural junction may have abnormal connective tissue that will cause dysphagia
- Can also be a component of SLE
- High grade obstruction and rupture can be the end result
- Causes
 - Space occupying lesions (such as tumors) can cause
 - Constrictions
 - Congenital narrowing
 - Acquired narrowing (occurs in chronic reflux patients) -causing acquired fibrosis
 - Esophageal varices
 - Any liver disease that leads to fatty degeneration or cirrhosis is capable of producing portal hypertension causing engorgement of venous system of paraesophageal veins
- Fixed Dysphagia
 - Tends to be more congenital
 - "Whenever I don't chew my food thoroughly I get the feeling of dysphagia but as long as I chew thoroughly and wash it down with fluid I'm fine"
- Total Dysphagia
 - Can't swallow solids or liquids
 - This is probably neurologic
 - Stroke most likely
- Have the patient point to where they feel the problem- very often the source of the problem
- Cancer can produce a progressive dysphagia- pushing from the outside
- Barium Swallows will allow us to watch esophagus work
 - Esophagram
 - Upper GI
- Other dynamic test
 - *Mynometer*- detects pressure
 - Induce swallowing and measure the pressure in each zone
 - Often inserted under endoscopic guidance so we can add precision
 - pH testing can also help indicate what's going in
 - Especially in determining presence of GERD

Overview: Patient starts with simple complaint of difficulty swallowing

- Ask them where they feel, under what conditions, what makes it better, worse, what can/can't swallow - foods, solids etc
- Can use anatomy to help give us landmarks of where we are
 - Pharynx down to C5
 - ◻ Pre-esophageal dysphagia
 - Esophagus starts right around C5 - tracheal air shadow moved forward to about 20 mm
 - ◻ Sternal notch C5 down is upper esophagus problem

Plummer-Vinson Syndrome

- Pre-esophageal dysphagia
- Profound iron deficiency anemia
- Traditionally two categories of people who acquire this syndrome
 - Very young kept on a long and solitary diet of mother's milk- not enough iron availability from that source
 - Older patients - could be GI blood loss or dietary deficiency
 - Poor fitting dentures and lack of money is a common source of dietary deficiency
- Series of webs criss-cross above the esophagus - *Pre-Esophagus webs*
 - Can't see on contrast studies
 - Seen with laryngoscope (mirror on stick) or any of flexible fiberoptic tools
- Never any trouble with liquids but "I'll cough and throw up if I try to drink my vegetable soup"
 - Solids comes up against webs- reverse peristalsis


- Spoon Nails- Koilonychia
 - Also may see splitting of and brittleness of fingernails
- Glossitis- Beefy Red Tongue
- Iron supplementation is proper management as well as blow a balloon up in the esophagus to break up the webs and then supplement with lots of iron
- If you break up the web and don't supplement enough iron the webs will come back and be thicker than before

Causes of pre-esophageal dysphagia

- Space occupying lesions- tumor
- Webs
- Para-esophageal varices
- Fibrosis from alcoholic

Esophageal Lesions

- Shotsky rings
 - A ring
 - Just a little ledge sticking out
 - Can become a membrane that causes problems
 - B ring
 - C ring
 - Represents junction between esophageal tissue and gastric tissue
 - Histologically different on both sides of the ring
 - GE junction
 - Rich area for cancer
 - Aging phenomenon
- Atresia patients
 - No hole or small hole penetrating
 - Everything that comes in comes back out

 *Edit this when we revisit it in detail*

Factors and Conditions Associated with Esophageal Cancer

- Smoking
- Alcohol
- Head and neck cancer
- Barrett esophagus- can lead to adenocarcinoma
 - Conversion of esophageal tissue into gastric tissue
 - GERD patients would benefit from this- appears to be the driving force- body gets insulted/stimulated and it reacts when it can
- Corrosive esophagitis
- Achalasia
- Tylosis (palmar and plantar)
- Plummer-Vinson syndrome

Apple Core Lesion

- Hallmark features of annular carcinomas
- Wrap all the way around
- The only amount of esophageal space left in the patient
- Can't pass solid or liquids

★ Narrowing that crosses the esophageal hiatus indicates **Gastric Carcinoma** that's spreading north

Adenoma- benign cancer - 40% of all adenomas will convert to adenocarcinoma so we want to follow this condition

Leiomyoma- virtually never causes trouble unless unluckily located near a sphincter causing stiffness

Polyps- can be anywhere within system

Can be pedunculated- attachment to wall and a head

Statistically associated with adenocarcinoma when the head is bigger than 1 cm (?)

Intussusception- telescoping of the bowel

Wide mediastinum on chest film can make us think of lung cancer

Proposed Risk Factors of Esophageal Cancer

Cigarette smoking

Alcoholism

Nutritional deficiencies

Carcinogens

Endemic microorganisms

Regional practices

Soil salinity

Squamous cell carcinoma Risk Factors

- Achalasia
 - Spasm of lower esophageal sphincter
- Celiac sprue
 - Hypersensitivity reaction- often in first part of duodenum
- Lye stricture
- Plummer-Vinson syndrome
- Head and neck cancer
- Tylosis

Adenocarcinoma Risk Factors

- Barrett's esophagus

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Scleroderma

- Vascular Obliteration and Fibrosis in Smooth Muscle
 - Weak LES
 - Ripples tend to be connected to inspiration
 - No measurable pressure wave (contractions) on the mynometer
 - Poor esophageal contractility
 - Lose normal advancing of peristaltic wave
 - Delayed gastric emptying

