GASTROINTESTINAL PATHOPHYSIOLOGY I

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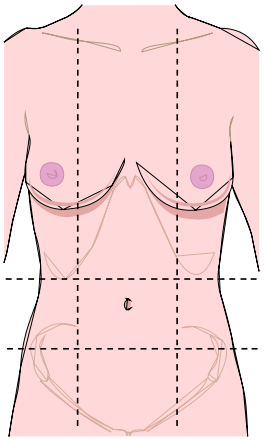
ABDOMINAL PAIN



- Common problem
  - 5%-10% ER visits
  - Top 10-15 problems seen in ED and outpatient settings
- Multiple etiologies - some are life-threatening
- Site, character and onset of pain is important in establishing etiology
- Rapidity of onset is important in establishing etiology
- Site described by one of two systems
  - 4 quadrants: upper right, upper left, lower right, lower left
  - 9 regions to describe location

Hypochondrial - Epigastric - Hypochondrial
Lumbar - Umbilical - Lumbar
Iliac - Hypogastrium - Iliac

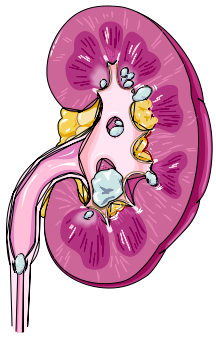
LIFE THREATENING CAUSES OF ACUTE ABDOMINAL PAIN

SOURCE	TYPE PAIN	PHYSICAL CHANGES	INITIAL ACTION
Abdominal aortic aneurysm	Referred - Mid to low back - Mid to upper abdominal pain	- Pulsatile mid to upper abdominal mass with AP and lateral movement - Hyper then hypotensive -> shocky - Lower extremity ischemia (“blue toes”)	- O2 high flow - Rapid IV volume and BP support - NPO - Rapid transport to imaging w surgical support - Admit; Type and X-match
Bowel Obstruction	Visceral - Campy w partial - Constant w complete - Diffuse, ill defined	- Inability to pass gas - Belly distended; tympanic - Abdominal xray shows large dilated loops of small or large bowel - + or - air fluid level possible	- IV access/ NPO - Rapid surgical consult - Pain relief per consult w surgeon - Transport - admit; Type- X-match
Ruptured spleen	Somatic -Instantaneous to rapid onset -LUQ or “shoulder strap” areas - Hx of abdominal trauma	- Hyper then hypotensive -> shocky - May be syncopal	- Rapid IV; volume support - NPO - Rapid surgical consult; - Transport/admit’; Type-Xmatch
Ruptured ectopic pregnancy	Somatic or referred - Unilateral or bilateral LQ in woman of child-bearing age (do HCG)	- May be syncopal - Localized RLQ or LLQ abdominal pain - Hypotensive/shock - Possible vaginal bleed - Assess first day last menses	- IV access - NPO - Quantitative BHCG - Consult OB-Gyn re pain relief - Transport- Admit; Rh-Type- X match

ACUTE ABDOMINAL PAIN BY RAPIDITY OF ONSET

ABRUPT ONSET (INSTANTANEOUS)

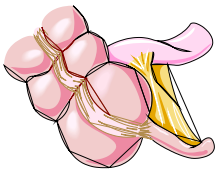
GASTROINTESTINAL	EXTRA-GASTROINTESTINAL
Perforated ulcer Ruptured Abscess or hematoma Intestinal infarct	Ruptured or dissecting aneurysm Ruptured ectopic pregnancy Pneumothorax Myocardial infarction Pulmonary infarction



Renal calculi with hydronephrosis

RAPID ONSET (MINUTES)

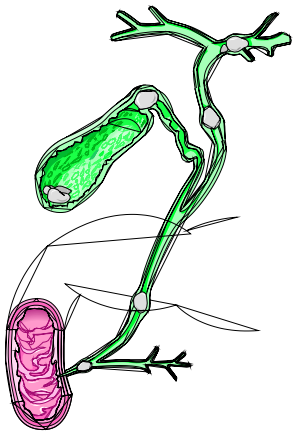
GASTROINTESTINAL	EXTRA-GASTROINTESTINAL
Perforated viscus Strangulated viscus Volvulus Pancreatitis Mesenteric infarct Diverticulitis Penetrating peptic ulcer High intestinal obstruction Appendicitis * * gradual onset more common	Urethral colic Renal colic Ectopic pregnancy



