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Myths of Gastroenterology

Eric D. Libby

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What Are GI Myths

– Annoyances/Inaccuracies
  • Pet peeves

– Leading to poor or expensive care
  • Unnecessary or harmful regimens
  • Tests that don’t help
  • Preventing beneficial treatment

– Negatively impact patient quality of life

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Patient sent from
the emergency room with
melanotic stool
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**melanotic** [mel"ah-not^ik]

- Characterized by the presence of melanin; pertaining to melanosis.

- Melena is defined as the passage of dark tarry stools containing decomposing blood that is usually an indication of bleeding in the upper part of the alimentary canal and especially the esophagus, stomach, and duodenum—compare hematochezia.

- **me·le·nic** (mə-ˈlē-nik) adjective

- Correct term: Melenic stool

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- **Myth**: melanotic stools suggest GI bleeding.

- **Reality**:  
  - Melanotic stools indicate pigment in the GI tract.  
  - Melenic stools are a sign of GI bleeding.

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You have an ulcer.  
You need to give up coffee.
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Reality Check

- Coffee stimulates acid secretion
- Coffee can produce dyspepsia
  - By enhancing esophageal reflux
    - Caffeine is not the only variable
    - Decaffeination does not reduce these effects of coffee
- Despite increasing acid, there is no evidence that coffee consumption is a risk factor for ulcer disease

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GI Benefits of Coffee

- Prevents gallstone formation
- Improves constipation
- Reduces progression to cirrhosis in hepatitis C
- Protects against cirrhosis from alcohol
- Prevents liver cancer

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- **Myth:** coffee causes ulcers and is generally bad for the gastrointestinal system.
- **Reality:**
  - Coffee increases acid secretion and worsens GERD.
  - Coffee does not increase risks for peptic ulcer.
  - Coffee decreases risks for gallstones, constipation, cirrhosis and liver cancer.
You have an ulcer.

You have to give up alcohol.

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Alcohol and Upper GI Tract

- Alcohol stimulates acid secretion and enhances GERD
- High concentrations damage gastric mucosal barrier
  - Alcoholism associated with acute gastric mucosal lesions characterized by mucosal hemorrhages (gastritis)
- There is no evidence that moderate alcohol intake causes or exacerbates peptic ulcer disease
  - Modest alcohol consumption may even promote ulcer healing
- Alcohol abuse interferes with patient compliance
  - Intoxicated people don't follow orders as well as sober ones

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Myth: alcohol causes peptic ulcers.

Reality:
- Alcohol causes GERD but not ulcers
- Alcohol in moderation may protect against ulcers.
- In setting of abuse, can cause gastritis and impair compliance.
You have diverticula.

You have to give up nuts, popcorn, tomatoes and all foods containing seeds.

Reality

• Intuitively: seed or nut fragment might lodge in diverticulum and cause perforation
• Dietary histories taken at presentation with diverticulitis/bleed often reveal recent intake
• No data to support a causal role
  – Seeds and nuts not found in resection specimens
  – Primary process thought to be erosion of diverticular wall by increased intraluminal pressure or inspissated food particles.
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Data

• No support for role or seeds or nuts
  – No prospective studies indicate increased risk

• Prospective study: JAMA 2008
  – 47,000 men gave medical and dietary information
  – Followed for 18 years with repeatedly administered questionnaires
  – Results:
    * No increased risk of bleeding with more nuts, seeds and popcorn
    * The lower the intake of nuts and popcorn, the lower the risk of diverticulitis
    * These foods appear to be protective

• We should tell patients with diverticula the opposite:
  – Eat lots of fiber, fresh fruits and vegetables
  – The more nuts and popcorn the better!

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• Myth: seeds, nuts & popcorn trigger diverticulitis and diverticular bleeding.

• Reality:
  – No data indicate these foods are responsible for diverticular complications.
  – More likely they are protective, their consumption should be encouraged.