Diverticulum

- *Zenker's Diverticulum*
 - Cricopharyngeus muscle defect
 - Right below thyroid cartilage
 - Puts defect about C5 and can get big enough to hang down several levels
 - Lays behind spine and directly behind tracheal shadow
 - Good example of a check valve - can only get stuff through and not back
 - Always in upper 1/3 of esophagus
 - Sometimes a born with lesion- never have closure
 - Sometimes acquired- powerful pressure events
 - See herniation- forcing mucosal tissue out through defect
 - Food tends to undergo putrefaction
 - When sleep patient they can relax and zenker's can decompress itself causing some putrid/foul smelling product to come up esophagus and potentially go down airway
 - Aspiration Pneumonitis
 - Person on second or third pneumonia need to be asking why?
 - Foreign body irritates bronchial lining (foreign body aspiration)
 - Instruct patient to spit it out when they vomit a little in the mouth
 - ◆ Represents sequestration- looking for small amount of vomit with mixed meals
 - ◆ Only very few things that will cause vomit with mixed meals
 - ◇ Obstruction
 - ★◇ Sequestration (in this case)
 - Treatment
 - Lavage it thoroughly and then stitch it up
- *Traction Diverticulum*
 - Tend to have Lymphadenopathy in hilar region of the lung (well invested area with lymph nodes)
 - End up having the lymph change fibrotically attached to outer layer of esophagus
 - This tissue will shrink over time tearing the outer annular fibers of the esophagus essentially creating a defect
 - End up herniating mucosal defect through acquired injury because of traction injury where fibrotic tissue goes back and forth
 - Do not get check valve mechanics
 - More slowly progressive
 - NO clinical significance
 - Perihilar region, middle 1/3 of airway, diaphragm
- *Pulsion Diverticulum*
 - Right at the diaphragm
 - No check valve mechanics
 - Distance from airway
 - Freely empties, freely fills
 - Patients appear to never have normal growth of circumferential muscle bands in esophagus
 - Non-reinforced mucosal layer

- Cases of pyloric stenosis which lead to obstruction and very large collection of material in the stomach
 - Can hold up to 15 L!
 - This can of pressure load in stomach doesn't allow the esophagus to have a chance- it tries though and can lead to tearing
- Instructions: Laying on right side will cause normal emptying into stomach
- Clinically benign- asymptomatic
- Found incidentally because looking for something else
- Will see a mass on spiral CT

GERD- Gastroesophageal Reflux Disease

- Reflux
 - Dysfunction of anti-reflux mechanisms
- Caustic material
 - Acid, pepsin, bile, pancreatic enzymes
 - Two valve problem if bile also excreted
 - LES and Pyloric have to malfunction
- Sufficient Duration of Contact
 - Inadequate clearance mechanism
- Signs and Symptoms
 - ★○ #1 Sign of Symptom of GERD is Retrosternal Burning
 - 50-60%- rub up and down on sternum with a couple of fingertips
 - Will also lead to hoarseness and change in timber of patient's voice - not quite the "smoker's" voice but head that way
 - Little coughs trying to clear their throats
 - Least common presenting sign/symptom is fixed dysphagia
- Medical Management
 - Histamine 2 Blockers - ability to raise histamine inhibited causing inhibition of release of gastrin inhibiting pepsin and HCL release by chief and parietal cells
 - Hypochlorhydric (decreased HCL)
- We can adjust!
- Heartburn in Pregnancy: Pregnant female experiences GERD because baby pushes up- in addition to increase in hormones (progesterone and estrogen specifically)
 - Frequency - 30-50%
 - Mechanisms
 - Hormonal: increased estrogen and progesterone (relaxes soft tissue)--> LES dysfunction
 - Mechanical: Enlarging uterus --> increased intra-abdominal pressure
 - Common Therapies
 - Antacids
 - Sucralfate
 - H2-blockers
- People with tight fitted clothing will have more GERD symptoms and this is something we can help them change
- Alter diet- eliminate secretogogic foods
 - Chocolate, peppermint, proteins generally, fatty foods
 - Tobacco, Alcohol, certain drugs
 - Sweeter drink post meal shut up satiety allowing more satisfaction
 - Same idea of mints
- If we have them in a lumbosacral support we have to warn them that they may have symptoms--essentially tight fitting clothing
- Not a good idea to have patient be first patient after lunch- LES is weak
- Testing
 - We have plenty of false negative tests when investigating for GERD
 - Barium swallow is one of the better exams
 - Patient upright, pre-scan with fluoroscope, watch them swallow, give fizzies then swallow again and look
 - If we don't see any contrast in LED then we say they're competent in the upright position
 - If you do it's called Grossly incompetent
 - Tilt patient back 45 degrees
 - Incompetent
 - Patient laying back- Demonstrating Normal competence
 - Minimally Incompetent
 - Head lower than body- *Tredelenberg test*
 - Normal for some leakage
 - Flexible Fiberoptics
 - Looking for evidence of bubbles rising up out of the stomach
 - Not unusual to find evidence of ulcer but still have a negative test
- Indications for Ambulatory esophageal pH monitoring
 - Patients with normal endoscopy and
 - Typical GERD symptoms unresponsive to anti-reflux therapy