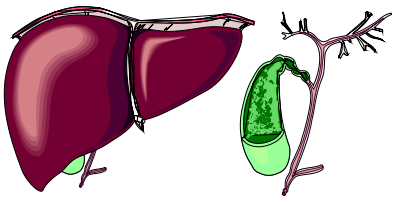
Appendix

GRADUAL ONSET (HOURS)

GASTROINTESTINAL		EXTRA-GASTROINTESTINAL
Appendicitis	Crohn's Disease	0
Strangulated hernia	Ulcerative colitis	Cystitis
Low small intestinal obstruction	Abscess	Pyelitis
Cholecystitis	Gastritis	Salpingitis
Pancreatitis	Intestinal Infarct	Prostatitis
Mesenteric lymphadenitis	Mesenteric Cyst	Threatened abortion
Meckel's diverticulitis	Peptic ulcer	Urinary retention
Colonic diverticulitis		Pneumonitis



Cholelithiasis
Gall Bladder showing stones



Liver and Gall Bladder

TYPES OF PAIN: VISCERAL - SOMATIC - REFERRED

REFERRED PAIN

- Pain felt distant to the involved or diseased organ
- "Classic distributions"
    - Diaphragmatic: radiate to supraclavicular area
    - Uretal colic: to lower abdominal quad, thighs, genitalia
    - Cardiac: epigastrium, chest, arm, shoulder, neck, jaw
- Commonly associated with cardiac, renal colic, inguinal hernia, abdominal aortic aneurysm

Cardiac: high, low, from arm or neck, down to anterior chest and epigastric region
Renal colic: may refer to thigh, genitalia, LQ of abdomen, CVAs
Aortic abdominal aneurysm: refers to mid to lower back

VISCERAL PAIN:

- Stretching of nerve fibers surrounding hollow or solid organs
- Gaseous, campy, colicky - may be ill-defined
- Usually felt midline but can be anywhere else
- May be, on exam, other than where it was described
- Commonly sources: unruptured AP, cholecystitis, bowel obstruction, renal colic

PARIETAL OR SOMATIC PAIN

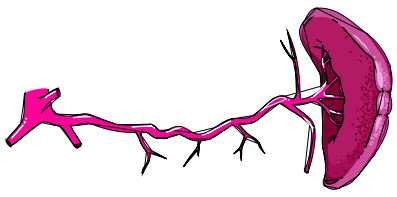
- Results from chemical or bacterial irritation of parietal peritoneum
- Sharp, more constant and more localized
- Usually indicates inflammation and clinically helpful because localized to area of pathology

COMPARISON SOMATIC VS VISCERAL PAIN

- Normal (somatic) sensation of abdominal wall: 7th -12th thoracic nerve roots
- Visceral innervation: 5th thoracic level and below (sympathetic)
- Bladder and rectum innervation: S2-S4 (parasympathetics)

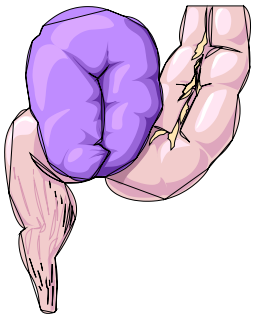
SPLENIC RUPTURE

- Most commonly injured organ w blunt abdominal trauma
- May be associated w other injuries as well
- Like AAA initially HTN-tachy -> rapidly shocky hypotensive and syncope
- Left "shoulder strap" pain plus LUQ abdominal pain
- Think mono in teenager wo trauma
- With rupture transport under close observation w BP support, IV volume, O2



BOWEL OBSTRUCTION:

- Different presentation as (f) whether early or late in process - partial vs complete obstruction.
- Vomiting if decreased ability to pass gas, increased cramping and subsequent abdominal distention
- Digital Rectal Exam (DRE): rectal vault may be empty or full
- Bowel sounds: diminished or w peristaltic "rushes" or high pitched tinkling sounds
- Flat plate: large dilated loops bowel (usually small) with or without air-fluid levels
- Adhesions are most freq cause so check sx hx
- Neoplasm also common cause



TREATMENT

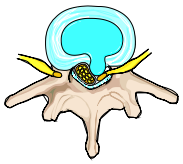
- NPO
- IV for any volume or elec depletion
- Sx consultation before any pain meds
- Transport under observation

