Vesalius SCALpe™: Stomach, small intestine, appendix (see also: abdominal folios)

Secretions

endocrine
gastrin: antral G cells
trophic effect on mucosal cells -> hyperplasia
2 forms: big (G34) and little (G17)
   G34 lo potency, longer half life, active in basal acid output (BAO)
   G17 hi potency, primarily during stimulated acid output
   90% of gastrin produced is G17
somatostatin: D cells body and antrum
   stimulated by antral acid
intrinsic factor
histamine: enterochromaffin-like (ECL) cells: fundus and body
acid (parietal cell), pepsinogen (chief cell): fundus and body, acid converts pepsinogen to pepsin
mucous
gel of water and glycoprotein
mechanical barrier
   impede ion/acid movement into mucosa
increased production stimulated by: vagus, cholinergic, prostaglandins
decreased production: anticholinergics, NSAIDs, h. pylori
mucosal barrier: mucous, HCO₃⁻, motility

stimulation of acid production by parietal cells
3 receptors: histamine, ACH, gastrin: stimulate common proton pump
   exchange H⁺ for K⁺
neural: vagus, ACH
humoral: gastrin, histamine
synergy: acid production amplified if more than one receptor occupied
blockade of one makes others much less responsive (vagotomy decreases gastrin-stimulated acid production)

two types of acid secretion: basal and stimulated
BAO
   ~2mEq/h, 10% of max
   vagal tone, ambient gastrin and histamine
vagotomy decreases BAO 75-90%
H₂ blockade decreases BAO 80-90%
stimulated
cephalic, gastric, intestinal phases
   concurrent, not necessarily sequential
cephalic phase
      smell etc. -> vagal stim -> ACH
      inhibited by vagotomy or atropine
gastric phase
  food -> gastrin, histamine
  protein/aa’s stimulate gastrin production
  alkalization stimulates gastrin
  mechanical distention causes vagal stimulation
inhibition
  vagotomy
  antrectomy: eliminates G cell mass
  antral acidification stimulates somatostatin release which inhibits G cell
  release of gastrin
increased gastrin: ZE, G-cell hyperplasia, retained antrum
  bombesin stimulates release of antral gastrin, not from ZE
MEN I with ZE and hyperpara, treat hyperpara first
intestinal phase
  chyme entering duodenum, completes small particle digestion
  elicits small acid secretory response
  duodenal acidification -> secretin -> block gastrin receptors
motility: solid emptying linear, liquid exponential, proportional to volume
  ZE speeds emptying

Duodenal ulcer

decreasing since 60s, before H2 blockers, M now = F
incidence of DU 10y younger than GU
h. pylori
  abnormal acid secretion, barrier breakdown, smoking 2X risk
  Gm- rod, slow growing, converts urea to ammonia, produce urease
  oral-fecal transmission, humans primary reservoir
  >50% of world pop infected/colonized (30% US)
  most asymptomatic, nearly all have antral gastritis
more common: old, Hispanic, African, lower social status
  found in 90% of DU, 80% of GU & chronic atrophic gastritis
5-7X increased risk PUD, recurrence markedly reduced by elimination
2-6X risk gastric adenoCa, lymphoma, MALT
h. pylori organisms protected by mucous in antrum (pH 7 beneath mucous)
increased gastrin, decreased somatostatin breaks down mucin
active Dx: urea breath test: 13C-labeled urea ingested, urease from h. pylori splits
off C, detected in breath
  endoscopic bx, clo test
  stool antigen, indicates active infection
passive: serologic, saliva or urine for IgG antibody to h. pylori (can’t differentiate
  past from active infection)
endoscopic bx, examine for organisms
DU greater acid than normal, increased parietal cell mass, increased sensitivity to
stimulation
loss of inhibitory signals, decreased sensitivity to antral acidification, decreased D cells
& somatostatin, decreased negative feedback

NSAIDS: 2nd most common cause, 10-20X risk with daily use
associated risks: elderly, co-morbid conditions, h. pylori infection
damages mucosal barrier, decreases mucosal prostaglandins

ulcers 1-2 cm from pylorus rarely malignant
more distal ulcers: ZE, malignancy, drug-induced, Crohn’s

most gastrinomas in duodenum; pancreas 2nd most common (gastrinoma triangle)
other causes of elevated gastrin: renal failure, post antrectomy retained antrum, short
bowel syndrome (decreased gastrin metabolism), vagotomy (alkaline antrum
causes G cell hyperplasia)