Overwhelm Mucosal Resistance

Normal Resting Pressures

Intra-thoracic = -5 mm Hg

Intra-abdominal = +5 mm Hg

Lower esophageal sphincter (LES) = +25 mm Hg

Net 15 mm of Hg pressure

Should be able to induce a little bit of leakage by pushing on stomach

- Atypical chest pain
 - Extra-esophageal disorders possibly related to GERD (asthma, chronic cough, chronic hoarseness, posterior laryngitis, globus)
- Patients with endoscopic esophagitis unresponsive to anti-reflux therapy
- Patients considered for anti-reflux surgery
- Lifestyle Modifications
 - Elevate head of bed
 - Lose excess weight
 - Eliminate
 - Tobacco
 - Alcohol
 - Bedtime snacks
 - Certain drugs
 - Fatty foods
 - Chocolate
 - Peppermint
 - Others

Peptic Esophagitis- Symptoms and Signs

- Sour taste
- Difficult or painful swallowing; choking
- Chest pain (r/o cardiac)
- Back pain
- Heartburn
- Epigastric pain or distress
- Nausea and vomiting
- Anemia (blood loss)

★ *Important to ask if cardiogenic or non-cardiogenic pain*

Chalasia

- Opening
- Vs. Achalasia (no opening)
- Uniformly true for youngsters
- Grossly incompetent LES

Hamartoma = normal tissue but a lot of excess

Spoke Wheel appearance (on contrast study)

- Almost always associated with a benign ulcer
- Benign ulceration and rugal folds radiate out from center point
- Separates tumor from ulcers
 - Tumor can acquire an ulcer

Hiatal Hernia and Reflux

- LES- pressure often low
- Gastric pouch- intrathoracic reservoir
- Diaphragm - no esophageal pinch
- Can appear as the Double Bubble Sign on Xray

Barrett's Esophagus

- Columnar epithelium replaces squamous epithelium in the distal esophagus
- Annual incidence of adenocarcinoma due to Barrett's esophagus
 - 800 cancers per 100,000
 - 1 cancer per 125 patients

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Achalasia

- No opening
- Neurologically based phenomenon (neurologic death event) where lower esophageal sphincter acquires abnormally high tone- compression values of 60 mm of Hg isn't uncommon (15 mm of Hg is common) becoming overall competent
- Can be autonomic imbalance
 - Too much sympathetic or too little asympathetic
- Loss of ganglion cells in wall of esophagus itself
- Degeneration of dorsal motor nucleus in brainstem
- Degeneration of vagal fibers
- CCK Octapeptide Test
 - CCK is recognized as a fairly strong trigger on nerve bodies which have an inhibitory effect on LES
 - If vagal nerve death then CCK does not stimulate the nerve body to relax the LES and instead it stimulates LES contraction

Hummingbird's beak/Bird's beak appearance- contrast term allowing us to see narrowing of channel on imaging

Clinical presentations

- Dysphagia for solids (90ish %)
- Dysphagia for liquids (85ish %)
- Difficulty belching (95ish %)
- Chest pain (40ish %)
- Nocturnal regurgitation (40ish %)
 - Some of these get aspiration pneumonitis (multiple mixed meals)
- Aspiration (15ish %)
- Loss of heartburn (15ish%)

A GERD patient who gets achalasia may lose their heartburn

A distended esophagus can hold 2-3 liters

Treatments

- Drugs
 - Nitrates
 - Responsive to dose responsive relationship
 - Takes an increasingly higher dose to have the same therapeutic results

- Ca++ channel blockers
- Botulinum toxin injection
- Pneumatic dilation (early dilation)
 - Literally tears apart the LES
- Myotomy (radial myotomy)
- Complete dilation

Esophageal Varices

- Bag of Worms appearance (X-ray term)
- Curvilinear protrusions into the lumen of the esophagus that contain venous blood (portal system lacking in valves)
- Problem isn't with the esophagus
- Arises out of a liver injury profile - portal hypertension
- Drinking alcoholics are the number one person to exhibit this

Axial/Sliding Hernia: only content in esophageal hiatus is stomach- gastroesophageal (GE) junction is in the thoracic cage - retrosternal burning is major complaint- double bubble sign

Mallory-Weiss syndrome

- Vertical lacerations found near to GE junction
- Severe retching and/or vomiting and hiccups that last for days and weeks
- They bleed (vomit up blood)
- Staph food poisoning can cause violent retching (even after stomach is empty)
 - Some strains of staph produce a neurotoxin to stimulate this
- Drinking alcoholics are at top of this list
- Gastritis and Gastroenteritis can trigger this

Chalasia

- Commonly seen in newborns
- If premature delivery, more likely
- This is a management thing- we don't really want to "do" anything about this
 - Have the kid propped up if not held when eat
 - Smaller meals interspersed with burping
- Self-limiting and a management problem only

Curling Phenomena

- Contrast based radiologic sign
- Patient able to take bolus of contrast in mouth but don't see contraction of upper 1/3 of the esophagus
- *Presbyesophagus* is an aged related phenomenon
 - Uncoordination between primary and secondary peristaltic waves
 - Complete unlinking between primary and secondary peristaltic waves later on
 - Finally an absent of the peristaltic waves
 - Middle and end of the disease will show the curling phenomenon
 - Patient needs to cut the food in smaller pieces and wash it down well
- Also called corkscrew appearance

