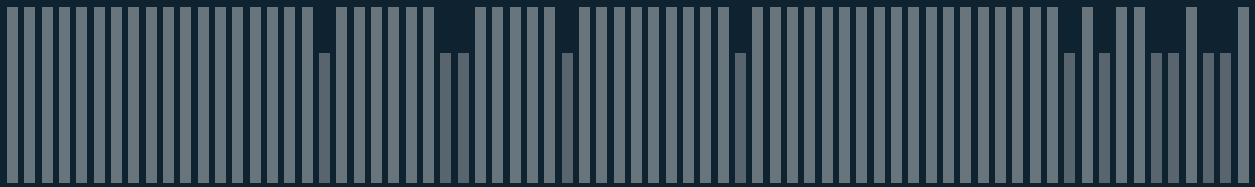


The ASCRS Textbook of Colon and Rectal Surgery

FOURTH EDITION



72 CHAPTERS • 210 PAGES

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INTRODUCTION

These are my own notes on the fourth edition of the ASCRS Textbook of Colon and Rectal Surgery.

I first made them as a fellow, working through the first edition chapter by chapter, and I updated them again when the second edition came out. This version follows the fourth edition's own structure, which has grown to seventy-two chapters. Where the new edition split a topic apart, I split my notes with it. Where it reversed something I had written down years ago, I changed the line rather than leaving the old one standing.

I still use these myself. I am on the teaching staff at Cedars-Sinai, and staying current in this field is not optional. The literature moves, guidelines change, and operations that were standard when I trained have quietly fallen out of favor. Reading a textbook cover to cover once every several years is not how anyone actually stays up to date. Working through it and condensing it is.

They are written for someone already trained, and they are terse on purpose. They are not a substitute for the textbook and they will not teach anyone colorectal surgery. What they will do is let you find the salient points of a chapter quickly — the numbers, the classifications, the decision rules, and the handful of trials that actually changed practice.

They are my notes, which means they carry my emphasis and, almost certainly, my typos and my errors. I have not proofread them the way a publisher would. Read them as a first draft and go back to the source when something matters.

I make them available at no charge. I have no reason to keep this to myself. If they help a resident, a fellow, or a colleague take better care of a patient, that is reason enough.

If you find an error, or you think I have gotten something wrong, please tell me.

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1 PERIOPERATIVE/ENDOSCOPY: ANATOMY AND

EMBRYOLOGY OF THE COLON, RECTUM, AND ANUS

ANAL CANAL

DEFINITIONS

Anatomic: dentate line → anal verge

Surgical (Milligan & Morgan): anorectal ring → anal verge

– IAS, EAS, puborectalis

- post. length 4.4 cm men, 4.0 cm women. Does NOT change w/ age
- MRI: no length difference by sex, but ant. & post. EAS shorter in women

“Tube within a tube”

- inner = IAS + longitudinal layer, autonomic
- outer = puborectalis + EAS, somatic – the conscious control

EPITHELIUM

Proximal canal pink, columnar rectal mucosa

Anal transition zone: 6-12 mm proximal to dentate, purple

- where most HPV-related dysplasia sits – cervix parallel

Columns of Morgagni, crypts at their base → the dentate line

Anal glands: avg 6 (range 3-12), concentrated posteriorly

- 2/3 enter the IAS, half end in the intersphincteric plane
- duct obstruction → abscess & fistula. Internal opening AT the dentate line

Anoderm: ~1.5 cm distal to dentate – squamous, NO hair, sebaceous or sweat glands

- at the verge: thickened, pigmented, hair-bearing = normal skin

Dentate line = true endoderm/ectoderm division

- above: autonomic, hypogastric vessels & lymphatics
- below: somatic, inferior hemorrhoidal system

INTERNAL ANAL SPHINCTER

Continuation of rectal circular smooth muscle, tonically contracted

Ends ~1 cm proximal to the distal EAS

~2 mm thick, 35 mm long. Volume equal by sex; women thicker medial/distal

CONJOINED LONGITUDINAL MUSCLE

0.5-2.0 mm, intersphincteric plane. Longitudinal rectal fibers + puborectalis fibers

Distal fibers = corrugator cutis ani → perianal skin & ischioanal fat

Fibers piercing the IAS into the subepithelial space = Treitz's muscle

- Thomson: disruption → cushion prolapse, outflow obstruction, hemorrhoidal bleeding
- Haas & Fox: limits incontinence after sphincterotomy

EXTERNAL ANAL SPHINCTER

Striated tube around IAS + CLM. Distal edge = intersphincteric groove

Milligan & Morgan's 3 divisions DISPROVEN by Goligher – one continuous sheet

- deep / superficial / subcutaneous persist as names only

Mid EAS → coccyx via anococcygeal lig. Proximal EAS continuous w/ puborectalis

- anteriorly forms part of the perineal body

Natural ant. defect in 75% of NULLIPAROUS women, below the puborectalis sling

- → overreporting of “obstetric defects” on isolated endoanal US
 - why anterior sphincterotomy is NOT done in women
- Inferior rectal br. of pudendal (S2,3) + perineal br. of S4
- contralateral overlap → reinnervation after unilateral injury

HEMORRHOIDS

Normal anatomy, present in the embryo. Function = continence

3 parts: lining, stroma, anchoring tissue to IAS & CLM

Terminal branches of the superior hemorrhoidal a. –

classic RA/RP/LL positions DISPUTED

A-V shunts, “corpus cavernosum recti” (Steltzner) → pulsatile bleeding at hemorrhoidectomy