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Don’t take Tylenol
  if you have liver disease.
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Background

- Acetaminophen is not inherently toxic at usual doses
  - Up to 6 extra-strength Tylenol (500 mg)/day or 8 regular (325 mg)/day
  - Metabolism to completely non-toxic molecule is typical
- Acetaminophen is highly hepatotoxic in high doses
  - Accidentally: taking multiple meds, each containing acetaminophen
    - Cold medications, sleep aids, narcotic combinations (Percocet, Vicodin)
  - Overdose is most common cause of fulminant liver failure in US
  - Metabolism to toxic byproduct only occurs via alternate pathway

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Usual Dose

Toxicity

Usual Dose

Glucuronide + Sulfate

Non-toxic

Overdose

Cyto P450

NAPQI

Toxicity

Cyto P450

Glutathione

NAP-SH

Binds cell membranes

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Usual Dose

Toxicity

Usual Dose

Glucuronide + Sulfate

Non-toxic

Cyto P450

Alcohol, Certain Drugs

NAPQI

Toxicity

Glutathione

NAP-SH

Binds cell membranes
Is Tylenol Toxic In Liver Disease?

- With alcoholic liver disease
  - Potentially, if still actively drinking or malnourished

- With hepatitis B or C
  - No increased risk of toxicity

- With cirrhosis
  - No increased risk of toxicity
  - Potentially decreased risk of toxicity
    - Due to reduced levels of cytochrome P450 enzyme activity

Why Are Patients Told To Avoid It?

- To help clarify the diagnosis
  - Patient still getting worked-up for cause of elevated LFTs
  - Possibly taking more acetaminophen than aware/admits
  - Work-up for fulminant liver failure

- Medico-Legal
  - Most warnings come from pharmaceutical manufacturers
  - Little to gain from capturing the “liver disease market”
  - Much more to lose from potential lawsuits
Is Tylenol Safe For Liver Patients?

- Alternatives to acetaminophen?
  - NSAIDs, Aspirin
    - Gastric/intestinal ulceration, platelet dysfunction, nephrotoxicity
  - Narcotics
    - Impaired clearance, hepatic encephalopathy
- "Therefore, acetaminophen can be used safely in patients with liver disease and is a preferred analgesic/antipyretic because of the absence of the platelet impairment, gastrointestinal toxicity, and nephrotoxicity associated with nonsteroidal antiinflammatory drugs."

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Myth: Don’t use Tylenol in liver disease.

Reality:
- Acetaminophen may be safer in patients with liver disease than in those without liver disease.
- It is the safest of all analgesics in liver disease.
- Just don’t overdose it.

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Don’t take a statin if you have liver disease.
Are Statins Hepatotoxic?

- Mild elevations of ALT seen in 10% of patients on statins
  - Considered a "class effect"
  - Not considered indicative of liver damage or dysfunction

- ALT ↑ of > 3 x normal in 1-3% of patients on statins
  - RCTs: Mevacor, Zocor, Pravachol, Lipitor: no different from placebo
  - When statin continued, ALT generally returns to normal

- Statin therapy has been associated with liver failure
  - Idiosyncratic reactions, not class effect
  - 51,000 liver transplants in US 1990-2002: 3 were attributed to statins
  - Estimated rate of liver failure from statins: 1-2 per million
  - Much higher incidence with many other drugs (eg. Augmentin)

Statin in Liver Disease

- Chronic liver disease or cirrhosis does not contraindicate statins

- Statins may help some liver diseases
  - Non-alcoholic fatty liver disease
  - Hepatitis C

- Should LFTs even be monitored on statins?
  - Financial cost of lab tests alone estimated at $3 billion/year
  - Health costs of discontinuing statins: increased cardiovascular disease
  - Expert panel of hepatologists recommends against monitoring of LFTs

- Why haven't black box warnings been changed?
  - FDA only changes them when requested by manufacturer

Myth: statins cause liver disease and should not be given to patients with liver problems.

Reality:
- Mildly ↑ LFTs due to statins don’t reflect liver injury.
- Serious liver injury, if it occurs, is exceptionally rare.
- Pre-existing liver disease not contraindication to statin.
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A patient with an ICD has a polyp and electrocautery is required.

Place a magnet over the defibrillator to protect it.

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Reality

• No reported cases of endoscopic cautery causing device injury
  • Cases in literature: Bovie cautery, during thoracic surgery

• Magnet placed over defibrillator does not protect it
  • Magnet reprograms device, or turns it off
    • Effect depends on specific model
    • May not turn back on again after magnet removed

• Magnet may prevent accidental shock from defibrillator
  • If device misinterprets electricity from cautery as VF

• ICD needs 3-5 seconds of VF to detect and charge up
  • Few endoscopists hold the pedal down > 1 second at a time

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• Myth: put a magnet over an implanted defibrillator to protect it from endocautery.