Diffuse esophageal spasm

- Acute vagal stimulation
- Squeezing in the chest
- Similar profile to MI infarction
- Don't know how to document because difficulty tracking it

Diabetic neuropathy of the esophagus

- Similar to diffuse esophageal spasm
- Same management
- Hx of juvenile onset diabetes is the major difference
- Documentation of nerve injury (not really found in presbyesophagus patients)

Esophageal atresia

- Underdevelopment/maldevelopment of the esophagus structure
- VOGTs classification system
 - Type I: complete absence of the esophagus
 - Type II: system is largely in place but no communication (blockage or plug- membrane or plug of tissue)- surgical management case - after the surgery the kid may grow up with a hiatal hernia but overall they'll do well
 - Type III: fistula (abnormal communication)
 - Three types- only talk about 3b

- 3b- 85-90% of esophageal atresia
 - ◆ Food gets pushed into lung tissue
 - ◆ Baby acquires aspiration pneumonia - mothers' milk, formula, water, etc
 - ◆ Tends to go down right primary bronchi more than the left
 - ◆ Merconium aspiration is likely though to be first cause

Duplication of the Esophagus (aka. Duplex esophagus)

- Usually there is a well-developed esophagus and an underdeveloped esophagus that runs parallel
- Problem is usually in the poorly developed one
- Most complete form is where the esophagus is divided into the tubes and there will be two openings into the stomach
- You can have it where
 - Starts as 2 goes to 1
 - Starts as 1 goes to 2
- Typical fix is to prevent content from entering underdeveloped part of esophagus rather than the complete removal

Duplication of the Stomach

We don't usually have complete independent sacs that we'd recognize as the stomach
 Tend to see bigger sac divided up into chambers- wall separating one side from another
 More likely to be a wall producing partitions in the stomach- not typical to have an additional organ
 Perpetually feel full

Dextroposition

- Stomach is on Right side and duodenal bulb is on the left side
- Some patients have a flip of their stomach
- Failure to complete rotations causing coiling of small and large intestine
- Part of a complete transposition called *Citus Inversus* (everything flips)
- More isolated the flip the more likely there will be complications (more complete the flip, less complications)

Pyloric Stenosis

- Supine contrast study typically used
- Congenital
 - Usually in utero
 - Get persistently strong neurological signals (increased sympathetics) to pyloric valve to contract leading to hyperplasia of the pyloric valve
 - Projectile vomiting (fountain of vomit)
 - Work a balloon- force it through pyloric valve, blow it up tearing the muscle apart
- Acquired
 - Most common cause is peptic ulcer disease
 - Ulcers are located immediately next to pyloric valve or in the pyloric channel itself
 - Healing is accomplished by fibrotic repair
 - Adhesion retraction is how stenosis occurs

3/24/09

Common places for air fluid levels in GI (all below the diaphragm)

1. Stomach
2. Cecum
3. Right before hepatic flexure
4. Right after splenic flexure

Hiatal Hernia

- Abdominal content that has passed back up through the esophageal hiatus
- "figure 8" xray sign (gas bubble above the diaphragm-there is only supposed to be esophagus stomach junction and another gas bubble below diaphragm)
- Burning pain is the most frequent sign- retrosternal
- Double bubble sign
- *Sliding hernia = axial hernia*
 - Most common
 - May or may not complain of dysphagia
- *Paraesophageal hernia = rolling hiatal hernia*: a portion of the stomach is rolled up next to the esophagus
 - GE junction AND a bit of the stomach are in the hiatus
 - More likely to have dysphagia
 - A lot more tissue above the hiatus (than the sliding hernia)
 - Mechanical events tend to produce more of the discomfort

Three types of hernias

1. Sliding/Axial
2. Paraesophageal/Rolling
3. Intrathoracic Stomach

- Retrosternal burning b/c of reflux most common complaint
- Paraesophageal is most likely cause of dysphagia
- Most serious complication is volvulus seen in intrathoracic stomach

- In plain films we may see a Negative density over heart, air fluid level above the heart but doesn't distinguish itself from sliding/axial- sliding/axial should be the first differential because the most common
- High pressure zone is below the diaphragm
- If ever gas collection overlying the heart (on xray) think hiatal hernia first
- If hear bowel sounds above the diaphragm, then likely hiatal hernia
- Most serious complication of hiatal hernia is *volvulus* (or twisting or folding of the stomach on itself)
 - Occurs because suspensory ligaments fail to hold stomach in place
 - Compromises blood supply (increased venous pressure = reduced blood flow)
 - Ischemic GI disease
- *Intrathoracic Stomach*
 - Acquired and congenital forms
 - Volvulus can occur- surgical emergency

Intusseption = telescoping/invagination

Diverticulum = outpouching, Herniation of mucosal tissue

Diaphragmatic Hernias - posterior diaphragm defect attachment to body wall

- Bochdalek hernia
 - In the back (laying on top of the psoas and QL)
- Morgagni hernia
 - Retrosternal

Umbilical Hernia

- In neonates linea alba hasn't sealed closed and there is a thin layer of visceral tissue holding contents in easy to breach
- Adults acquire because had surgeries that dissected linea alba and didn't get a good repair
 - Sometimes had a bad fit of pneumonia and coughing essentially disrupts linea alba