PELVIC FLOOR

Perineal body = EAS + superficial & deep transverse perinei + bulbospongiosus

- “EAS complex” – purse-string, not donut
- MUST repair the perineal body during overlapping sphincteroplasty

Levator ani = pubococcygeus, puborectalis, iliococcygeus

- LA injury in 55% of women w/ prolapse vs. 16% without
- puborectalis IS part of the LA – shares a primordium w/ ICM & PCM
- perineal & inf. rectal br. of pudendal + direct S3/S4 (levator ani n.)
- PCM & PRM mainly pudendal; ICM mainly direct sacral

Puborectalis:

- pubis & UG diaphragm fascia → sling behind the anorectal junction
- anorectal ring = upper IAS + puborectalis
- horizontal force, ↓ anorectal angle – MOST important factor in gross continence

Iliococcygeus:

- ischial spine & obturator fascia → distal sacrum, coccyx, anococcygeal raphe. Lifts

Pubococcygeus:

- obturator fascia & high post. pubis → anococcygeal raphe, sacrum, coccyx tip
- forms the levator hiatus. Hiatal lig. suspends the intrahiatal organs
- hiatal enlargement → prolapse. This is the part torn in traumatic vaginal delivery

RECTUM

DIVISIONS

From anal verge: lower 0-7, middle 7-12, upper 12-15 cm.
May extend past 15

Upper rectum vs. sigmoid: NO taenia coli, NO appendices epiploicae

Ant. peritoneal reflection ~9 cm F, 9.7 cm M – no significant sex difference

- highly variable, altered by dz such as prolapse

FASCIA

Mesorectum: a surgeon's term, not in the Nomina Anatomica

- contained by the fascia propria – NOT a barrier to infection or CA spread
- incomplete excision → ↑ local recurrence

Presacral fascia: over sacrum, covers presacral veins & hypogastric nerves

- elevate the fascia propria OFF it – leaving it intact prevents presacral bleeding

Retrosacral fascia: S3-S4 → fascia propria 3-5 cm proximal to the anorectal junction

- MUST be sharply incised in TME. Behind it = supralelevator / retrorectal space

Waldeyer's: ambiguous eponym – presacral, retrorectal, or both

Denonvilliers': fusion of the 2 walls of the embryologic cul-de-sac

- rectovesical pouch → pelvic floor. In women = rectovaginal septum; rectocele = its defect
- 2 microscopic layers, NOT separable at dissection
- cavernous nerves at its anterolateral border. Fascia propria lies DORSAL to it

LATERAL LIGAMENTS

Trapezoid, mesorectum → endopelvic fascia at the midrectum. Include in the TME specimen

Church: primarily NERVES and connective tissue – middle rectal a. usually NOT in them

- MRA in only 22% of specimens in one cadaveric series
- Miles: "seldom require a ligature." Heald never mentions them

VALVES OF HOUSTON

Classic 3 valves in only 45.5%; 32.5% two; 10.25% four
Kohlrusch's = middle, on the RIGHT, 9-11 cm, largest

- proposed intraluminal marker for the ant. peritoneal reflection

ANORECTAL SPACES

Perianal – ext. cushions, subQ EAS, distal IAS. Cephalad limit = dentate line

Intersphincteric – where cryptoglandular sepsis begins

Submucous – proximal to dentate, the internal cushions

Ischioanal – LARGEST. Pyramid, apex at levator insertion on obturator fascia

- fat, pudendal branches, superficial internal pudendal vessels

Superficial postanal – anococcygeal lig. to skin

Deep postanal (retrosphincteric space of Courtney) – levator to anococcygeal raphe

- connects the two ischioanal spaces → HORSESHOE abscess

Supralelevator – peritoneum above, pelvic wall lateral, rectum medial, levator below

Retrorectal – presacral fascia to fascia propria. No major vessels or nerves

BLOOD, LYMPH & NERVES

Superior rectal a.: IMA continuation past the left iliacs. UNPAIRED

- bifurcates in 80%; ~8 terminal branches in the distal rectal wall

Middle rectal a.: paired, present only 12-28% (one series 91%)

- from the ant. division of the internal iliac or the pudendal

Inferior rectal a.: paired, from internal pudendal in Alcock's canal. Entirely EXTRApelvic

Venous: middle & inferior → systemic via internal iliac; superior → portal via IMV

- the 2 systems explain rectal varices in portal HTN

Lymph: upper 2/3 → IM then para-aortic. Lower 1/3 → along SRA, laterally along MRA to internal iliac

- above dentate → IM & internal iliac. BELOW dentate → superficial inguinal

Sympathetics L1-3 → preaortic plexus → 2 hypogastric nerves → pelvic plexus

- pelvic plexus on the sidewall at the lower 1/3 of the rectum

Parasympathetics S2-4 = nervi erigentes, join the hypogastrics at the pelvic plexus

Periprostatic plexus = subdivision of the pelvic plexus

IAS: sympathetic L5, para S2-4. Anal sensation = inf. rectal br. of pudendal

COLON

GROSS

~150 cm. Cecum ~7 cm diameter, sigmoid ~2.5 cm – caliber ↓ proximal to distal

3 differentiators from small bowel: appendices epiploicae, taeniae coli, haustra

- libera (ant.), mesocolica (posteromedial), omentalis (posterolateral)
- outer longitudinal layer IS circumferentially complete, just thicker at the taeniae
- appendiceal base → rectosigmoid junction, where they converge
- haustra septated by the plicae semilunares

