Small and Large Bowel Obstruction

October 20, 2004
Resident Teaching Conference
Kutz / Burchard
Small Intestine Function

• Transport
  ▪ Distension stimulates contractions

• Mixing and peristalsis

• Absorption
  ▪ Carbs, proteins direct absorption
  ▪ Lipids converted to chylomicrons
    • Fat soluble vitamins
  ▪ Vitamins & Minerals
    • Ca^{+2}, FeSO_{4}, B12, Bile Salts
Colon Function

- Water Absorption
  - Right side primarily

- Transport

- Storage
<table>
<thead>
<tr>
<th>Mucosal Resistance</th>
<th>Leaky</th>
<th>Moderately Leaky</th>
<th>Moderately Tight</th>
<th>Tight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous P.D.</td>
<td>3 mV</td>
<td>6 mV</td>
<td>12 mV</td>
<td>20 mV</td>
</tr>
<tr>
<td>Absorptive Mechanisms</td>
<td>Na-nutrient Na-H</td>
<td>Na-Cl Na-nutrient Na</td>
<td>Na SCFA</td>
<td>NaCl SCFA</td>
</tr>
</tbody>
</table>

Diagram shows the flow of fluid through different parts of the digestive system, with volume measurements for each section.
The strength layer of the bowel in intestinal anastomoses is the:

A. Mucosa
B. Submucosa
C. Muscularis mucosae
D. Muscularis externa
E. Serosa

Why a 1 Layered Closure Works!
FIGURE 24-45.
The blood supply to the jejunum and ileum: the ileocolic artery lies at the base of the mesentery of the intestine; the stem of the superior mesenteric artery swings into the mesentery.
G U R E 24-47.

c marginal artery (colored).
Note: intestinal wall is shown much thicker than in actuality
The inferior mesenteric vein drains to the:

A. Portal Vein
B. Splenic Vein
C. External Iliac Vein
D. Internal Iliac Vein

IMV → Splenic v → Portal v
Which of the following statements about small bowel motility is true?

A. Oral feeding stimulates production of migrating motor complexes (MMC’s)
B. If motility is impaired, absorption of nutrients is also impaired
C. The frequency of MMC’s returns to normal within 6 - 24 hours after surgery
D. Vagotomy-induced diarrhea is due to increased secretion secondary to denervation
E. The Migrating Motor Complex is Electric, Boogy-woogy-woogy
Motility - Slow Waves

• Caused by cyclic activation and deactivation of the cell Na\(^+\)/K\(^+\) Pump

• NOT Contractions

• Depolarization during each slow wave brings the membrane potential of smooth muscle cells closer to threshold
  - Increases probability of action potential $\rightarrow$ contraction

• “Spikes” are superimposed on slow waves and are action potentials (contractions)
Motility Small Bowel

A. Slow Waves and spikes
   A. Gastric – 3/min
   B. SB – 7-12/min
   C. Colon – 12/min

B. Electric activity

C. Contraction
Intestinal Motility

• Peristalsis
  ▪ Transport
  ▪ Most regulation at level of stomach and duodenum
  ▪ Small bowel and Colon rely more on enteric nervous system coordinating peristalsis

• Segmentation
  ▪ Mixing
  ▪ Non-propagated, back-and-forth contraction
  ▪ Decreases in rate from proximal to distal
Intestinal Motility - Reflexes

- **Gastroileal**
  - Increased TI motility and emptying into colon

- **Ileogastric**
  - TI distention decreases gastric motility

- **Intestinal**
  - Overdistension of intestine decreases intestinal motility overall
Intestinal Motility

• Fasting
  ▪ Migrating Myoelectric Complex
    • Occur every 75-90 minutes Stomach to TI
    • Clears accumulated fluid

• Colon
  ▪ Segmental contractions – Responsible for Haustra
    • Facilitates water and salt absorption
    • Mixing motions, little peristalsis with segmentation
    • Mass Movements 3-4 per day (peristalsis)
      ▪ Contents moved distally for long distances
      ▪ Gastrocolic reflex through PNS when stomach stretches
  ▪ Enteric nerves primarily inhibitory
Which of the following stimulates colonic motility?