• Reality:
  – Magnet may turn off ICD, leaving patient vulnerable.
  – Cautery bursts < 2 seconds should not trigger shock.
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A patient with a pacemaker has a polyp and endoscopic cautery is required for removal.

Place a magnet over the defibrillator to protect it.

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Reality

- Magnet placed over pacemaker does not protect it
  - Magnet reprograms device
    - Generally puts it into “asynchronous” mode
    - Pacemaker fires at baseline rate, not waiting for pause or atrial trigger
  - Magnet prevents pauses during prolonged cautery
    - In OR, surgeons cauterize for long periods
    - Few endoscopists hold down pedal for longer than a second
  - Unless endoscopist is leadfoot, pacing should not be impaired

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- Myth: putting a magnet over a pacemaker is necessary when endocautery is used.

- Reality:
  - Unless the endoscopist stands on the pedal, the pacemaker should not be compromised.
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- **Myth:** Endoscopically-applied electrocautery poses a significant risk of damage to implanted pacemakers and defibrillators.

- **Reality:**
  - No published reports of damage to devices by cautery during endoscopic procedures.

- **Separate Reality:**
  - There are lawyers out there waiting for this
  - Probably best to tell your patient to get device interrogated by cardiologist within a week.

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You should receive antibiotics before endoscopy if you've had a hip replacement.

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**Background**

- Bacteremia can infect implanted devices
- Prosthetic joint infections can be devastating
- Joint infections after dental procedures reported
- Antibiotic prophylaxis to prevent joint infections (?)
- Bacteremia can occur during endoscopy
- Orthopedists recommend giving antibiotics
Activities Causing Bacteremia

Rates of bacteremia:
- Brushing/flossing teeth: 20-68%
- Using toothpicks: 20-40%
- Chewing food: 7-12%
- GI Endoscopy: 5-10%

Blood from stomach & intestines is filtered by liver and spleen before entering the systemic veins.

Endoscopy and Joint Infection

- Entire medical literature: 2 case reports
- First patient (1990): esophageal cancer
  - Got 3 sessions of YAG laser Rx to esophagus
  - Known high risk for bacteremia: 40% vs. 5-10%
- Second patient (1994): colonoscopy + EGD
  - Acute B-cell lymphoma
  - On chemotherapy
  - Common variable immunodeficiency
  - Splenectomy
  - Only 5 months s/p THR

Reality

- Orthopedists recommendation
  - AAOS – “consider” antibiotics for a long list of procedures
  - One of which is GI endoscopy
- Other Societies
  - IDSA (Infectious Disease specialists) member survey –
    - Half would never give prophylactic antibiotics for endoscopy
  - Half would only give Rx during first 6 months following joint surgery
  - American Society of Colon & Rectal Surgeons – Not recommended
  - ASGE – Not recommended
- Incidence of C. Difficile in USA is at highest ever
  - Added healthcare cost > $1 billion
  - Mortality from C. diff – 14,000 deaths in last year
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• **Myth**: routine endoscopy poses credible risk for infecting prosthetic joints.

• **Reality**:  
  – Only 2 cases of joint infection ever reported.  
  – 14,000 deaths from C. difficile last year alone.  
  – ASGE, ASCRS and IDSA oppose antibiotic use for prophylaxis of joints during endoscopy.

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Amylase is 5,000 lipase 10,000.

This is severe pancreatitis.

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Reality

• Amylase, lipase levels **diagnose** pancreatitis  
  – Higher levels make the diagnosis more certain

• **Degree of elevation has no prognostic value**

• **Prognostic models for pancreatitis**  
  – Scoring: Ranson, APACHE II, SIRS, BISAP, CT  
  – Other labs: Hct, CRP, BUN  
  – No scoring system actually includes serum measurement of either amylase or lipase.
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• Myth: higher amylase or lipase signifies more severe pancreatitis.

• Reality:  
  – Amylase and lipase are useful only for diagnosis.  
  – Their degree of elevation is irrelevant to prognosis.

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A patient has pancreatitis and is kept NPO.

We should start TPN.