Cecum ~8 cm long, 7 cm wide. Competent ICV → closed-loop obstruction

Appendix: orifice 3-4 cm from ICV, 2-20 cm long, 5 mm caliber

Ascending 15-18, transverse 45-50, descending 25, sigmoid 35-45 cm

White line of Toldt = lateral peritoneal reflection, the mobilization guide

Rectosigmoid junction: NARROWEST large bowel, 2-2.5 cm

BLOOD SUPPLY

Watershed just proximal to the splenic flexure = midgut / hindgut border

True right colic ABSENT in up to 20% – may come off ileocolic or middle colic

Middle colic off the SMA at the inferior border of the pancreas

- left branch is the SOLE splenic flexure supply in up to 33%
- accessory middle colic a. in 36.4%, 85% off the SMA

IMA: 3-4 cm above the aortic bifurcation, L2-L3

- left colic, sigmoidals, then superior rectal over the left common iliac

Marginal a. of Drummond – discontinuous or absent in some

- Griffiths' critical point (splenic flexure): absent in up to 50%
- Sudeck's critical point (rectosigmoid): doubted, but a fluorescence study supports it

IMV does NOT follow its artery – lateral to the ligament of Treitz to the splenic vein

- dissect posterior to it for the medial-to-lateral plane

LYMPH & NERVES

Epiploic → paracolic (MOST filtering) → intermediate → principal → cisterna chyli

Sympathetic: right colon lower 6 thoracic splanchnics; left colon L1-3

Parasympathetic: right colon posterior (right) vagus; left colon S2-4

EMBRYOLOGY

Hindgut = distal to splenic flexure incl. canal proximal to dentate → IMA

Midgut = below the pancreatic papilla to proximal 2/3 transverse → SMA

- vitelline duct to yolk sac; failure to obliterate = Meckel's

Dentate line = fusion plane of the endodermal & ectodermal tubes

Cloaca fuses w/ the proctodeum. Urorectal septum divides the cloacal plate by wk 8

Anal tubercles fuse by wk 10 – horseshoe dorsally, perineal body anteriorly

EAS forms BEFORE the IAS, from the posterior cloacal sphincter

- IAS from rectal circular muscle. IAS migrates caudad, EAS enlarges cephalad

MIDGUT ROTATION – 3 STAGES

1. Herniation, wks 6-8. 90° CCW, sagittal → horizontal. Anomalies rare

2. Return, wk 10. Additional 180° CCW – duodenum behind the SMA, colon anterior

- most anomalies arise here

3. Fixation – cecum to RLQ; duodenum, ascending & descending fuse at wks 12-13

Nonrotation: small bowel right, colon left. Volvulus at the DJ junction & midtransverse

Malrotation: cecum fails the full 270°, sits mid-upper abdomen. Ladd's bands → duodenal obstruction

Reversed rotation: clockwise – transverse colon POSTERIOR to SMA, duodenum anterior

Omphalocele: midgut retained in the umbilical sac, membrane-covered, defect > 4 cm

Internal hernia: failure of fixation – paraduodenal, paracecal, intersigmoid

OTHER MALFORMATIONS

Colon duplication: mesenteric cyst / diverticulum (heterotopic gastric or pancreatic) / long duplication (rarest, shares a wall)

Meckel's: antimesenteric ileum, w/in 50 cm of the ICV, up to 3% of the population

- band to umbilicus → obstruction; ectopic mucosa → bleed or perforate
- Littre's hernia = Meckel's in an indirect hernia
- NO hard indication to excise if incidental

Colonic atresia: only 5% of GI atresias. Vascular – membrane, cord, or absent segment

Hirschsprung's: absent myenteric ganglion cells, arrested neural crest migration. IAS ALWAYS involved

ANORECTAL MALFORMATIONS

Developmental arrest – “syndrome of caudal regression” (Duhamel)

Skeletal & urinary anomalies up to 70%. Also TE fistula, cardiac, abdominal wall

Anal stenosis: 25-39% of newborns, symptomatic in only 25% of those

- most dilate spontaneously in the first 3-6 months

Membranous atresia: “covered anus” – thin membrane over the blind canal

Anal agenesis: rectum to below the puborectalis, ends blind or as a perineal/vulvar/urethral fistula. Sphincter normally sited

Anorectal agenesis: MOST COMMON imperforate anus.

Anus = a dimple, sphincter normally sited, usually urethral or vaginal fistula

- high fistulae form wks 6-7; low (perineal) wks 8-9

Rectal atresia (“high”): rectum and canal separated by an atretic segment

Persistent cloaca: FEMALES only. Total failure of urorectal septal descent

2 PERIOPERATIVE/ENDOSCOPY: COLONIC PHYSIOLOGY

EMBRYOLOGY

Wks 3-4: primitive gut from folding of the endoderm-lined yolk sac

- endoderm → mucosa. Mesoderm → muscular wall, connective tissue, serosa

By wk 4, 3 regions defined BY BLOOD SUPPLY

- foregut (celiac) – distal esophagus, stomach, proximal duodenum
- midgut (SMA) – distal to the CBD in the 3rd part of duodenum → proximal 2/3 transverse
- hindgut (IMA) – distal transverse, descending, sigmoid, rectum
- vitelline duct fails to obliterate = Meckel's

Wk 5 elongation → physiologic herniation. Wk 6: 90° CCW around the SMA

Wk 10: return + a further 180° CCW. Cecum reenters LAST, RUQ → right iliac fossa

Loss of the dorsal mesentery fixes ascending & descending retroperitoneally

- cecum, transverse & sigmoid keep theirs – hence mobile

Cloaca divides at wk 6 into anterior urogenital, posterior anorectal, sphincteric

