

## Vesalius SCALpel™ : 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,  $\text{HCO}_3^-$ , motility

stimulation of acid production by parietal cells

3 receptors: histamine, ACH, gastrin: stimulate common proton pump  
exchange  $\text{H}^+$  for  $\text{K}^+$

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

alkalinization 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: <sup>13</sup>C-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: 2<sup>nd</sup> 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 2<sup>nd</sup> 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  
kidneys 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  
gastric lymphoma: Rx surgical resection  
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 lookinf 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

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)

Cushings: 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 O<sub>2</sub> 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: 2<sup>nd</sup> 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 most 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

stage	TNM	5y survival
Ia	T1N0	60-95%
Ib	T1N1, T2N0	44%
II	T1N2, T2N1, T3N0	29%
III	T2N2, T3N1/2, T4N0	13%
IV	M1	3%

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/m<sup>2</sup> (wt. kg/ht. in m<sup>2</sup>)  
12X reduction life expectancy  
consequences of morbid obesity:  
hypoxia from sleep apnea  
DVT from polycythemia  
pulmonary hypertension  
right heart failure  
diabetes  
GERD  
BMI > 35Kg/M<sup>2</sup>, 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/M<sup>2</sup>) 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<sup>++</sup>: 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)

jejuno-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

carcinoid: 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  
<sup>99m</sup>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 2<sup>nd</sup> 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: NH<sub>3</sub>, Ca, Fe, Pb, Li

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