**GASTROINTESTINAL PATHOPHYSIOLOGY I**

Lois E. Brenneman, MSN, CS, ANP, FNP

**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

**LIFE THREATENING CAUSES OF ACUTE ABDOMINAL PAIN**

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

## Abdominal Pain by Rapidity of Onset

### ABRUPT ONSET (INSTANTANEOUS)

**Gastrointestinal**
- Perforated ulcer
- Ruptured Abscess or hematoma
- Intestinal infarct

**Extra-Gastrointestinal**
- Ruptured or dissecting aneurysm
- Ruptured ectopic pregnancy
- Pneumothorax
- Myocardial infarction
- Pulmonary infarction

### RAPID ONSET (MINUTES)

**Gastrointestinal**
- Perforated viscus
- Strangulated viscus
- Volvulus
- Pancreatitis
- Mesenteric infarct
- Diverticulitis
- Penetrating peptic ulcer
- High intestinal obstruction
- Appendicitis *
  
*gradual onset more common*

**Extra-Gastrointestinal**
- Urethral colic
- Renal colic
- Ectopic pregnancy

### GRADUAL ONSET (HOURS)

**Gastrointestinal**
- Appendicitis
- Strangulated hernia
- Low small intestinal obstruction
- Cholecystitis
- Pancreatitis
- Mesenteric lymphadenitis
- Meckel’s diverticulitis
- Colonic diverticulitis

**Extra-Gastrointestinal**
- Crohn’s Disease
- Ulcerative colitis
- Abscess
- Gastritis
- Intestinal Infarct
- Meckler’s Cyst
- Peptic ulcer

- Cystitis
- Pyelitis
- Salpingitis
- Prostatitis
- Threatened abortion
- Urinary retention
- Pneumonitis

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**Cholelithiasis**
Gall Bladder showing stones

**Liver and Gall Bladder**

Renal calculi with hydronephrosis

Appendix
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
PELVIC SOURCES OF ABDOMINAL PAIN

- High acuity: ectopic 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 build up 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

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REFLUX ESOPHAGITIS - GERD

TERMINOLOGY

GERD - Gastroesophageal reflux disorder
Reflux esophagitis
“Heartburn”

PATHOPHYSIOLOGY

- Pathological process: gastric acid in frequent contact with 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
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.
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

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**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

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**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 antiinflammation 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
- 500,000 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% ofGU
- 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, pancreatitis
- 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 ULCER

<table>
<thead>
<tr>
<th>DIFFERENTIATING GASTRIC AND DUODENAL</th>
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<tbody>
<tr>
<td>DUODENAL ULCER</td>
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<tr>
<td>GASTRIC ULCER</td>
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<tr>
<td>Incidence</td>
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<tr>
<td>Peak age, 40 yrs</td>
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<tr>
<td>Men: Women = 1:1</td>
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<tr>
<td>Prevalence: 10% of population</td>
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<tr>
<td>4 X greater incidence than gastric ulcer</td>
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<tr>
<td>Peak age 50-60 yrs</td>
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<tr>
<td>Men:women = 2.5-1</td>
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<td>Lifetime prevalence: 10%</td>
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<tr>
<td>Pathogenesis</td>
</tr>
<tr>
<td>Acid mediated. Hyperacidity</td>
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<tr>
<td>Gastric colonization w H pylori in 90-95%</td>
</tr>
<tr>
<td>Associated diseases</td>
</tr>
<tr>
<td>Hyperparathyroidism, chronic pulmonary disease, chronic pancreatitis alcoholic cirrhosis</td>
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<tr>
<td>Ulcerogenic drugs, tobacco</td>
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<tr>
<td>Blood group 0</td>
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<tr>
<td>- Dysfunction or lack of normal mucosal barrier</td>
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<tr>
<td>- Normal to lo Hcl production</td>
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<tr>
<td>- Presence of H pylori gastritis</td>
</tr>
<tr>
<td>- Ulcerogenic drugs, tobacco</td>
</tr>
<tr>
<td>- Chronic bile reflux</td>
</tr>
<tr>
<td>Pathology</td>
</tr>
<tr>
<td>90% in duodenal bulb</td>
</tr>
<tr>
<td>90% in antrum and lesser curvature</td>
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<tr>
<td>Obstruction</td>
</tr>
<tr>
<td>Common</td>
</tr>
<tr>
<td>Rare</td>
</tr>
<tr>
<td>Malignancy</td>
</tr>
<tr>
<td>Almost never</td>
</tr>
<tr>
<td>Incidence about 4%</td>
</tr>
<tr>
<td>Signs and symptoms</td>
</tr>
<tr>
<td>Pain-food-relief patter</td>
</tr>
<tr>
<td>Usually well nourished</td>
</tr>
<tr>
<td>Seasonal exacerbation</td>
</tr>
<tr>
<td>Nightly pain possible</td>
</tr>
<tr>
<td>Food-pain pattern</td>
</tr>
<tr>
<td>Anorexia, weight loss common</td>
</tr>
<tr>
<td>Night pain possible</td>
</tr>
</tbody>
</table>

**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

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12
H2 RECEPTOR ANTAGONISTS

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

PROTON PUMP INHIBITORS

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

ANTIBIOTICS USED IN TREATING H. PYLORI

- Amoxicillin
- Metronidazole (Flagyl)
- Clarithromycin (Biaxin)
- Tetracycline

TREATMENT

- 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 with 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 regimens using different combos of drugs - some FDA-approved

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