EAS forms wk 10 in the posterior cloaca. IAS wk 12 from rectal circular muscle

COLONIC WALL & EPITHELIUM

Fecal production ~128 g/day, ↑ w/ dietary fiber

- 25-54% of dry solids = BACTERIAL BIOMASS

4 layers: mucosa (epithelium, lamina propria, muscularis mucosae), submucosa, muscularis propria, serosa

- Meissner's submucosal plexus – ion transport
- Auerbach's myenteric plexus b/n circular & longitudinal – smooth muscle fxn
- longitudinal fibers concentrated as the 3 taeniae, fanning out at the rectum
- circular layer continuous cecum → anal canal, thickens into the IAS

Interstitial cells of Cajal: c-kit-positive mesenchymal, the PACEMAKER of the ENS

- link the submucosa electrochemically to the myenteric plexus
- cell of origin of GIST – arises from the WALL, not the mucosa

3 epithelial cell types:

- enterocytes (columnar, absorption & secretion) + goblet cells = ~95%
- neuroendocrine cells – chemoreceptors, initiate digestive & protective responses

Stem cells at the crypt base. Epithelium renewed every 4-5 days

- CRYPT: proliferative, undifferentiated, SECRETES chloride
- SURFACE: differentiated, low turnover, ABSORPTIVE

ABSORPTION & SECRETION

VOLUMES

1500-2000 mL/24h arrives from the ileum

- all but 100 mL absorbed, and all but 1 mEq of Na and Cl
- stool Na ~30 mmol/L, K ~75 mmol/L

Capacity ↑ to 5-6 L and 800-1000 mEq Na/Cl daily – compensates for small bowel failure

Proximal colon = storage, mixing, water & electrolyte absorption (Cannon, 1902)

Rectosigmoid = conduit, but can join the compensatory response

SODIUM

Active, against an electrochemical gradient. 90% absorbed in exchange for K secretion

Proximal colon: electroneutral, coupled luminal Na⁺/H⁺ and Cl⁻/HCO₃⁻ exchange

Distal colon: ENaC on the luminal side, Na⁺-K⁺-ATPase basolateral

- Cl⁻ absorbed via luminal CFTR

Na⁺-K⁺-ATPase: 3 Na out for 2 K in – net one positive charge across the basolateral membrane

C. difficile inhibits the Na⁺/H⁺ exchanger

IBD: BOTH electrogenic (channel) and electroneutral Na⁺/H⁺-coupled NaCl absorption reduced

DRA (downregulated in adenomas) = Cl⁻/OH⁻ exchange. Mutation → congenital chloride diarrhea

Cholera: toxin binds the crypt brush border → adenylyl cyclase → ↑ cAMP → Cl⁻ & HCO₃⁻ secretion → secretory diarrhea

POTASSIUM

Both membranes permeable to K. Luminal K ~ serum K (4-5 mEq/L). NET SECRETION

Prolonged diarrhea → HYPOKALEMIC METABOLIC ACIDOSIS

- K secretion + Cl⁻/HCO₃⁻ exchange also make stool water alkaline

ALDOSTERONE

↓ fecal Na 30 → 2 mEq/L, ↑ fecal K 75 → 150 mEq/L

Two mechanisms, two sites – conserve Na at the expense of K

- distal colon: activates Na⁺-K⁺-ATPase
- proximal colon: activates Na⁺/H⁺ exchange

WATER

Mucosal surface area ~2000 cm² – true absorptive area larger, crypts absorb as well as secrete

Stool water is HYPERTONIC to plasma, 350-400 mOsm/L

- bacterial solute production + relative impermeability of the colonic membrane

Passive movement along osmotic & hydrostatic gradients.

Absorption > secretion = net absorption

Adrenergic (α) or anticholinergic stimuli ↑ absorption

SHORT-CHAIN FATTY ACIDS

Proximal colon bacteria ferment carbohydrate → acetate, propionate, BUTYRATE

- butyrate = the main energy substrate of the colonic epithelium
- SCFA supply ~10% of daily caloric requirement. Up to 100 mMol produced luminally

Potent stimuli of Na and water absorption – butyrate the most effective

Fecal SCFA are REDUCED in acute diarrhea – may explain the impaired Na absorption

May ↓ inflammation in UC and diversion colitis. Butyrate hypothesized to ↓ CRC risk

VITAMIN K

Cannot be synthesized endogenously – diet plus colonic microbiota

- small bowel: carrier-mediated, energy-dependent
- colon: PASSIVE diffusion

INNERVATION

ENS = LARGEST single division of the ANS, 200-600 million neurons

Hirschsprung's: myenteric AND submucosal ganglia both absent → no relaxation, no peristalsis

Sympathetic – inhibitory:

- proximal colon from the superior mesenteric ganglion; distal from the inferior mesenteric
- distal rectum & anal canal from the hypogastric plexus
- tonic inhibition of secretion via α -2 receptors on submucosal secretomotor neurons
- contracts sphincters, inhibits enteric neurons – both DELAY transit

Parasympathetic – excitatory:

- vagus → cecum, ascending, transverse
- pelvic nerves S2-4 → descending, sigmoid, rectum
 - enter near the rectosigmoid junction, project BOTH orally and aborally in the myenteric plane
- damage to excitatory pathways → severe constipation

EAS is striated – somatic pudendal, transmission by ACh

Mediators:

- excitatory: ACh, tachykinins (substance P)
- inhibitory: NO, VIP, possibly ATP

PAIN

Rectal pain travels PELVIC pathways

Sensory neurons run 3 CNS pathways: vagal, thoracolumbar, lumbosacral

Distending the descending or sigmoid colon = cramping, gas or pressure in the lower abdomen, low back, or perineum