**DU treatment**

eradicate h. pylori, rarely need chronic Rx
20-40% recurrence when Rx with PPI alone
triple Rx: PPI (omeprazole), amoxicillin/clarithromycin
failure due to non-compliance
cures 90%, reinfection 0.5%/y

surgical indications (limited)
medical intractability, complications: perforation, obstruction, bleeding (more
common complication than perforation)
paradoxical aciduria: late gastric outlet obstruction -> hypovolemia
kneys conserve Na, excrete H & K
vagotomy decreases stimulation of parietal cells
incomplete vagotomy most likely cause of recurrent ulcer
G cell hyperplasia post vagotomy, but does not cause acid hypersecretion
antrectomy decreases antral gastrin secretion
truncal vagotomy eliminate vagal stimulation down to right colon
20% gastric outlet problems without drainage procedure
parietal cell vagotomy less complications, 2% recurrence
perforation:
shock, > than 24h patch only
selective non-op management (stable, no peritonitis, contained)

chronic pyloric stricture
dilatation works short-term
truncal vagotomy and antrectomy if necessary
difficult duodenum vagotomy and gastro-J

**bleeding**
resuscitate IV, RBC, warm lavage (cold inhibits clotting), EGD
endoscopy > 75% success stopping bleeding
visible vessel high rate of rebleeding
surgery
shock, repeat bleed, > 6U/24h, recurrent bleed, readmission
duodenal cancer assoc w FAP/Gardner’s, VonRecklinhausen
tricobezoar surgery, phytobezoar enzyme Rx


**Duodenum, polyp/cancer**

polyposis

FAP: multiple, 300X risk duodenal Ca
duodenal Ca is the second most common malignancy in FAP, associated death
despite proctocolectomy
complete clearance of duodenal polyps in FAP requires resection, scope
surveillance not effective
isolated villous adenoma usually around ampulla

**Gastric ulcer**

incidence stable, older 55-65, mechanism unclear
risks: NSAID/ASA, alcohol, tobacco, h. pylori (in 80%), chronic and atrophic gastritis,
presence of acid
NSAID inhibition of prostaglandin alters mucosal barrier
normal acid secretion v DU hypersecretion
types (Johnson classification):

1 anywhere in body proximal to antrum, lesser curve, normal acid secretion, 60% of GUs , (association with blood type A)
2 body plus DU (subset of DUs), high acid, 20%, decreasing incidence
3 pre-pyloric/channel, (subset of DUs), high acid, 20%
4 high lesser curve, adjacent to GE jct, subtype of 1, lo acid, <10%, difficult to treat
5 drug-related (ASA, NSAID), anywhere in stomach, Rx stop drug

triple therapy like DU, EGD & multiple bx perimeter and base to R/O malignancy
increased chance of malignancy with large (10%) medical Rx highly successful (90%), need for surgery < 10%
rescope 6w after completion of medical Rx looking for healing (malig ulc can also show signs of healing)
surgical indications: giant ulcer (>3cm), < 50% healing, non-compliance, early recurrence, complication (bleeding, perforation, obstruction)
type 1: antrectomy (removes major site of h. pylori colonization), Billroth I, no vagotomy (normal acid) (ulcer excision, parietal cell vagotomy?)
type 2: antrectomy with ulcer, Billroth I, vagotomy (parietal cell vagot?)
type 3: “ “ “ or II, no parietal cell vagotomy (outlet obstruction)
type 4: extended antrectomy with ulcer bx or excision, Billroth II

type 5: depending on location V&P or V&A
bleeding excise ulcer
perforation: pts usually older, sicker, higher mort (40% v 10% for DU)

controversy: resection or closure
distal gastrectomy + truncal vagotomy (type 2 and 3, hi acid)
biopsy ulcer if not resected, eliminate h. pylori
highly selective vagotomy not indicated
hypertrophic polyps in 50% of pts with h. pylori and atrophic gastritis
multiple polyps associated with increased cancer risk