NON-GI SOURCES OF ABDOMINAL PAIN

SICKLE CELL DISEASE

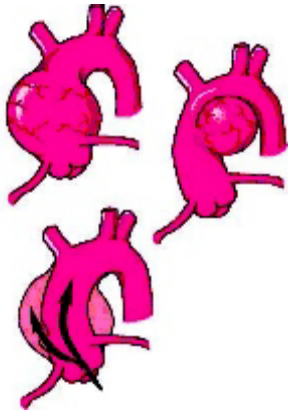
- Abdominal pain when in crisis along w leg pain, fever, H/A, epistaxis
- Must r/o infection (blood culture and P/E)

NEUROGENIC



- Primarily spinal disc disease or viral (Varicella Zoster)
- DJD from osteo may cause pain around the abdomen
- Zoster lesions are usually midline to midline, unilateral
  - Pain following same pattern as lesions (if present)
  - if no lesions (prodromal-post-herpetic neuralgia) -> pain more diffuse (less defined) but follows dermatomes

ABDOMINAL AORTIC ANEURISM (AAA)



- Static (arrest at certain size) or insidiously enlarge -> decompensate
- CXR pick up if calcification; CAT or u/s more accurate
- Asymptomatic to 4 cm in diameter
- Beyond 4 cm -> can rapid decompensation via rupture or dissection

- **Pain described as tearing; severe mid-lower back pain**
- Can confuse with severe back muscle spasm
- May radiate to genitals, sacrum, flank -> confuse with severe uretal colic
- Patient will rapidly decompensate, fluctuating BP: elevated BP -> barely perceptibly/shocky BP
- Very agitated with uncontrollable pain -> "shocky" quickly sets in
- **Pulsatile abdominal mass**: epigastric notch and umbilicus
- "Classic sign:" **lower extremity ischemia with "purple toes"**
- Present at end-stage of decompensation
- Primary mgt: IV volume, BP support, O2 and stat OR

- High acuity: **etopic pregnancy , ovarian torsion, spontaneous AB, ovarian cyst rupture**
- Lower acuity: **salpingitis (PID) tubo-ovarian abscess (TOA), hydrosalpinx**
- PID and TOA can smoulder and be insidious over days-weeks

- Low level pain
- Intermittent low-grade fever (f) slow buildup of "irritating" products of infection
- ESR often elevated (>15); CBC may/may not show leukocytosis w shift left
- 15% greater incidence of TOA; 20% greater incidence or recurrence of PID
- 8% increased incidence of ectopic
- Greater incidence of adhesions-bowel obstructions
- Mimics: Adhesion pain from adhesions, endometriosis or Mittelschmerz

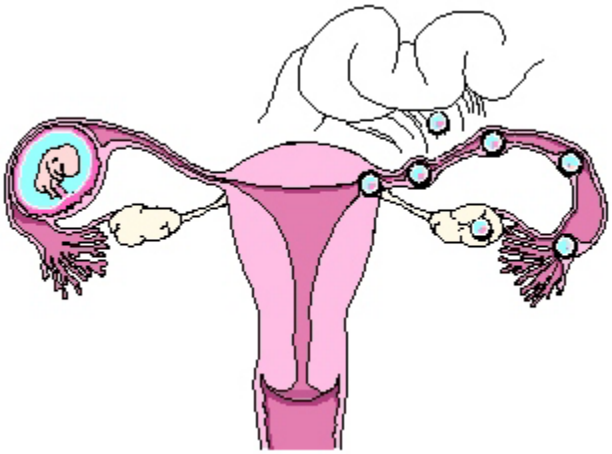
Ectopic pregnancy: unilateral or generalized abdominal pain - **life threatening bleeding**

- Up to 1% of all pregnancies; increased incidence with history PID, TOA, abdominal sx
- Pain with or without vaginal bleeding
- Pelvic hematoma may be present
- Syncope: Can present w syncope alone but abdominal pain presents relatively quickly
- Other causes of syncope:
  Pulmonary HTN, arrhythmias, drugs, TIA, carotid sinus syndrome, psychogenic causes, postural hypotension

- **Pregnancy test:** Mandatory for any childbearing-aged woman present w referred or LQ pain
- Get baseline hgb/hct and assess for vaginal bleed
- Check for ortho stasis
- Sometimes get palpable adnexal mass and cervical motion tenderness (CMT)
- No CMT w intrauterine pregnancy (IUP)
- Remember IUP can have concurrent PID
  - Menstrual history to include last 2 normal menses
  - If pregnant obtain ECD
  - ESR, ANA, anti-DNA
  - U/A may show hematuria if renal involvement