A. Cholecystokinin
B. Secretin
C. Somatostatin
D. Vasointestinal Peptide
E. All of the Above

Secretin stimulates $\text{HCO}_3^-$ by the Pancreas and bile from liver, Somatostatin is an inhibitory hormone that shuts secretion off and decreases motility, VIP reduces gastric motility and gastric acid production.

When chyme enters duodenum, hypertonicity and proteins stimulate CCK which causes GB contraction. Gastroileal and gastrocolic reflexes stimulate TI emptying into colon, think that CCK augments motility of colon at this time to help move the fluid TI is emptying into it.
Obstruction - Pathophysiology

- **Dependent Upon:**
  - Degree of Obstruction
  - Duration of Obstruction
  - Presence and Severity of Ischemia

- **Results in:**
  - Accumulation of fluid and air
  - Bacterial overgrowth
    - Maximal by 24 hrs after obstruction
    - Gut translocation to nodes and portal system
  - Once Distended get:
    - Impaired fluid and nutrient absorption
    - Secretion of isotonic fluid (intravascular $\rightarrow$ intraluminal) thought secondary to bacterial overgrowth
Obstruction - Pathophysiology

- Systemic symptoms thought secondary to:
  - Hypovolemia
  - Bacterial translocation (typically *E. coli*)
  - Ischemia worsens general inflammatory state, bacterial translocation, and fluid requirements

- Large Bowel Obstruction
  - Ileocecal valve plays prominent role in pathophysiology of LBO
  - If competent valve = closed loop obstruction
  - Cecal ischemia around 10-13 cm
Obstruction

- 2 Varities:
  - Mechanical
    - 90% of Mechanical SBO due to adhesions, hernias, or cancer
    - Colonic obstruction accounts for only 10-15% of all mechanical obstructions
      - Most commonly carcinoma, diverticulitis, or volvulus
  - Non-mechanical (ileus)
Mechanical Obstruction

- Acute vs Chronic
- Partial vs Complete
- Simple vs Closed loop
- Gangrenous vs Nongangrenous

- Natural history, response to treatment, and associated M&Ms vary based on type of obstruction
a. SMALL INTESTINAL OBSTRUCTION

b. COLONIC OBSTRUCTION
### Table 2  Causes of Small Bowel Obstruction in Adults

**Extrinsic causes**
- Adhesions*
- Hernias (external, internal [paraduodenal], incisional)*
- Metastatic cancer*
- Volvulus
- Intra-abdominal abscess
- Intra-abdominal hematoma
- Pancreatic pseudocyst
- Intra-abdominal drains
- Tight fascial opening at stoma

**Intraluminal causes**
- Tumors*
- Gallstones
- Foreign body
- Worms
- Bezoars

**Intramural abnormalities**
- Tumors
- Strictures
- Hematoma
- Intussusception
- Regional enteritis
- Radiation enteritis

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*Approximately 85% of all small bowel obstructions are secondary to adhesions, hernias, or tumors.*
<table>
<thead>
<tr>
<th>Table 3  Causes of Colonic Obstruction</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Common causes</strong></td>
</tr>
<tr>
<td>Cancer (primary, anastomotic, metastatic)</td>
</tr>
<tr>
<td>Volvulus</td>
</tr>
<tr>
<td>Diverticulitis</td>
</tr>
<tr>
<td>Pseudo-obstruction</td>
</tr>
<tr>
<td>Hernia</td>
</tr>
<tr>
<td>Anastomotic stricture</td>
</tr>
<tr>
<td><strong>Unusual causes</strong></td>
</tr>
<tr>
<td>Intussusception</td>
</tr>
<tr>
<td>Fecal impaction</td>
</tr>
<tr>
<td>Strictures (from one of the following)</td>
</tr>
<tr>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td>Endometriosis</td>
</tr>
<tr>
<td>Radiation therapy</td>
</tr>
<tr>
<td>Ischemia</td>
</tr>
<tr>
<td>Foreign body</td>
</tr>
<tr>
<td>Extrinsic compression by a mass</td>
</tr>
<tr>
<td>Pancreatic pseudocyst</td>
</tr>
<tr>
<td>Hematoma</td>
</tr>
<tr>
<td>Metastasis</td>
</tr>
<tr>
<td>Primary tumors</td>
</tr>
</tbody>
</table>
Mechanical Obstruction - Presentation