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Reality

• Problem
  – Pancreatitis patients are usually unable to eat
  – Food in stomach or duodenum stimulates pancreatic secretion
  – Concept of “putting pancreas to rest”
  – Catabolic state: malnutrition can complicate management

• More Problems
  – Patients with pancreatitis at high risk of infection
  – TPN associated with increased risk of infection
  – Catheter site infections
  – Risk of sepsis leading to bacterial translocation

• Potential Solutions
  – Most patients with mild or moderate pancreatitis can eat in a week
  – Nutritional support not necessary in these
  – Enteral feeding feasible in most moderate/severe pancreatitis
  – Nasojejunal feeding tube placed beyond the ligament of Treitz
Randomized Trial Results

- Fifteen RCTs of nutrition in pancreatitis
  - Enteral nutrition versus none:
    - No change in infectious risks
    - Significantly lower mortality in enterally fed
  - Parenteral versus none:
    - No change in infectious risks
    - Significantly lower mortality in TPN group than no nutrition
  - Parenteral versus enteral:
    - Lower mortality in enteral feeding group
    - Much lower infection rate in enteral feeding group

- Conclusion: Enteral feeding better than TPN

Largest RCT of TPN - Critically Ill

- Early versus late initiation of TPN
  - Over 4,500 patients, randomized
    - Half began TPN < 2 days, other half > 8 days
    - Early group spent more time in ICU, on mechanical ventilation and had longer hospital stays

- Conclusion: Hold the TPN, or at least start it later

Myth: pancreatitis patients should start on TPN.

Reality:
- Most of these patients need no nutritional support.
- Enteral feeding superior to TPN, even in pancreatitis.
- TPN should only be considered as a last resort.
A cirrhotic has altered mental status.

Serum ammonia should be checked to see if this is encephalopathy.

Hepatic Encephalopathy

- Toxins absorbed from the gut not adequately cleared by liver
- Result in altered brain function
- Ammonia is one of those toxins
- Elevated levels generally seen in hepatic encephalopathy
- Ammonia not clearly cause of encephalopathy
- Altered amino acids in brain – mimic neurotransmitters
- Brain edema due to glutamate
- Infusion of NH₃ in cirrhotic patients does not induce encephalopathy
- Serum measurement of ammonia
  - Venous levels can be unreliable
  - More reproducible
    - Arterial NH₃ more reproducible, but needs art line or arterial stick

NH₃ in Liver Disease

- Ammonia levels correlate only loosely with encephalopathy
- High ammonia levels common in cirrhotic patients
  - 40% of patient without encephalopathy had high levels
  - Many grade 3-4 encephalopathy with normal NH₃ levels
  - Large overlap between NH₃ levels and clinical findings
- Even when measured correctly, not sensitive or specific
- “Reliance on ammonia levels to make the diagnosis of hepatic encephalopathy is inappropriate and perhaps dangerous if it results in failure to see other causes of altered mentation in patients with chronic liver disease.”
- More reliable than serum ammonia – examine for asterixis
Myth: an elevated serum ammonia level is diagnostic for hepatic encephalopathy.

Reality:
- Many cirrhotics have high NH$_3$ without encephalopathy.
- Encephalopathy can be present without elevated NH$_3$.
- Clinical assessment for asterixis is more reliable.

I have abdominal pain.

My doctor says it’s due to gastritis.

Facts About “Gastritis”

- Gastritis and gastropathy: primarily histological diagnoses
- Found on most biopsies from the stomach (gastritis or gastropathy)
- Mucosa most often grossly normal
- Patient usually asymptomatic
- Caused by H. pylori, NSAIDs, alcoholism or autoimmune
- In many cases, no specific cause for inflammation identified
- Even when cause known, usually no correlation with symptoms
- Rare cases where gastritis is symptomatic
- Acute infection with H. pylori
- Severe alcoholic gastritis (when bleeding)
- H. pylori-associated with peptic ulcer disease (or gastric cancers)
- ? Bleeding from NSAID erosions
Effect of “Regression to the Mean”

Does Treating Gastritis Help?

- Sometimes patients get better with treatment
  - Placebo effect, or “regression to the mean.”
- Randomized trials
  - H. pylori eradication: No difference in treated vs. placebo
  - H2 Blockers, PPIs: Some improvement for treated
    - May merely represent response of GERD

Myth: gastritis caused my abdominal pain.

Reality:
- My doctor is not really sure what caused my abdominal pain.
What We Know Is True.....

“Half of what we are about to teach you is untrue. Unfortunately, we don’t know which half.”