SIGNS AND SYMPTOMS

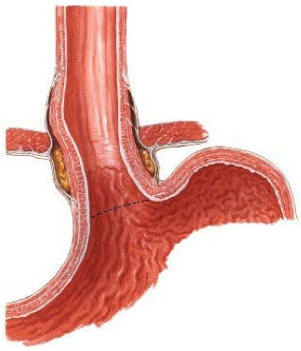
- Late or missed menses
- Breast tenderness
- Unexplained wt gain
- Nausea
- Rapid onset of usually unilateral lower quad abd pain



Ectopic Pregnancy

Ovarian Cyst

REFLUX ESOPHAGITIS - GERD



TERMINOLOGY

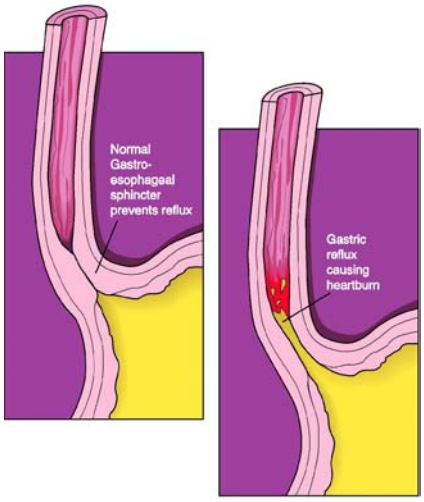
GERD - Gastroesophageal reflux disorder
Reflux esophagitis
"Heartburn"

PATHOPHYSIOLOGY

- Pathological process: gastric acid in frequent contact w esophageal mucosa
- Lower esophageal sphincter (LES)
  - pressure prevents reflux of gastric contents from reaching esophageal mucosa
  - when LES pressure decreases -> reflux occurs
  - unknown whether decrease of LES is result or precedes reflux
- Contributing or predisposing factors
  - Transient LES relaxations
  - Low or hypotensive LES
  - Anatomic disruption of sphincter: **hiatal hernia**
- Individuals with reflux clear acid more slowly than do normal individuals.

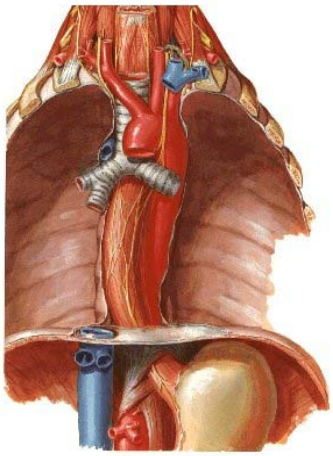
CLINICAL PRESENTATION

- **Heartburn** most common: burning sensation in chest after meals or when lying down
- Specific foods may provoke via decrease LES: chocolate, peppermint, ETOH
- Bitter or sour taste in mouth/ mouthful of fluid (**waterbrash**)
- Relief w antacids helps confirm
- Odynophagia only if severe and long standing esp if ulceration



- Dysphagia: food sticking in esophagus
  - if esophagitis: symptoms are transient
  - if organic stricture: dysphagia persists

- Night sweats associated w GERD in some pts
  - usually modest - pt rarely mentions spontaneously
- Suspect GERD where patient is experiencing certain symptoms even without C/O heartburn
  - chest pain
  - asthma
  - cough



DIAGNOSTICS

Barium Swallow
- Simplest test; least sensitive
- Often first test ordered
- Determines stricture but will show REFLUX only 25% of time and usually if severe

pH probe during esophageal manometry
- Most sensitive; usual pH is >4
- Probe positioned in esophagus can determine fall in pH (from GERD) 85% of time
- LES pressure simultaneously determined (85% of pt will have decreased pressure)

Endoscopy and biopsy
- Determines damage to esophageal mucosa
- **Barrett's esophagitis**: premalignant condition from chronic mucosal damage
- Most sensitive diagnostic test

Esophageal acid aka Berstein Test
- Tube placed in esophagus into which drips 0.1 HCl
- Confirms heartburn if test reproduces symptoms when subsequently disappear on D/C
- **24 Hour ambulatory pH recording**
- Confirms whether pt has prolonged acid reflux
- Determines whether timing of symptoms correlates with acid reflux