Conservative Management for Hernias

- Drug of choice to address retrosternal burning is an H2 blocker reducing acid producing capacity
- Is hiatal hernia reducible, non-reducible (incarcerated) or strangulated
 - We're worried about the strangulated version
 - Reducible and non-reducible will produce bowel sounds, strangulated will not
- Heel drop
 - 2-3 8 oz of water (benign weight in body) then rise up on toes and then drop back on heels
- Manual reduction
 - Raise patient on table to where their waist is up making stomach dependent
 - Have take big breath in flattening diaphragm
 - Force way back into left aspect of abdomen and then push and traction down
- Surgery option teases out two bundles of diaphragm, criss crosses them and then stitches them down

Three things to collect barium

1. Gastric Diverticulum
 - Size of opening tends to be big
 - Extends outside lumen and wall thickness
 - Incidental finding of outpockets
 - Could look like an ulcer in the early days- diverticulum doesn't hurt when you push on it however
 - *Stretched stomach can hold 13 liters*
 - Overly dilated stomach is a cause of rupture
 - As the stomach stretches the muscle layer thins and therefore it no longer has potent contractions
2. Peptic Ulcer disease
3. Tumor form of ulceration
 - a. Crater occurs because leading fast growing edge demands all the blood causing tumor to outgrow blood supply causing tumor to die, crater out and collect barium



Gastritis - inflammation of gastric mucosa

- Causes
 - Chronic aspirin use is huge source of gastritis
 - Excess alcohol intake - secretagogic secretion of gastric juices
 - Viral gastritis
 - Can occur secondary to chemo therapy
 - Uremia
 - Complications of a renal process
 - Stress
 - Cytomegalovirus and helicobacter pylori
 - Will attack the gastric wall
 - H. pylori is the case 15% of the time

- Intervention
 - If source is from taking an adult aspirin is the cause talk to your patient about taking a baby aspirin
 - Alcohol intake
 - Don't drink it on an empty stomach, this may take care of it
 - Drink less
 - With food poisonin- just don't eat the food a gain, store it better
 - Chemo- easy to digest foods might help some but they are probably on an H2 blocker to help already
- Diagnosis
 - If it's the first time
 - Listen, make best guess and stand by
 - Endoscopy + biopsy is the gold standard
 - Only 3-5% have this done
 - The only endoscopic finding in acute gastritis will be an area of a little redness (erythema)
 - ◻ The next change after erythema is erosion
 - ★◦ No contrast based test to ID
- Acute vs. Chronic Gastritis
 - Histologic change seen on an endoscopic exam is the only way to differentiate- seen in chronic gastritis
 - No histologic change seen in acute gastritis patients
 - Worse cases of chronic gastritis is when you have fibrotic repair losing rugal folds and have bland surface
 - "Leather bottle stomach"
 - Demonstrating hypochloridria
 - Signs and symptoms are the same as acute just less intense and more tissue involved
 - Inflammatory bowel disease (Chron's disease) can create this
 - Signs and Symptoms
 - Tendency to continue the adding of damages tissue
 - Look at drugs they are taking
 - IBD- chron's may produce

Menetrier's disease aka hypertrophic gastritis

- Investment of abnormal tissue that displaces gastric, parietal and chief cells
 - Hyposecretory
- Lack of sufficient HCL and pepsin so patient can't properly prepare proteins for uptake into SI
- Can't take protein out of SI to put in body
- Decrease osmotic pressure so can't keep fluid in vascular tree
 - Puffy weight loser
 - Large chain proteins found in the fecal material
- There isn't a great fix but taking simple proteins and protein drink is good
- Diagnosis
 - Contrast Exam: See thickened rugal folds
- 10% of these patients will undergo malignant degeneration to adenocarcinoma r

Zollinger-Ellison Syndrome aka Gastrinoma

- Can stimulate thickening of rugal folds due to hypertrophy
 - On contrast exam- looks similar to menetrier's
- Plenty of gastric output
 - Hypersecretory
- Lots of region of weeping blood because prolonged secretion of HCL can overwhelm mucosa tissue
- Clinical triad consisting of
 - Gastric acid hypersecretion
 - Severe peptic ulcer disease
 - Non-beta islet cell tumors of the pancreas
- The tumors produce gastrin (G17 and G34); referred to as "gastrinomas"
- Tumors localized usually to head of pancreas, duodenal wall or regional lymph nodes
- About one-half of gastrinomas are multiple and two-thirds malignant
- About one-fourth have multiple endocrine neoplasia syndrome (MEN I) - tumors of parathyroid, pituitary and pancreatic islets
- Fasting and stimulated plasma gastrin are sensitive and specific in the dx of zollinger-ellison syndrome
 - Normal serum gastrin is < 150
 - ZE serum gastrin 150-100,000
- Treatment
 - If "isolated" duodenal wall tumor is present on CT and/or visceral angiography, surgical resection followed by measurement of gastric acid secretion
 - If no evidence of tumor, or evidence of metastatic tumor, H+/K+ ATPase inhibitor or H2-receptor antagonist therapy in a dose determined individually to suppress fasting acid output to <10 mmol/h. Drug efficacy checked at 2 mo intervals
 - Total gastrectomy is generally obsolete

The lowest level of damage to the mucosa is erythema
Erosion is limited defect in mucosa

Ulcer- extended through mucosal layer into submucosal tissue
 Perforation- all the way through

Acid Output Testing

- Key way we recover normal secretory GI problems, hypersecretory GI problems, hyposecretory GI problems
- BAO (base/basal acid output)
 - Pre-stimulus assessment
 - The stomach at rest
 - Histamine is stimulus which is a strong stimulator of gastrin (produces maximal elevation of gastrin)
- PAO (peak acid output)