6 April 2009
adenomatous polyps marker for whole stomach risk; remove all

**Acute gastritis** (acute gastric mucosal lesion [AGML]/stress ulcer/erosive gastritis/hemorrhagic gastritis, acute mucosal ischemia)

- multiple superficial erosions fundus, mostly body, bleeding
- acid and activated pepsin in the presence of hypoperfusion/mucosal ischemia
- pepsin is inactivated at pH4.5 (prior question of increased nosocomial pneumonia from gastric bacterial colonization with titrating gastric pH up, particularly with carafate, not supported by recent studies)
- stress: burn, trauma, prolonged ICU stay (@ 24-48h)
  - Cushing's: head trauma/surgery, single ulcer stomach or duodenum
  - Curlings: burn, gastric erosions frequently extending to duodenum, diffuse
- multiple factors: acid necessary, mucosal ischemia/loss of barrier, ischemia/reperfusion injury, injury at epithelial level

**Rx**

- prevention: adequate resuscitation to prevent ischemia/reperfusion
- neutralize acid, H2 blockers highly effective, or pH titration (more difficult)
- reverse coagulopathy, maintain O2 sat, lavage
- endoscopy effective for discrete lesions
- persistent or diffuse bleed, next step angio/vasopressin via left gastric, embolize bleeding vessels, 80% effective
- if non-op measures fail, surgery: do less than total gastrectomy (40% mortality)
  - devascularize all but short gastrics, oversew, truncal vagotomy

**Dieulafoy’s lesion (described 1896)**

- tortuous submucosal artery with overlying ulceration (mechanical, no inflammation)
- not associated with h. pylori
- also found in esophagus, small intestine, colon and rectum
- Endoscopic Rx: hemoclip (most effective, also successful for colonic Dieulafoy’s ), heater probe, ethanolamine injection 78% permanent hemostasis
- angioembolization
- surgery last resort
- (bleeding scan not indicated for hematemesis, scope)

**Gastric malignancy**

- adeno 90%, lymphoma < 5%, sarcoma (GIST) 1-3%
- gastric adeno: 2nd leading cause of death worldwide; US decreased to ‘80s, increasing again most present as advanced
- increasing incidence proximal and GE jct, 50%, worse prognosis
- risks: h. pylori (distal lesions mostly), Asian/diet, chronic gastritis, adenomatous polyps, pernicious anemia, prior partial gastrectomy > 20y, Menetriers, family hx, smoking
Dx/staging:
- EGD/Bx,
- CT chest/abdomen/pelvis (good for bulky tumor and mets, poor for T and N staging)
- endoscopic ultrasound (EUS) best for T & N staging
- PET use evolving; laparoscopy best for Dx of peritoneal disease
- tumor depth correlates with nodal disease

Surgical Rx
- preop chemorads (5FU bolus) increases R0 (negative margin) resection rate (another source says it does not?)
  - (no change 5y survival with chemo/rad?)
- minimum of 15 nodes must be examined
- only 30% eligible for curative resection, staging laparoscopy IDs mets in 30%
- op mort directly related to extent of resection
- 5cm margin necessary (submucosal spread)
- resect directly involved adjacent organs (liver, spleen, pancreas), no prophylactic resection
- perigastric LNs along named vessels (celiac, common hepatic, splenic)
- resect greater and lesser omentum
- distal stomach: subtotal gastrectomy (total rarely necessary), Billroth II, loop with entero-enterostomy (Braun) or Roux-y limb > 50cm (prevent bile reflux)
- mid-stomach: total gastrectomy, Roux-y, D2 lymph node dissection
- proximal: total, distal esophagectomy (extended gastrectomy), Roux reconstruction

<table>
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<tr>
<td>IV</td>
<td>M1</td>
<td>3%</td>
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palliative
- metastasis or peritoneal seeding: no resection for cure, no change survival
- palliative surgery only for bleeding or obstruction refractory to other Rx
- Endoscopic laser, radiotherapy, stent, feeding tube
- resection preferable to bypass