COMPLICATIONS

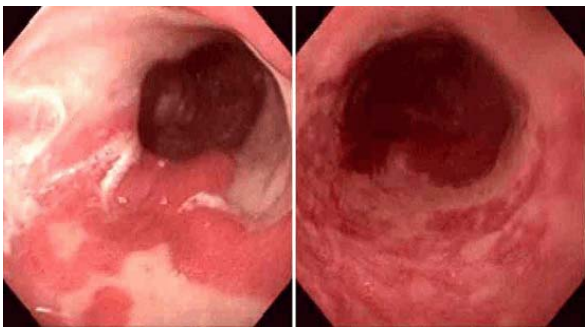
- Esophageal stricture with severe reflux
  - Results from inflammatory process extending into submucosa
  - Usually at distal esophagus and most often smooth vs irregular stricture w CA
  - ENDOSCOPY OR BX mandatory to r/o CA
  - Treatment: antireflux regiment and dilation w bougies or dilators
  - Occasionally require sx if re-stricture or tortuous

- Progressive dysphagia
  - Related to size of food bolus
  - Initially solids not liquids
  - Barium swallow w marshmallow or bread

Barrett esophagus: tight esophageal stricture

- Esophageal ulceration and hemorrhage from esophagitis
  - Uncommon; surgery needed if not respond to medical mgt
  - Ulcer: continuous vs intermittent pain
  - Hemorrhage: dx via esophagoscopy

- **Barrett's Esophagitis:** aka columnar dysplasia of esophagus
  - Uncommon but significant
  - Portion of squamous epithelium replaced w columnar epithelium
  - High predilection (10%) for developing esophageal adenocarcinoma
  - Endoscopy w biopsy q year if low-grade dysplasia and q 2 years of no dysplasia



These two endoscopic views demonstrate **Barrett esophagus** areas of mucosal erythema of the lower esophagus, with islands of normal pale esophageal squamous mucosa. If the area of Barrett mucosa extends less than 2 cm above the normal squamocolumnar junction, then the condition is called "short segment" Barrett esophagus, as shown

MEDICAL THERAPY

Primary goal: keep gastric acid away from esophageal squamous epithelium

1. Prevent reflux
   - Elevate HOB - avoid lying down p meals
   - Avoid eating 3 hours pre HS

   Substances which increase LES pressure
   Protein meal, coffee*, Urecholine (bethanechol), metoclopramide, antacids, a-adrenergic agents, b-adrenergic agents

   Substances which decrease LES pressure
   ETOH, chocolate, peppermint, smoking, fatty foods, b-adrenergic agents, estrogen and progesterone, caffeine, calcium blocking agents, diazepam, barbiturates

   * Coffee contains protein which increases LES, caffeine which decreases LES

2. Neutralize gastric acidity: antacids or H2-receptor antagonists

Antacids
- **AlOH-MgOH (Maalox)** 30 ml after meals and HS
- Avoid Calcium antacids (TUMS) - calcium stimulates acid production (Gastrin)
- Can cause diarrhea if > qid to 6x day

ANTACIDS

Aluminum hydroxide: can be constipating (Amphojel, Alternagel) - constipation
Magnesium hydroxide (MOM) laxative effect; contraindicated renal failure - diarrhea
Combo MgOH and AlOH (Maalox, Mylanta): each cancels side effects of other
Calcium carbonate (TUMS, Rolaids, Oscal) can cause rebound hyperacidity
Sodium bicarbonate: (Alka-Seltzer *) not recommended as can cause electrolyte derangements

* Salicylate and antacid (Aspirin 325 mg, sodium bicarbonate 1.916 g, citric acid 1 g; effervescent tabs

H2 Receptor Antagonists

- Significantly reduce gastric acid secretion
- BID dosing
- Do not increase LES strength

H2 RECEPTOR ANTAGONISTS

Tagamet (cimetidine)
Zantac (ranitidine)
Axid (nizatidine)
Pepcid (famotidine)

Proton Pump Inhibitors: moderate to severe esophagitis
- Much greater acid suppression than H2 blockers
- No longer has black box warning
- Rebound hyperacidity ?
- Will heal 90% of esophagitis w 12 week period

PROTON PUMP INHIBITORS

Prilosec (omeprazole) 20 and 40 mg
Prevacid (lansoprazole) 15 and 30 mg
Rabeprazole (Aciphex) 20 mg delayed-release e-c
Pantoprazole (Protonix) 40 mg delayed release e-c
Esomeprazole (Nexium) 20 and 40 mg

Alginic Acid Gaviscon

- Weak antacid with foam (alginic acid)
- Coats esophagus

- Forms protective barrier stomach to esophagus
  - Floats to top of gastric contents.
  - Pt "burps" alginic acid vs acid
  - Alginic acid is non-burning