Inflammation changes both the neurons and the ascending pathway used – post-infectious IBS

IBS = brain-gut axis dysregulation, altered visceral sensitivity

- ↓ epithelial serotonin reuptake transporter (SERT) expression – significance unknown

MOTILITY

Propulsion, accommodation, storage, and rapid emptying of a variable segment

Tone accommodates, stores and distributes. Diurnal variation

- ↑ slowly after a meal, ↓ during sleep, ↑ DRAMATICALLY on waking

HAPCs: most frequent in the morning, postprandially, and just before defecation

Gastrocolic response: begins within minutes, lasts up to 3 hours

- fat and carbohydrate STIMULATE. Amino acids and protein INHIBIT

Tone is NOT slow-wave driven – actin-myosin, neurogenic & mechanical modulation

- narrows the lumen → makes phasic contractions more efficient
- sets wall tension, so it determines the perception of distension

Colonic motor complexes: 1-2 per hour, ~6 minutes each

Rectal motor complexes: NOT synchronized with small bowel MMCs. Function unclear

DEFECATION

Rectal afferents sense filling → rectoanal inhibitory reflex

- IAS RELAXES, EAS CONTRACTS – the moment of choice

To proceed: puborectalis & EAS relax → straighten the anorectal angle, open the canal

Propulsion from rectosigmoid + diaphragm + abdominal wall

IAS = 80% of resting anal tone, sympathetic. EAS & floor = sacral roots and pudendal

MODULATORS OF MOTILITY

Cholinesterase inhibitors (neostigmine) ↑ motility

Clonidine (α -2 agonist): ↓ tone, ↓ phasic activity, ↓

perception of distension, ↑ compliance – for IBS-D

- yohimbine (α -2 antagonist) does the opposite, and promotes fluid & electrolyte absorption

Alosetron (5-HT₃ antagonist): ↑ compliance, delays transit, ↓ rectal sensitivity. Approved for IBS-D in women

- ischemic colitis 0.15% vs. 0.0% placebo → WITHDRAWN

Prucalopride (5-HT₄ agonist): FDA approved for chronic constipation. NO Q-T effect

Opioids: δ , μ , κ – GI effects are μ . Morphine ↑ phasic segmental activity, ↓ fasting tone, blunts the gastrocolic response

- opioid-induced constipation in 41-81% of treated pts

Lubiprostone: PGE₁-derived, activates apical ClC-2 + EP receptors + CFTR → fluid secretion

- accelerates transit but does NOT change colonic motor activity. Chronic constipation & female IBS-C

Bile acids: 5 mmol/L stimulates phasic contractions – not reached unless ileal resection

- chenodeoxycholic acid at physiologic dose → propagated contractions, ↑ rectal sensitivity
- ileal dz, resection or radiation → BA spill into the colon → diarrhea
- delayed-release CDCA accelerates transit in women w/ IBS-C

Laxatives:

- osmotic (PEG, Mg citrate, Na phosphate, lactulose, sorbitol) – hypertonic, pull fluid in
- stimulant (bisacodyl, Na picosulfate, glycerol) – trigger HAPC sequences → mass movement
 - bisacodyl & picosulfate also anti-absorptive and secretory
 - bisacodyl works via MUCOSAL AFFERENTS – blocked by topical lidocaine

Sacral nerve stimulation:

- FDA approved for fecal incontinence. Role in constipation UNCLEAR
- S3 stimulation induces propulsion throughout the colon, ↑ stool frequency in slow transit
- Kamm: of 27 implanted pts, 20 had delayed transit before and only 9 after

MICROBIOME

~110 trillion bacteria in the adult intestine. > 700 volatile organic compounds in feces

Firmicutes + Bacteroidetes = > 98% of gut bacteria

Established after birth; modifiable by diet, illness, antibiotics, but resistant to change later

Composition altered in IBS, IBD, colorectal CA, autism

- adjusted for diet and transit, the microbiome separated health from constipation w/ 92% accuracy

Crohn's: LOWER overall diversity. ↑ Proteobacteria and Bacteroidetes vs. controls and UC

UC: low or absent Faecalibacterium prausnitzii. Roseburia ↓ in IBD generally

Ciprofloxacin: ↓ diversity, richness and evenness – ~1/3 of species change in abundance

- most rebound, but may take up to 6 months
- broad-spectrum ANTI-ANAEROBIC agents cause the longest-lasting shift

3 PERIOPERATIVE/ENDOSCOPY: ANORECTAL

PHYSIOLOGY

APPLIED ANATOMY

Rectum = the reservoir. 12-15 cm. Proximal & distal margins still debated

- identified in the abdomen by the ABSENCE of haustra, taeniae, epiplocae
- becomes the anal canal as it passes through the levators

Valves of Houston: do NOT contain all wall layers, no known function

- middle valve = the ant. peritoneal reflection, most consistent in presence & location

Embryologic canal: anal verge → dentate line

Surgical / functional canal (Milligan & Morgan, 1934): anal verge → anorectal ring

- 2.5-5 cm, SHORTER in females

Columnar above the dentate, squamous below – 2

- separate vascular, lymphatic & nerve inputs
- above: hypogastric vessels, autonomic. Below: inferior hemorrhoidal, somatic
- transition (cloacogenic) zone = the 1-2 cm b/n them

SPHINCTER COMPLEX

IAS: 2-3 mm circular band, distal continuation of the rectal circular smooth muscle

- MAXIMALLY CONTRACTED at all times
- levator ani proximally, superficial & subQ EAS distally