• Distinguished from ileus or pseudo-obstruction by:
  ▪ **Location** – mid-abdomen (others diffuse)
  ▪ **Character** – colicky
    • Proximal – periodicity every 3 - 4 mins
    • Distal SB or Colon – every 15-20 minutes
    • Between episodes of nausea, vomiting, cramping
  ▪ **Severity** of pain – generally severe, worsens over time (ileus tends to be constant)
Mechanical Obstruction - History

- Did this ever happen before?
- Change in bowel habits (acute vs chronic)
- Progressive vs acute abd. distention
- Weight loss
- Flatus?
- Prior Surgeries
- Hx of abdominal CA?
- Hx of Inflammatory Bowel Dz
- Prior abdominal XRT?
- Meds: Anticoagulants, Anticholinergics, Opioids, Antihistamines, Alpha-agonists, Catecholamines
Obstruction - Exam

- Want idea of vitals, hydration status, and cardiopulmonary status
- NGT, Foley, IV placed
- Volume and character of NG aspirate:
  - Clear - GOO
  - Bilious – Mid to distal SBO
  - Feculent – Distal SBO to LBO
- aMI or Pneumonia a possible cause?
  - Why CXR part of acute abdominal series
Mechanical Obstruction - Exam

• Abdominal Exam:
  - **Observe**
    - Distention – More in distal or chronic obstruction
    - Symmetry – Assymmetric may be mass vs volvulus
    - Incisions
    - Visible peristalsis – acute obstruction
  - **Auscultate**
    - High-pitched
    - Rushes
    - Tingles
    - Absent – late obstruction
  - **Palpate**
    - Inguinal, Femoral, Umbilical, Incisional hernias.
    - Pain out of proportion of exam (closed-loop obstruction)
  - **Percuss**
    - Dull – Fluid or mass
    - Tympanic – Air (intraluminal or not)
    - Peritoneal irritation

• DRE or Digital Ostomy Exam for mass, impaction, peristomal hernia...etc.
### Table 26.2. SYMPTOMS AND SIGNS OF BOWEL OBSTRUCTION

<table>
<thead>
<tr>
<th>Symptom or sign</th>
<th>Proximal small bowel (open loop)</th>
<th>Distal small bowel (open loop)</th>
<th>Small bowel (closed loop)</th>
<th>Colon and rectum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td>Intermittent, intense, colicky; often relieved by vomiting</td>
<td>Intermittent to constant</td>
<td>Progressive, intermittent to constant; rapidly worsens</td>
<td>Continuous</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Large volumes, bilious and frequent</td>
<td>Low volume and frequency; progressively feculent with time</td>
<td>May be prominent (reflex)</td>
<td>Intermittent, not prominent; feculent when present</td>
</tr>
<tr>
<td>Tenderness</td>
<td>Epigastric or periumbilical; quite mild unless strangulation is present</td>
<td>Diffuse and progressive</td>
<td>Diffuse, progressive</td>
<td>Diffuse</td>
</tr>
<tr>
<td>Distention</td>
<td>Absent</td>
<td>Moderate to marked</td>
<td>Often absent</td>
<td>Marked</td>
</tr>
<tr>
<td>Obstipation</td>
<td>May not be present</td>
<td>Present</td>
<td>May not be present</td>
<td>Present</td>
</tr>
</tbody>
</table>

The most helpful diagnostic radiographic procedure in suspected SBO is:

A. CT of Abdomen and Pelvis
B. UGI gastrograffin contrast study
C. Supine and Erect plain films of abdomen
D. U/S of the abdomen
Imaging – Plain Films

- Dilated loops, SB dilation? AFLs?
- Are AFL’s and bowel loops in same place on supine and upright films?
- Is there gas throughout the entire colon?
  - Ileus or pSBO
- Paucity of distal colonic gas or abrupt cutoff of colonic gas w/proximal distention and/or AFLs?
  - Suggesting complete or near-complete LBO
- Massive dilation of colon, especially of the cecum or sigmoid?
  - Suggestive of volvulus or pseudoobstruction
- Evidence of strangulation?
  - Thickened SB loops, mucosal thumb printing, pneumatosis cystoides intestinalis, or free air
- Are there biliary or renal calculi? Pneumobilia?
  - GS ileus? Renal stone causing ileus?
Mechanical Obstruction - Labs