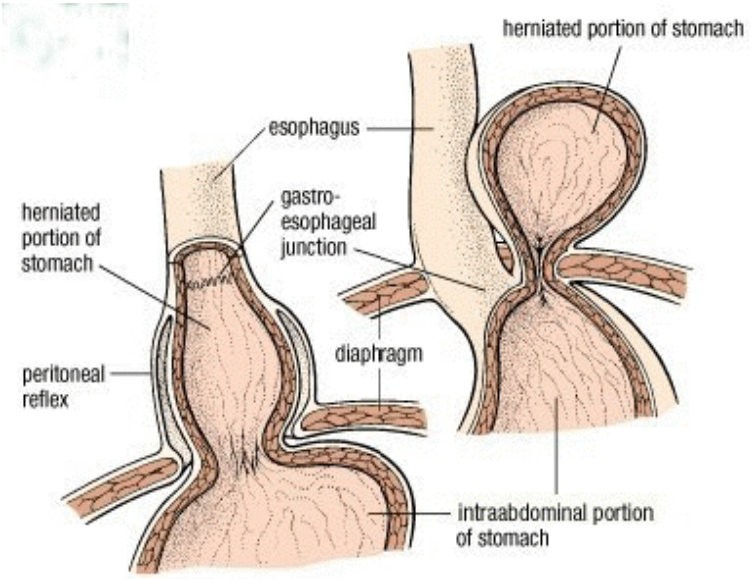
3. Increase LES pressure.

Reglan (metoclopramide) approved for diabetic gastroparesis

- potent dopamine inhibitor; S/E profile can be a problem
- 10 mg ac/hs
- increases LES and increases gastric emptying
- S/E profile (25%-50%) restlessness, tremors, parkinsonisms, tardive diskinesia

Propulsid (cisapride) prokinetic agent **- WITHDRAWN FROM MARKET**

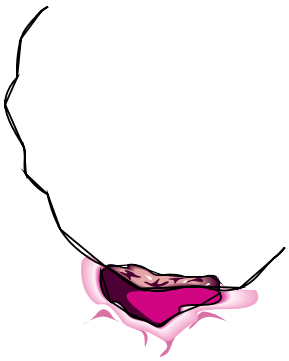
- Free of CNS s/e of Reglan
- Similar therapeutic effects 10 mg ac/hs
- Speeds transit time in gut - increases gastric and intestinal motility

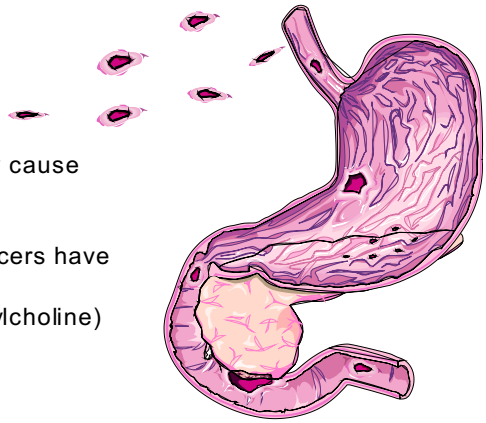


sliding esophageal and paraesophageal hernias: in sliding esophageal hernias (left) the upper stomach and cardioesophageal junction slide in and out of thorax; in paraesophageal hernias (right) all or part of the stomach pushes through diaphragm next to gastroesophageal junction

PEPTIC ULCER DISEASE

- **"Imbalance" between erosive properties** of HCL (gastric) acid, pepsin, and bile **vs protective** mechanisms of mucosa.
    - Mucus secretion
    - Prostaglandin availability
    - Mucosal blood flow
    - Bicarbonate secretion.





- Hypersecretion of gastric acid and pepsin may cause peptic ulcers
- Other factors may have physiologic role also
    - Only 40% of patients with duodenal ulcers have increased gastric acid
    - Neuronal input (other than vagal acetylcholine) may contribute to stress ulcers.