CLM: b/n IAS and EAS. Outer rectal fibers from the anorectal ring → puborectalis

- contribution to continence minimal – acts as the SCAFFOLD of the sphincter complex

EAS: striated, continuous w/ puborectalis. Perineal body anteriorly, anococcygeal lig. posteriorly

- tonically active even at rest, voluntary contraction on threat of incontinence

Levator ani:

- pubococcygeus: posterior pubis → anococcygeal lig. & coccyx
- puborectalis: U-shaped sling to the posterior pubis. Palpable as the TOP of the anorectal ring
- iliococcygeus: ischial spine & obturator fascia → anococcygeal raphe & coccyx

INNERVATION

Sympathetics L1-3 → preaortic → hypogastric plexus, join nervi erigentes (S2-4) → pelvic plexus

IAS motor: sympathetic L5 + parasympathetic S2-4

EAS: inferior rectal br. of pudendal (S2,3) + perineal br. of S4

- UNILATERAL pudendal transection does NOT impair EAS function – crossover at the cord

Levator ani: S3 & S4 roots, perineal branch, inferior rectal nerve

- pudendal branches → pubococcygeus & puborectalis; direct S3/S4 → iliococcygeus

Upper canal sensory endings:

- Meissner's corpuscles – touch
- Krause's bulbs – temperature
- Golgi-Mazzoni bodies – pressure
- genital corpuscles – friction
- the organized endings in the transition zone are what make SAMPLING possible

Inferior rectal br. of pudendal = anal sensation, and some solid vs. gas discrimination

CONTINENCE

Requires rectal compliance + a competent sphincter that can assess and release on command

Resting tone:

- IAS 50-70%
- hemorrhoidal cushions an additional 15% – expand to seal proximal to the anal opening
- most of it MYOGENIC – slow, constant waves of contraction
- IAS gets excitatory SYMPATHETIC and inhibitory PARASYMPATHETIC input, NO-mediated

IAS injury → PASSIVE incontinence / seepage

EAS injury → URGE incontinence

EAS contributes little at rest – its job is voluntary & reflexive contraction against rectal distension

- FATIGABLE: defers a large volume for only seconds to minutes despite normal compliance
- after EAS contraction, urgency & tenesmus fade as the rectum adapts

Puborectalis:

- 90-100° anorectal angle at rest – its critical role in continence
- flap-valve theory: pushes the anterior rectal wall onto the upper canal
- alternative: acts as a deeper sphincter working with the EAS

RECTAL SENSATION & COMPLIANCE

Rectal distension does NOT evoke pain – unlike the rest of the lower GI tract

Baseline rectal pressure ~5 mmHg. Anal canal pressure 10-14x that

- the gradient is what defers stool back into the sigmoid

NO proprioceptive receptors in the rectum

- compliance from slowly adapting mechanoreceptors = rectal intraganglionic laminar endings (rIGLEs)
- respond to tension and to RAPID distension

Defecation is actually sensed at the LEVATORS and anal canal

- why the sense of defecation is preserved after proctectomy w/ ileoanal or coloanal anastomosis

ANORECTAL REFLEXES

RAIR: intrinsic INTRAMURAL reflex. Rectal distension → upper IAS relaxes

- lets stool or flatus reach the receptors of the upper canal = SAMPLING

- lower IAS tone + EAS + puborectalis push feces back up and delay defecation
- occurs every 8-10 min, lasts < 10 seconds
- ABSENT in Hirschsprung's – no myenteric ganglia
- injury to it → poor function or incontinence after rectal resection

Cutaneous anal reflex: EAS contracts to touch or pain of the anal skin. Spinal, needs S4

Bulbocavernosus reflex: EAS contracts on squeezing the glans or clitoris – or on pulling a Foley

Cough-anal reflex: EAS contracts on cough or sniff

- this is what holds continence through cough, sneeze, laugh

NORMAL DEFECATION

1. Contents reach the rectum, sensory mechanisms allow it to stretch
 2. RAIR → IAS relaxes. Sampling distinguishes feces from flatus, solid from liquid
 3. If deferred: sphincters contract, rectum keeps distending. Urge lasts only a FEW SECONDS
 4. If proceeding: pelvic floor relaxes, Valsalva
 - puborectalis relaxes & lengthens → anorectal angle straightens
 - CLM contracts → anal canal SHORTENS
 - pelvic floor descends
 5. Sphincters relax, rectosigmoid contractions propel through the canal
 6. Closing reflex: exaggerated EAS contraction + restoration of IAS resting tone
- Squatting or hip flexion straightens the angle and ↑ intraabdominal pressure

PHYSIOLOGIC TESTING

Anal Manometry:

- resting tone NORMAL 40-80 mmHg – IAS plus the high-pressure zone (normal 2.0-4.0 cm)
- maximum squeeze NORMAL 40-80 mmHg – this is EAS function
- RAIR tested by inflating a balloon in the distal rectum
- absent RAIR: Hirschsprung's, or after proctectomy
- no standardized normal values across age or sex – interpret with caution

PNTML:

- stimulate where the nerve enters the ischiorectal fossa at the coccyx
- NORMAL 2 ± 0.2 ms
- prolonged in idiopathic incontinence, rectal prolapse, sphincter injury
- operator dependent, LOW sensitivity and specificity

Balloon Insufflation:

- 3 endpoints: first detectable sensation, urge to defecate, maximum tolerable volume
- urge = ↑ compliance, volume rising with pressure = “adaptive relaxation”