GIST
- origin: intestinal pacemaker cells of Cajal
- most common GI tract sarcoma
- formerly called leiomyoma/leiomyosarcoma
- asymptomatic until grow large
- found anywhere in the GI tract, most common in stomach, unpredictable behavior,
bleeding common
malignant potential: size > 5-10 cm, mitotic index > 5-10m/10 HPFs;
less than these criteria < 2% recurrence, Gleevac not indicated
exceeding both criteria 85% recurrence v 10-15% if only one
metastasis only proof of malignancy: local recurrence, peritoneum, liver
hallmark C-Kit positive (membrane-bound tyrosine kinase receptor; receptor activating
mutation)(immunohistochemistry stain)
positive in 90-95%, predicts response to Gleevac
1-3% of gastric neoplasms, 50% of GISTS occur in stomach
occasional adjacent organ involvement, resection of adjacent involved structures may be
curative
malig spread hematogenously (not lymphatics) to liver, lung
complete wedge resection with 1cm margin depending on location
operate without biopsy
larger may require more formal gastrectomy
soft friable, take care not to rupture
rare LN met, lymph node dissection not necessary
Gleevac/imatinib: oral tyrosine kinase inhibitor
approved for recurrent and metastatic
not recommended for surgically resectable
outcome (likely to change with Gleevac use)
lo grade and < 5cm  90%  5y w resection alone
hi grade and > 5cm  30%  (50-60% if only hi grade or only > 5cm)
metastatic  <10%

Gastric carcinoid

1 sporadic: normal gastrin, large, solitary
can metastasize causing carcinoid syndrome (flushing, hypotension,
bronchoconstriction)
partial gastrectomy and LN dissection
2 with achlorhydria/pernicious anemia: increased gastrin, multiple small tumors in body
70% of gastric carcinoids
lo malignant potential
nonfunctional: gastric carcinoids produce little 5 hydroxytryptamine, but lack the
enzyme to convert to active serotonin, therefore serum serotonin and 5HIAA
are normal
treat increased gastrin with antrectomy, multiple <1cm regress
H2 blockers and PPIs have no role in treatment
3 associated with MEN1 & ZE: increased gastrin, multiple small carcinoids, low malig
potential
measure chromogranin A in all carcinoid pts, useful for monitoring
(small intestine most common site of carcinoid, 50% distal ileum, 70% metastatic at Dx)
(colonic carcinoid rare, usually large, R colon, older pt 60-70y)
(rectal carcinoids more commonly identified; syndrome rare with colonic, appendiceal,
rectal carcinoids)
Gastric lymphoma

MALT: mucosa-associated lymphoid tissue
associated with h. pylori, antigenic response to infection
monoclonal B cell proliferation
lo grade (can transform to hi grade), LN/marrow involvement rare
treat h. pylori with triple Rx gives 80% remission rate
focused wedge for hi-grade MALT
> 50% of all primary GI lymphomas occur in stomach, age > 50, increasing worldwide
most associated with systemic lymphoma
hi-grade lymphoma (non-h. pylori related)
resection, chemo, radiation all have roles
primary resection or as salvage after chemo
resect like adeno, submucosal spread, frozen section margin
local perigastric lymphadenectomy only
gastric lymphoma with extragastric disease chemo +/- RT, systemic Rx
survival
IE stomach only 90%
IIE-1 stomach and local LNs 82%
IIE-2 stomach and distal LNs 40%

Post-gastrectomy syndrome

occurs in 25% of gastrectomy patients, ~1% disabling
causes remain obscure
avoid reoperation, leads to more complications
only after all other approaches fail

Bariatric surgery

100lb over ideal weight, BMI > 35-40kg/m² (wt. kg/ht. in m²)
12X reduction life expectancy
consequences of morbid obesity:
  hypoxia from sleep apnea
  DVT from polycythemia
  pulmonary hypertension
  right heart failure
  diabetes
  GERD
BMI> 35Kg/M², metabolic syndrome= medical necessity
50% of morbidly obese have GERD, 20% severe; gastric bypass alone treats as effectively as
  Nissen in non-obese
Roux-Y gastric bypass most effective:15-30cc pouch, 75-150cm Roux limb
acute complication, leaking Roux-Y gastrojejunostomy most serious, <2% incidence
older, heavier (>50Kg/M²) with comorbid conditions higher risk
present as sepsis: early increased respiratory rate/respiratory failure, tachycardia (>120), hypotension; elevated WBC, oliguria late  
R/O PE, hypovolemia, bleeding  
gastrografin UGI, CT poor (22%) sensitivity  
reexplore for hemodynamic instability, resp failure  
primary repair often fails, leave large drains