- **H Pylori** (urease producing gm negative rod): **major role**
    - Strongly associated with gastritis; humans are only reservoir
    - 75% of patients w gastritis
    - 90% of patients w duodenal ulcers us w gastritis
    - More prevent: older adults, African Americans, Hispanics, lower socioeconomic status
    - Transmission: person to person; iatrogenic w contaminated GI equipment documented
    - Bacteria which survives hostile acidic environment
    - Found between gastric epithelial cell and overlying mucus gel
    - Causes ulceration via cumulative effect of gastritis
        Increased acid secretion -> gastric metaplasia, duodenitis, reduced duodenal bicarbonate secretion and mucosal damage
    - Implicated: gastric adenocarcinoma, primary gastric lymphoma
    - Virtually all H pylori patients demonstrate antral gastritis
    - Eradication -> healing of gastritis and cure of PUD
    - Most H pylori-positive individuals do no develop peptic ulcers

Major causes of peptic ulcer disease (PUD)
- H. pylori infection
- NSAID
- Hypersecretory states e.g. Zollinger-Ellison Syndrome (ZES)
- Idiopathic (rare)
- Other: gastrinoma, mastocytosis, annular pancreas, HSV, CMV, candida albicans

NSAIDs
- > 1% US population uses NSAIDs; most well-tolerated
- 1/3 chronic users experience GI discomfort
- Serious complications: **ulcer, hemorrhage-perforation, gastric outlet obstruction**
    - Affects 2%-4% with daily use x 1 year
    - **Life threatening complications can arise without warning**
- Endoscopic exam after single dose 650 mg - 1300 mg ASA
    - Virtually all patients show surface epithelial damage
    - Gastric antrum most frequent site of damage
- **Advanced age most important risk factor for GI bleed and death.**
- Damaging effects thru local irritative effects and systemic effects
    - Cyclooxygenase (COX) inhibition
    - Depletion of endogenous prostaglandins
- **NSAIDs reduce both COX-1 and COX-2**
    - Results in antipyresis and antiinflamation at cost of GI toxicity
    - Cox 1 inhibition is responsible for GI side effects; Cox 2 promotes ulcer healing

- **Cox-2 inhibitors**: introduced 1999
    - **Selectively inhibit Cox 2** thus permits beneficial effects without GI toxic effects
    - Higher cost; equally effective. Good choice for elderly and patients at risk

Risk factors
- Cigarette smoking
- ASA - other analgesic use
- Blood group O
- H pylori
- No data to support: ethanol ingestion or specific dietary factors
- Stress possible trigger (also impairs treatment) role but no direct evidence

EPIDEMIOLOGY:

- 25 million Americans suffer from PUD
- 500,ooo to 850,000 new cases of PUD
- More than 1 million ulcer-related hospitalizations
- Gastric ulcer incidence same for both sexes; duodenal ulcers 2:1 males vs females
- Incidence increase age > 70 yrs esp if using NSAIDs
- Genetic predisposition suggested

SIGNS/SYMPTOMS

- Most common s/s is gnawing or burning pain in the epigastrium (dyspepsia)
    - 80-90% patients
    - Well localized to epigastrium; not severe
- 50% report relief with food or antacids w recurrence 2-4 hours later
- Nocturnal pain which wakes patient: 67% of DU and 33% of GU
- Asymptomatic periods up to weeks with months to years when pain free
- Typically timing (can occur at other times, as well)
    - When stomach is empty
    - Between meals
    - Early morning hours

- Lasts minutes to hours
- Less common symptoms
    - Nausea, vomiting, loss of appetite
    - Bleeding can occur
- If bleeding is prolonged -> anemia - weakness/fatigue
- If bleeding heavy -> hematemesis, hematochezia or melena
- Dx of PUD generally based on history
- Physical exam: benign except for possible epigastric tenderness
    - No definitive finding on P/E to distinguish from other organic or functional GI disorders
    - Complications may produce clinical findings
        - Ruptured ulcer, gastric outlet obstruction, bleeding, pancreatis

    - Ruptured ulcer: peritoneal findings (distended abdomen, rigidity, increased tympany
    - Gastric outlet obstruction:
        - Signs/symptoms volume depletion, succussion splash on shaking patient
        - Fluid in cavity
    - Bleeding: occult blood; hematemesis; if severe: hypotension-tachycardia
    - Pancreatitis if posterior DU erodes through wall into head of pancreas
        Severe pain, acute abdomen (distention, rigid abdomen, absent BS)