	Hyper	Normal	Hypo-Early	Hypo-Late
BAO	Increased	E/N	E/N	Low
Histamine challenge				
PAO	E/N	E/N	Increased-L	Very Low
	BAO < or + to PAO yet BAO is high, PAO is E/N	BAO < PAO with each value falling with the normal range	PAO < PAO; PAO is Low	BAO < or = to PAO; BAO- Low

Peptic Ulcer

- Ulcers tend to occur near mucosal junctions
- Ulcers may be caused by non-acid/peptic disorders
 - Esophagus
 - Herpes simplex
 - Tablet induced
 - ◻ Tetracycline
 - ◻ KCl
 - ◻ Other
 - Cytomegalovirus
- Duodenal ulcer can be diagnosed by barium contrast x-ray or by endoscopy

3/30/09 Abe's notes

Peptic Ulcers con...

- Cause is not rooted in hypersecretory status
- Originally called peptic ulcer because it was originally thought that pepsin was the causative agent
- Peptic ulcer is a common disease
 - ◻ The annual incidence of active ulcer in the U.S. is about 1.8% - 500,000 new cases per year
- Rather the cause is rooted in H. Pylori (campylobacter pylori) - it is an infection
 - ◻ H pylori is able to survive in the stomach because it secretes enzymes that neutralize the acid
- Zantac (H2 blocker) decreases irritating acid secretion, but patient may actually end up with more and larger ulcers
- Peptic ulcer disease remains one of the most prevalent and costly GI diseases
- Prevalence
 - ◻ Patients seen by physician - 3-4 million/yr
 - ◻ Patients self-medicating -3-4 million/pear
- Total patinet visits to physicains - 12-14 million
- Hospitalizations - >400,000
 - ◻ Total days - ~4 million
 - ◻ Deaths - ~9,000
 - ◻ Operations >130,000
- Total costs (indirect plus direct) - ~7billion
- Peptic ulcers are caused by increased aggressive factors and/or decreased defensive factors which both will cause mucosal damage and then ulcers
- Gastroduodenal mucosal integrity is determined by protective (defensive) and damaging (aggressive) factors

★ Relapse rate: 80% got signs or sx back after going of the drug (H2 blockers)

Relapse rate for antibiotic treatment: less than 10%

**numbers for 198*

Protective	Damaging
HCO ₃	H ⁺
Mucus	Pepsins, Smoking
Blood flow	Ethanol, Bile acids
Growth factors	Ischemia, NSAIDS
Cell renewal , PGs	Hypoxia, H. pylori

- Ingestion of NSAIDS daily is a major factor in ulcer pathogenesis, complications and death
 - ◻ 3x10⁶ people in the US take NSAIDS daily
 - ◻ About 1 in 10 pt taking NSAIDS daily have an acute ulcer
 - ◻ 2-4% of NSAIDS users have GI complications each year

- >3000 deaths/yr and >25,000 hospitalizations/yr are attributable to NSAID-induced GI complications
- Compared to the general populations NSAIDs increase the risk of GI complications ~2 to 10 fold
- NSAIDs cause both acute and chronic gastroduodenal injury
 - All NSAIDs produce mucosal damage
 - Ulcer risk is dose-related
 - Acute mucosal response does not predict subsequent ulcer risk
 - ◆ Don't acquire and resistance over time
- Gastric acid secretory rates are altered in patients with upper gastrointestinal diseases
 - Normal
 - ◆ Basal 1-8
 - ◆ Peak pentagastrin 6-40
 - Increased with
 - ◆ Duodenal ulcer
 - ◇ 1-15 basal
 - ◇ 12-60 peak pentagastrin
 - ◆ Gastrinoma
 - ◇ 5-80 basal
 - ◇ 20-110 peak pentagastrin
 - Duodenal ulcer is a disease of multiple etiologies
 - ◆ Gastric acid and pepsin secretion are required (thought to be the pain generators and H. Pylori is the problem)
 - ◆ Antral gastritis is present and almost universally associated with H. pylori
 - ◆ Proximal duodenal mucosal bicarbonate secretion is frequently (~70%) impaired
 - ◆ Nocturnal acid secretion and duodenal acid load are frequently increased
- **Helicobacter pylori** has high urease activity
 - High molecular weight
 - pI (isoelectric point) 5.9
 - ★ □ 20 times more active than proteus vulgaris
 - High affinity for urea (Km= 0.8mM)
 - Urease reaction is the basis for the diagnostic ¹⁴C/¹²C breath tests
 - Presence of ammonia will raise local pH
- Tests of H. Pylori
 - Endoscopic biopsy test
 - ◆ Culture
 - ◆ Histologic exam
 - ◇ 95% sensitivity and 98% specificity
 - ◆ Rapid urease test
 - ◇ 90% sensitivity and 95-100% specificity
 - Non-onvasive tests
 - ◆ Serologic - igG antibodies to H. pylori
 - ◇ 80-95% sensitivity and specificity
 - ◆ Urea breath test - ¹³C or ¹⁴C urea --> CO₂
 - ◇ 95% sensitivity and specificity
- Genetic factors influence ulcer incidence
 - Identical twins ~5X
 - First degree relatives ~3 X
- Certain diseases are associated with an increased prevalence of ulcer