- HYPOsensitivity (high volumes): neuropathy, altered reservoir
- HYPERsensitivity (low volumes, urgency & frequency): IBD, proctitis, functional bowel dz

Defecography:

- liquid barium + air into rectum, then paste into rectum ± vagina. Seated on a commode
- films at rest, squeeze, and Valsalva
- measures anorectal angle, perineal descent, paradoxical puborectalis contraction

FUNCTIONAL DISORDERS

FECAL INCONTINENCE

Etiology is usually MULTIFACTORIAL – mechanisms are redundant, single deficits are tolerated

- obstetric trauma may not declare itself for DECADES, as compensation fails

Sphincter defect: sphincterotomy, fistula surgery, hemorrhoidectomy, obstetric injury, trauma

Idiopathic = denervation of the pelvic floor and/or sphincter

- age, chronic straining, distension from constipation, outlet dysfunction
- repeated TRACTION on the pudendal nerve → denervation + fibrous replacement of the EAS
- ↓ sphincter pressures w/ age, and in women after menopause. PNTML prolonged in older women

vs. continent controls: ↓ resting and squeeze pressures, longer PNTML, higher mucosal

electrosensitivity thresholds, lower volume & pressure thresholds for urge, impaired anal sensation

- this combined motor AND sensory deficit is the rationale for sacral and posterior tibial nerve stimulation

Spinal cord injury: ↓ resting tone, loss of voluntary control, loss of anorectal reflexes

- loss of rectal sensation blunts RAIR; unopposed distension → involuntary sphincter relaxation

DISORDERS OF DEFECATION

Inadequate propulsion, dyssynergia, or both. Also anatomic – rectocele, intussusception, prolapse, stricture

Idiopathic outlet dysfunction: resting pressures may be elevated, but findings vary widely

- impaired rectal sensation or ↓ RAIR reduces the autonomic emptying response

New constipation after hysterectomy w/ ↓ rectal sensation – the parasympathetics reach the rectum via the LATERAL VAGINAL WALLS, at risk in lateral pelvic dissection

Anismus: paradoxical PUBORECTALIS contraction on EMG w/ straining & Valsalva, despite adequate rectal pressure

Physiology does NOT reliably predict the syndrome

- one study: no difference in perineal descent or manometric EAS b/n incontinence and obstructed defecation

ANORECTAL PAIN

Diagnosis of exclusion – no trauma, fissure, thrombosed hemorrhoid, abscess, or tumor

Chronic proctalgia: > 20 minutes, chronic or recurring

- LEVATOR ANI SYNDROME if posterior traction on the puborectalis on DRE reproduces the pain

Proctalgia fugax: < 20 minutes, with resolution b/n episodes

Either may follow anorectal surgery, but MORE often a/w anxiety and post-traumatic stress

Manometry and other testing are variable – they do not make the diagnosis

UROGYNECOLOGIC OVERLAP

Incontinence, obstructed defecation, prolapse, rectocele, enterocele, sigmoidocele and anorectal

pain coexist w/ urinary incontinence or retention, uterine prolapse, chronic pelvic pain

- shared pelvic floor & urogenital diaphragm musculature, overlapping nerve roots

4 PERIOPERATIVE/ENDOSCOPY: ENDOSCOPY

OFFICE ANORECTAL EXAM

POSITIONING

Prone jackknife – BEST exposure, needs a proctoscopic table

- avoid: poor mobility, recent abdominal surgery, obesity, late pregnancy, knee/hip/back arthritis

Left lateral (Sim's) – hips & knees 90°, buttocks over the edge, assistant retracts the right buttock

Describe findings ANATOMICALLY – clockface depends on the position the pt is in

DRE

May be OMITTED: fissure, thrombosed external hemorrhoid, irreducible internal hemorrhoids, tight stricture, large mass

- neutropenia is only a RELATIVE contraindication – little data behind the old teaching

Puborectalis: posterior pressure should make the anus “gape” then recover = intact reflex arc

ANOSCOPY

No prep, no sedation. Beveled/slotted end PREFERRED – lets you band. Reinsert the obturator before rotating

RIGID PROCTOSCOPY

Best tool for the exact distance of a rectal lesion from the anal verge

25 cm x 19 mm standard (15 & 11 mm available), marked at 1 cm. Enemas, no sedation

Aim posteriorly toward the sacrum. Advance to ~20 cm comfortably

Withdraw sweeping side to side, flattening the valves of Houston

Open the viewing window before removal to release retained air

Contraindicated: fissure, thrombosed hemorrhoids, stenosis, recent anorectal surgery

ENDOANAL / ENDORECTAL US

EAUS for sphincter defect: sens & spec approach 100%. H₂O₂ via the external opening maps a fistula

ERUS for rectal CA: 66-92% accurate for T, 64-88% for N
- MRI has largely REPLACED it for staging – still useful for superficial disease

2D for sphincter, 3D for CA staging. 3-20 MHz – higher freq = better resolution, less depth

EAUS levels:

- distal: hyperechoic EAS only
- mid: EAS + hypoechoic IAS
- upper: U-shaped puborectalis

ERUS 5 layers:

- interface / mucosa / submucosa / muscularis propria / perirectal fat – hyper, hypo, hyper, hypo, hyper
- uT1 submucosa, uT2 into not through MP, uT3 through MP – “scalloped”, uT4 adjacent organ

BOWEL PREPARATION

~25% of US & W. European colonoscopies have an INADEQUATE prep

Inadequate → ↓ ADR, longer procedure, repeat scopes, shorter surveillance intervals

Iso-osmotic – PEG-ELS (GoLYTELY, NuLYTELY):