DIFFERENTIATING GASTRIC AND DUODENAL

	DUODENAL ULCER	GASTRIC ULCER
Incidence	Peak age, 40 yrs Men: Women = 1:1 Prevalence: 10% of population 4 X greater incidence than gastric ulcer	Peak age 50-60 yrs Men:women = 2.5-1 Lifetime prevalence: 10%
Pathogenesis	Acid mediated. Hyperacidity Gastric colonization w H pylori in 90-95% Associated diseases Hyperparathyroidism, chronic pulmonary disease, chronic pancreatitis alcoholic cirrhosis Ulcerogenic drugs, tobacco Blood group 0	- Dysfunction or lack of normal mucosal barrier - Normal to lo Hcl production - Presence of H pylori gastritis - Ulcerogenic drugs, tobacco - Chronic bile reflux - Familial predisposition- Not related to blood group
Pathology	90% in duodenal bulb	90% in antrum and lesser curvature
Obstruction	Common	Rare
Malignancy	Almost never	Incidence about 4%
Signs and symptoms	Pain-food-relief patter Usually well nourished Seasonal exacerbation Nightly pain possible	Food-pain pattern Anorexia, weight loss common Night pain possible

DIAGNOSIS

- NSAIDs use: D/C NSAIDs followed by diagnostic assessment for H pylori
- Non-NSAID induced: 3 options
    - Single short-term trial (2 wks) of anti-ulcer therapy
    - Diagnostic evaluation (especially if age > 50

Detection of H pylori (3 options)

- **Endoscopy with biopsy** (invasive with risks)
    - Histology (sensitive and specificity 90%)
    - Culture (least sensitive method of detection)
    - risks: IV sedation, perforation, bleeding, bacterial contamination

- **Serology for IgG** antibodies to H pylori antigen: 95% specificity-sensitivity
    - False negatives w recent use: antibiotics, bismuth, or omeprazole
    - Quantitative titers decrease over 6-12 months
    - Serology is positive for years
    - Good initial choice due to low cost and ease

- **Breath test** with C-labeled urea: 95% sensitivity and specificity
    - Orally administered ^{13}C-labeled and ^{14}C-labeled urea
    - H pylori metabolizes urea rapidly -> labeled carbon absorbed
    - Labeled carbon measured as CO2 in pts expired breath
    - Indicates presence of H pylori

TREATMENT

H2 RECEPTOR ANTAGONISTS

Tagamet (cimetidine)
Zantac (ranitidine)
Axid (nizatidine)
Pepcid (famptodome)

- Goals: eradicate H pylori, heal ulcer, prevent recurrence
- Non-H pylori ulceration
    - Trial of **antacids** and/or H2 antagonists or proton pump inhibitors
    - Monotherapy with antacids not mainstay of treatment
    - Antacids used as adjuvant therapy
- **H2 antagonists**
    - Inhibit secretion by histamine, muscarine agonists and gastrin
    - Inhibit fasting and nocturnal secretions; secretions by food and insulin
    - Alleviate symptoms; prevent complications of PUD
    - Short term therapy (4-6 weeks) heals DU/GU; reduced dose maintenance thereafter

- **Proton pump Inhibitors**
    - Suppress gastric acid secretion at secretory surface of parietal cell.
    - Dose related; inhibits basal and stimulated acid secretion
    - Initially prescribed 4-6 weeks -> reduced dose maintenance therapy

- Diagnostic testing if drug therapy does not alleviate symptoms

- **H-pylori Treatment**:
    - NIH recommendations:
        - All patients infected be treated w antimicrobials whether initial or recurrence
        - Co-administration of antisecretory drugs
    - Treatment of asymptomatic pts is controversial: no supporting evidence
    - Empiric treatment is discouraged

TREATMENT REGIMENS FOR H PYLORI

- H Pylori (urease producing gram negative rod): major role
- Treatment regiments involve **combination of drugs - antibiotics, acid-suppressants, bismuth**
- Variety of appropriate treatment regiments using different combos of drugs - some FDA-approved

- Long term recurrence rates lower **with eradication via antibiotic regimen** (esp with bismuth)

PROTON PUMP INHIBITORS

Omeprazole (Prilosec)
Lansoprazole (Prevacid)
Rabeprazole (Aciphex)
Pantoprazole (Protonix)
Esomeprazole (Nexium)

    - Treatment of ulcers with H pylori
        - Proton pump inhibitor or H2 agonist
        - Antibiotic therapy (eliminate bacteria)

    - Three (3) classes of drugs have direct effect on H pylori
        - Antibiotics
        - Bismuth salts
        - Proton pump inhibitors
    - Difficult to eradicate: most treatment regimens combine agents from 2 or 3 classes

- All patients with active peptic ulcer disease should also receive 6 weeks of acid suppression
    - H2 receptor antagonist
    - Proton Pump Inhibitor

ANTIBIOTICS USED IN TREATING H. PYLORI

Amoxicillin
Metronidazole (Flagyl)
Clarithromycin (Biaxin)
Tetracycline