metabolic consequences:
protein-caloric deficiency
anemia: Fe absorption requires acid and exposure to dietary Fe  
  absorbed mainly in duodenum and proximal jejunum  
B12: total body store 200 micrograms, daily need 2 mcg; deficiency late, anemia,  
glossitis, numbness, tingling hands and feet, mental status changes, check @  
  3-6mo, then Q6mo-1y
folate: anemia, rarer (folate absorbed proximal small intestine)
thiamine: absorbed duodenum and prox jejunum, deficiency results in beriberi  
cardiac: failure not reported in bariatric population  
neurologic: related to emesis; symmetric numbness, tingling ant thighs  
  progressing to bilat lower ext weakness/paralysis (axonal degeneration)
Ca\textsuperscript{++}: preferentially absorbed duodenum, prox jejunum; also D and other fat-soluble vits. (A,D,E,K)
laparoscopic gastric bypass higher incidence of small bowel obstruction  
  3 common sites  
  herniation through or scarring at mesocolon (retrocolic Roux-Y)  
  behind Roux limb (Petersen hernia)  
  jejun-jejunal anastomosis  
lower incidence with antecolic Roux limb  
symptoms (chronic abdominal, periumbilical pain) mandate exploration, 2% negative  
lap  
  most can be repaired laparoscopically  
vertical banded gastroplasty associated with severe reflux  
lap band: gastric prolapse most common complication  
  50% expected wt loss

**Small intestine**

Crohns: 50% I-C, 30% small bowel alone (highest recurrence), 20% colon alone  
  smoking detrimental (v. UC where may be beneficial)  
marked lymphangiectasia  
bloody diarrhea rare  
steroids for acute, not chronic; AZA, 6MP for chronic  
prednisone for small intestine, sulfasalazine for colon  
TPN maintains body mass, speeds closure of hi output fistula  
60% of small intestine tumors in ilium, 50% benign  
  obstruction, intussusception manifestations  
Peutz Jegher’s hamartomas low malignant potential: STK 11 mutation
canceroid: appendix most common, 75% near tip, 3% mets
ilium next, most likely to metastasize (35%)
rectum least common
bronchus, ovary
30% multiple or presence of another tumor
> 2cm -> 90% mets
cutaneous flush most common manifestation
anti-serotonin agents: methysergide, cyproheptadine, p-chlorophenylalanine,
somatostatin/octreotide
streptozotocin, 5FU may help
$^{99m}$Tc lights up gastric mucosa in Meckel’s
blind loop: stasis, bacterial overgrowth
pain, diarrhea, steatorrhea (bile salt deconjugation), amenorrhea, wt. loss
B12 breakdown by BT -> megaloblastic anemia
return of small intestinal motility post op (migrating motor complex/MMC) 6-24h
risk factors for small intestine cancer: celiac sprue, Crohn’s, FAP, Peutz-Jegher’s
(scleroderma not a risk factor)
celiac associations: lymphoma, esophageal, small bowel adenocarcinoma
heat does not trigger visceral pain

Appendix

emesis before pain with appendicitis
GALT (gut-associated lymphoid) tissue -> immunoglobulins, process thymic independent lymphocytes
appendicitis: young obstruction of lumen by lymphoid hypertrophy, older fecolith 30%
not more common in pregnancy, most 2nd trimester
open more wd. infection, laparoscopic more intraabdominal infection

Retroperitoneum

retroperitoneal fibrosis
2/3 idiopathic
drugs: methysergide, ergotamine, hydralazine, methyldopa, beta blockers
IVP best diagnostic test
mesenteric tumors: malignant at the root, benign at the periphery
locally aggressive
retroperitoneal sarcoma: 10% 5y survival
peritoneal dialysis can remove: NH3, Ca, Fe, Pb, Li

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Dhage-Ivatury S. Update on the surgical approach to mucocele of the appendix. JACS, 202(4), April '05: 680-684.