Disease	Risk ratio
Chronic pulmonary disease	3-5X
Hepatic cirrhosis	5-8X
Chronic renal failure (azotemia)	1.5 -3X

- ★ NSAIDS deaths alone according to AMA = 125,000/year
 - Smokers have: a higher incidence of ulcers
 - More ulcer recurrences
 - More frequent complications
 - Greater ulcer-related mortality
 - A portion of this effect can be attributed to smoking-induced chronic pulmonary disease
 - Cigarette smoking also decreases pancreatic bicarbonate production
 - There are a number of myths surrounding the causes and treatment of ulcer disease
 - Spicy foods
 - ◆ The more it delays gastric outlet the more likely it allows sx to be related to it.
 - Alcohol

- Psychological stress
- Ulcer is an executive's disease
- A bland diet heals ulcers
 - ◆ The definition of a bland diet is something that is unclear
 - ◆ The easier that the food can be moved from the stomach to the SI the less problems they will have
- The diagnosis of ulcer disease by symptoms alone is imprecise

Trico-bisor

- Bisor is a collection of solid substance in the stomach
- A tricobisor is a hairball

3/30/09 Hanna's Notes

Peptic Ulcer Disease Remains One of the most Prevalent and Costly Gastrointestinal Disease

Prevalence	# per year
patients seen by physician	3-4 million
patients self-medicating	3-4 million
Total patient visits to physicians	12-14 million
Hospitalizations	>400,000
total days	~4 million
deaths	~9,000
operations	>130,000
Total costs (indirect plus direct)	~7 billion

★ 1985 data

★ **Peptic Ulcer Disease is primarily due to an infection of *helicobacter pylori* (rather than a hyper-secretion problem)**

- 80% of patients who take H2 blockers get worse once they stop taking them since they don't have hyper-secretion problem as the cause of peptic ulcer disease
- Number of new ulcers increases while on the H2 blockers but the patient isn't as irritated because there isn't as much acid irritating the ulcers.
- Eventually they decide that the patient is "cured" and take him/her off the H2 blockers... then you see big problems!

Peptic Ulcer is a common disease

- The annual incidence of active ulcer (GU & DU) in the US is about 1/8% - 500,000 new cases per year
- In addition, there are approximately 4 million ulcer recurrences per year

★ *Actually, the number is much less now that we are treating as bacteria*

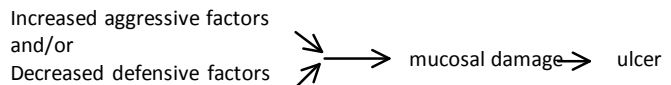
★ **90% of patients who takes antibiotics for peptic ulcer disease get better (including when they stop taking it)**

- ★ *H. Pylori burrows through the mucosal lining*
- ★ *Some patients really do hyper-secrete Zolinger ellison*

Hospitalizations for bleeding upper gastrointestinal lesions are increasing along with increased NSAID use

- Prescriptions for NSAIDs tripled from 1975 to 1985
- *Increased use of duodenal and gastric erosions secondary to increased use of NSAIDs*

Peptic Ulcers are caused by increased aggressive factors and/or decreased defensive factors



Gastroduodenal Mucosal Integrity is Determined by Protective ("defensive") and damaging ("aggressive") factors

Protective	Damaging
HCO ₃ ⁻	H ⁺ NSAIDs
Mucus	Pepsins Hypoxia
Blood flow	Smoking H pylori
Growth factors	Ethanol
Cell renewal	Bile acids
PGs	Ischemia

The ingestion of non-steroidal anti-inflammatory drugs (NSAIDs) daily is a major factor in ulcer pathogenesis, complications and death

- ~3x10⁶ people in the US take NSAIDs daily
- About 1 in 10 patients taking NSAIDs daily have an acute ulcer
- 2-4% of NSAID users have GI complications each year
- >3000 deaths/yr and >25,000 hospitalizations/yr are attributable to NSAID-induced GI complications
- Compared to the general population NSAIDs increase the risk of GI complications ~2 to 10 fold

NSAIDs are associated with an increased incidence of bleeding ulcers

	Risk Ratio
Gastric ulcers	↑ 10-20x
Duodenal ulcers	↑ 5-15x

NSAID ingestion causes both acute and chronic gastroduodenal injury

	Gastroduodenal lesions	Frequency
Acute (1-2 weeks)	Mucosal erythema Subepithelial hemorrhages Erosions Incr fecal....	

- All NSAIDs produce mucosal damage
- Ulcer risk is dose-related
- Acute mucosal response does not predict subsequent ulcer risk

Gastric Acid secretory rates are altered in patients with upper GI diseases

Normal acid output

Basal = 1-8 mmol/h
Peak pentagastrin = 6-40 mmol/h

Decreased with:

Pernicious anemia
Gastric atrophy
Gastric ulcer
Gastric cancer

Increased with:

Duodenal ulcer
Gastrinoma

Duodenal Ulcer is a disease of multiple etiologies

- Gastric acid and pepsin secretion are required
 - *Now we know its H. pylori*
- Antral gastritis is present and almost universally associated with H. pylori
- Proximal duodenal mucosal bicarbonate secretion is frequently (~70%) impaired
 - *Seen especially in smokers*
- Nocturnal acid secretion and duodenal acid load are frequently increased