- CBC
- Electrolytes
  - Including Mg$^{+2}$ and Ca$^{+2}$
- BUN/Cr
- Coags
- U/A (hematuria, UTI)
SBO from adhesions. Note fixed loop of small bowel in right pelvis (arrow) that doesn’t change position with different patient position – suggests adhesion.
Adjunctive Tests

- **Sigmoidoscopy**
  - When large amounts of air extend down to the rectum
  - Flex or Rigid will exclude rectal or distal sigmoid obstruction

- **CT Scan or U/S**
  - When normal plain films but history and exam are consistent with obstruction
  - Plain films in SBO will be nondiagnostic approximately 30% of the time
Mortality

- Strangulation obstruction is cause in about 10% of SBO’s
  - Mortality of 10-37%

- Simple SBO mortality < 5%

- Classic signs of continuous abd. pain, fever, tachycardia, peritoneal signs, and leukocytosis NOT sensitive or specific for strangulation
  - 50% of patients w/strangulation were not recognized preoperatively
3 Reasons:
1. Low likelihood of spontaneous resolution of complete obstruction
2. High risk of strangulation of complete SBO (8 – 22%)
3. Difficulty in detecting strangulation clinically until late in course
## Nonmechanical Obstruction

### Table 1 Causes of Ileus

<table>
<thead>
<tr>
<th>Intra-abdominal causes</th>
<th>Extra-abdominal causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intraperitoneal problems</td>
<td>Thoracic problems</td>
</tr>
<tr>
<td>Peritonitis or abscess</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>Inflammatory condition</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>Mechanical: operation, foreign body</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Chemical: gastric juice, bile, blood</td>
<td>Rib fractures</td>
</tr>
<tr>
<td>Autoimmune: serositis, vasculitis</td>
<td>Metabolic abnormalities</td>
</tr>
<tr>
<td>Intestinal ischemia: arterial or venous, sickle-cell disease</td>
<td>Electrolyte imbalance (e.g., hypokalemia)</td>
</tr>
<tr>
<td>Retroperitoneal problems</td>
<td>Sepsis</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Lead poisoning</td>
</tr>
<tr>
<td>Retroperitoneal hematoma</td>
<td>Porphyria</td>
</tr>
<tr>
<td>Spine fracture</td>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>Aortic operation</td>
<td>Hypoparathyroidism</td>
</tr>
<tr>
<td>Renal colic</td>
<td>Uremia</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td>Medicines</td>
</tr>
<tr>
<td>Metastasis</td>
<td>Opiates</td>
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<tr>
<td></td>
<td>Anticholinergics</td>
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<tr>
<td></td>
<td>Alpha agonists</td>
</tr>
<tr>
<td></td>
<td>Antihistamines</td>
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<tr>
<td></td>
<td>Catecholamines</td>
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<tr>
<td></td>
<td>Spinal cord injury or operations</td>
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<tr>
<td></td>
<td>Head, thoracic, or retroperitoneal trauma</td>
</tr>
<tr>
<td></td>
<td>Chemotherapy, radiation therapy</td>
</tr>
</tbody>
</table>
Gallstone ileus is best treated by:

A. Prokinetic agents
B. Initial control of the cholecystoenteric fistula
C. Ileocecectomy
D. Removal of the obstructing stone by enterotomy
E. None of the above

Board Answer: Do *nothing* to the cholecystoenteric fistula!
b. COLONIC OBSTRUCTION

- Neoplasms (60%)
- Volvulus (20%)
- Diverticular stricture (10%)
- Miscellaneous (10%)
Volvulus of the Colon

- Sigmoid most commonly involved
  - 75% of all colonic volvulus
  - 10% of all colonic obstructions
  - 180° or > counterclockwise twist
- Cecal volvulus next most common
- Transverse colon volvulus is rare
  - 4% of colonic volvulus
- Splenic flexure volvulus VERY rare
Sigmoid Volvulus – Predisposing Factors

- Long and highly-mobile sigmoid
- Lengthy mesosigmoid
  - Narrow at its base
- Adhesions between proximal sigmoid and rectosigmoid
  - With long loop of colon inbetween
- Chronic constipation and high-fiber diet
Sigmoid Volvulus - Pathogenesis

- Closed-loop type obstruction
- Proximal colon dilates
  - Extent depends on ileocecal valve competence
- Simple or Strangulated
  - Venous then arterial obstruction
  - With simple form takes a few days for vascular compromise to develop
  - In acute fulminating variant much more rapid course seen
Sigmoid Volvulus - Varieties

• Acute Fulminating Type
  ▪ Mortality 37-80%
  ▪ Younger patient, sudden onset, rapid course
  ▪ Early vomiting, severe pain, peritonitis, and gangrene
  ▪ Minimal distension often, hard to diagnose

• Subacute Progressive Type
  ▪ Generally older pt., more gradual onset
  ▪ Hx prior attacks, chronic constipation
  ▪ Abdominal distension often extreme
  ▪ Late vomiting, pain is minimal, no peritonitis
Sigmoid Volvulus - Diagnosis

- Plain films of Abdomen
  - Massively dilated and distended bowel loop
  - Both ends in pelvis, bow of loop cephalad
  - “Bent inner tube” sign
  - Air/fluid level within loop
  - Proximal colon & SB may be dilated

- Barium enema
  - “Birds beak”
Sigmoid Volvulus
Sigmoid Volvulus - Treatment

• Rigid sigmoidoscopic detorsion, decompression, & placement of rectal tube
  ▪ RT inserted past obstruction point
  ▪ Successful 77 - 90% of time
  ▪ Mortality rate 1.2 - 5.5%
  ▪ Preferred initial treatment

• Colonoscopic decompression (alternative)

• Surgery if:
  ▪ Decompression not successful
  ▪ Ischemic or necrotic bowel encountered
Sigmoid Volvulus – Surgical Treatment

- Recurrence rate is high (33 - 60%)
- Some advise elective op after 1st episode
- Elective:
  - Resection of redundant colon
  - Small transverse LLQ incision
- Sigmoid fixation another option
- Emergent: Resection +/- stoma
Cecal Volvulus
Ogilvie’s Syndrome
(Acute Colonic Pseudo-obstruction)

- Massive dilation of cecum, right, and transverse colon (non-mechanical), to splenic flexure
- Dilated SB loops in > 50%
- Symptoms:
  - Distension
  - Nausea and vomiting (2 out of 3)
  - Abdominal pain in 80%
    - Generally mild to moderate
  - 50% have flatus or diarrhea
  - Peritonitis if perforated only
- Bowel sounds may be normal or increased
Conditions associated with Ogilvie’s Syndrome

- Non-operative Trauma
- Non-GI surgery (Gyn, Ortho, Cardiac)
- Pancreatitis, Cholecystitis
- Diabetes, Malignancy
- Narcotics, antidepressants, anticholinergics
- Neurologic or Respiratory disease
- Electrolyte (hypo K+, Ca+2), Acid / Base disorder
- Radiation in past
Ogilvie’s Syndrome: DDx and Dx

- Plain abdominal films to diagnose and follow cecal diameter (Q12-24 hrs)
  - Normal is < 9 cm
  - If ≥ 14 cm → perforation in 23%

- Differential Includes:
  - Fecal impaction
  - Cecal or Sigmoid Volvulus
  - Ischemic Bowel
  - Mechanical Obstruction
Ogilvie’s Syndrome: Treatment

• Correct electrolyte problems and underlying condition
• D/C narcotics and anticholinergics
• NPO with NGT, +/- Rectal Tube
• Colonoscopic decompression difficult but often successful
  ▪ May need 2nd decompression
• Long colonic drains can be placed via colonoscope (old)
• Neostygymine is the treatment of choice now
• If signs of perforation then OR
Figure 12  Shown is an algorithm outlining an approach to management of ileus.

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Figure 13  Shown is an algorithm outlining an approach to management of pseudo-obstruction.