★ **Don't forget mid-thoracics which contain an on/off for gastric secretions**

Mean parietal cell number is increased in DU but not in GU

- *Duodenal ulcer patients do tend to have higher acid outputs, and have greater density of parietal cells - true hypersecretory patients*
 - *Still may not need anti-secretory medication*
 - *May just need to quit smoking*
 - *Bicarb in stomach increases pH and stomach drives it down by acid production*
 - *If person continues to smoke, this process continues/gets worse*

Helicobacter Pylori has high urease activity

- High molecular weight
- pI (isoelectric point) 5.9
- 20 times more activity than *proteus vulgaris*
- High affinity for urea
- Urease reaction is the bases for the diagnostic ¹⁴C/¹³C breath tests
 - *Easiest test for H. pylori*
- The presence of ammonia raises local pH

Helicobacter pylori infection can be detected by various methods

	Sensitivity	Specificity
Endoscopic biopsy tests		
Culture	60-90%	100%
Histologic examination	95%	98%
Rapid urease test	90%	95-100%

Non-invasive tests

Serologic
(IgG antibodies to H. pylori)
Urea breath test
(¹⁴C or ¹³C urea --> *CO₂)

Certain diseases are associated with an increased prevalence of ulcer

Diseases	Risk ratio
Chronic pulmonary disease <i>Hypoxia (CO₂ retainers)</i>	3-5x
Hepatic cirrhosis <i>Portal system HTN (venous engorgement) (its another form of hypoxia)</i>	5-8x
Chronic renal failure <i>Azotemia - destabilizes membranes</i>	1.5-3x

Genetic Factors Influence Ulcer Incidence

Identical twins ~5x greater risk
First degree relatives ~3x greater risk

Cigarette Smoking is Strongly Associated with ulcer disease

- Smokers have

- A higher incidence of ulcers
- More ulcer recurrences
- More frequent complications
- Greater ulcer-related mortality
- A portion of this effect can be attributed to smoking-induced chronic pulmonary disease
- Cigarette smoking also decreases pancreatic bicarbonate production

There are a number of myths surrounding the causes and treatment of ulcer disease

- Spicy foods
 - Alcohol
 - Psychological stress
 - Ulcer is an executive's disease
 - *Related to psychological stress he guesses*
 - A bland diet heals ulcers
 - *No one even knows what a bland diet really is*
 - *Foods that delay gastric emptying will hurt, foods that increase gastric emptying are helpful*
 - *Less protein and fat, more carbohydrates are needed*
- Controversial - may cause ulcers or aggravate ulcers
Spices may ride in on foods that aggravate ulcers - may not be the spices themselves

★ **The diagnosis of ulcer disease by symptoms alone is imprecise**

★ *Don't drink milk/buttermilk to coat stomach - old wives tale*

★ **Remember:**

*Benign ulcer is barium contained within the wall
Diverticulum is barium outside the wall - herniation (wide mouth, asymptomatic)
Cancerous ulcer would be pool of barium suspended in the lumen*

Diff Dx Early feeling of fullness

- Minetrere
- Zollinger ellison
- Dilated stomach?
- Bezoars
 - Collection of insolubles in the stomach
 - Like hair, glue (phyto bezoar), clay

3/31/09

Gastric Neoplasm

1. Metastasis
 - a. Number one cancer to spread to the stomach is melanoma
 - i. Great ability and frequency of spreading to the abdomen
2. Primary neoplasm
 - a. Tend to find due to looking for something else
 - b. May have dysphagia depending on where the neoplasm is located
 - c. Can have neovascularity
 - d. **Benign**
 - i. Adenoma-classic polypoid disease
 - 1) Tend to move into the lumen
 - 2) Two forms
 - a) *Pedunculated polyp*- strand or stalk attaching to luminal wall
 - b) *Sessile version*- plateau broad based lesion
 - Bigger they get the less likely they are benign and more likely to convert to adenocarcinoma
 - 40% of cases undergo malignant degeneration into adenocarcinomas
 - ii. Leiomyoma
 - 1) Develop in the wall and tend to infiltrate the stomach wall
 - 2) Wall stiffness
 - 3) Dampening of the peristaltic wave seen (loss of movement)
 - 4) 40% of cases also undergo malignant degeneration
 - iii. Lipoma
 - 1) 5% of benign neoplasms
 - iv. Other
 - 1) 15%
 - e. **Malignant**
 - i. Adenocarcinoma
 - 1) Account for 90-95% of all malignant neoplasms to the stomach
 - ii. Lymphoma- 3%
 - iii. Leiomyosarcoma- 2%

- iv. Malignant GI Cancers are currently the 2nd leading cause of death
 - v. 3:2- Male: Female ratio over 50
 - vi. Cigarette smoking is a player
 - vii. Top 3 countries with gastric cancers- Japan, Iceland and Chili
 - 1) Genetic influence not a factor
 - 2) Similarity in food- specifically fish and pollution
 - viii. Things that contribute to gastric neoplasm
 - 1) Diet
 - a) Over eating of fats
 - b) Nitrites
 - c) Smoked meats
 - 2) Low socioeconomic factor
 - a) Food choices more likely to be poor
 - 3) Occupation
 - 4) Tends to run in families
 - 5) Achlorhydria
 - 6) Coal-fired power plants, coal mining and coal as it relates to steel production
 - 7) Smoke (smokers, fireman or smoked food)
- JMPT article showed that Spinal manipulation as a single intervention gave remission 10 days sooner than traditional tx*