- 4 L. GOLD STANDARD comparator. 15% cannot finish it
- 2 L PEG + ascorbic acid (MoviPrep): equal, better tolerated – but 1 L extra clears on top

Hypo-osmotic – PEG-3350 + sports drink (MiraLAX-Gatorade):

- cheap, tolerated, NOT FDA approved as a prep. Usually + bisacodyl
- unbalanced electrolytes – reports of severe hyponatremia

Hyper-osmotic:

- Mg citrate: not FDA approved, toxicity in the elderly & renal dz
- oral Na sulfate (Suprep): = 2 L PEG-ELS, > 4 L SF-PEG-ELS. No fluid/lyte shift
- Na phosphate: aqueous form WITHDRAWN – acute phosphate nephropathy
 - tablets (32 with 2 L) remain, box warning. Mimics IBD on the mucosa – AVOID in IBD
- Na picosulfate + Mg citrate (Prepopik): low volume, osmotic + laxative

Split dose:

- better prep, higher ADR, better tolerated – regardless of agent
- 2nd dose 3-8 hrs before, and MUST be done ≥ 2 hrs before sedation (ASA)
- split-dose 4 L PEG-ELS = the current ASGE standard

Poor prep:

- low quality = can't detect lesions < 0.5 cm → repeat EARLY
- prep + clears starting 2 days out; antiemetic if gastroparesis/dysmotility
- clears vs. low-residue diet: NO difference. Last resort – admit and give it by NGT

Antibiotics:

- NOT routine. Not for endocarditis, vascular grafts, or orthopedic prostheses
- high-risk cardiac + established GI infection with enterococci → cover
- CAPD: peritonitis 6.3% without prophylaxis. ISPD – ampicillin 1 g + aminoglycoside ± Flagyl IV

Anticoagulation:

- diagnostic ± biopsy = LOW risk. Polypectomy = HIGH risk, and you rarely know in advance
- ASA / NSAIDs: do NOT routinely stop. If held, 7 days
- clopidogrel 5-7 d; warfarin 5 d; LMWH 12 hrs; Xa & DTI 1-5 d by half-life
- no bridging noninferior for arterial thromboembolism – bridging tripled major bleeding

SEDATION

Anesthesia use rose 1/3 (2009) → 1/2 (2013) of cases – as much as \$1.5 billion/yr

Moderate (benzo + opiate):

- slow onset, recall, prolonged recovery, emesis
- 45% of “moderate” sedations reach DEEP sedation at least once
- naloxone & flumazenil at hand

Propofol:

- GABA. Rapid on/off, amnesia, best operating conditions & satisfaction
- easily deep – often true general anesthesia
- nurse-administered / endoscopist-directed protocols are safe & cost-effective

COLONOSCOPY

INSTRUMENTATION

133-170 cm. Adult OD 12.8-13.2 mm, pediatric 11.6-11.7, neonatal 9.7-9.8. Variable stiffness on modern scopes

INSERTION MANEUVERS

Torque: right hand on the shaft. Stiffens, reorients the tip, prevents loops

Tip deflection: BOTH knobs, thumb & index of the LEFT hand. Right hand never leaves the shaft

Dithering / jiggle: rapid in-out & side-side to “accordion” the colon onto the shaft

Air aspiration: suction PULLS the tip around a flexure without pushing a loop

Slide-by: blind push with max deflection. NEVER by an unsupervised trainee

- stop immediately for resistance or mucosal blanching. Very painful if unsedated

TECHNIQUE & LANDMARKS

Left lateral. Double-glove the right hand, strip the outer glove after DRE

Suction port is at 5 o'clock – rotate so fluid is inferior, tip just above the air-fluid line

Partial repeated suction – full depression pulls mucosa into the port

Rectosigmoid: acute angle, worse after hysterectomy.

Short advance/withdraw + clockwise torque

- MOST FREQUENT SITE of perforation – no slide-by here

Sigmoid: most tortuous, high tone, unfixed. Diverticula get mistaken for the lumen

- pushing through a loop can trigger a vasovagal – withdraw and reduce

Splenic flexure: blue shadow of spleen, pooled fluid

Transverse: TRIANGULAR lumen from the taenia. Loops here – external pressure

Hepatic flexure: blue liver shadow. The “fool’s cecum” of the novice

- intermittent suction draws the tip toward the cecum; supine + abdominal pressure

Cecum: “crow’s foot” converging on the appendiceal orifice

- photodocument the appendiceal orifice and IC valve. MUST see the landmarks
- transillumination or RLQ pressure is NOT proof of cecal intubation

TI: reduce ALL loops, then hook the valve edge. Granular mucosa, active motility

- routine ileoscopy debated – gross findings 1%, pathology 0.3% at screening
- MANDATORY for IBD and obscure GI bleeding

Routine rectal retroflexion does NOT increase detection of important neoplasia

ADJUNCTS

CO2 insufflation:

- absorbed and exhaled – less distention, less post-procedure pain
- best with combined laparoscopy, esp. an incompetent IC valve. Protective against barotrauma

Water insufflation:

- ↓ pain and looping, stabilizes the wall for resection of large polyps
- but ↑ incomplete colonoscopy and ↑ adenoma MISS rate

Chromoendoscopy:

- 0.5-1% indigo carmine by spray catheter. Pit pattern & dysplasia
- best use = IBD surveillance, where dysplasia is flat. Costs time

Narrow Band Imaging:

- filters out red. Adenomas brown on blue-green; serrated lesions PALE (hypovascular)
- ↑ ADR vs. white light; = chromoendoscopy for UC-associated neoplasia

THE DIFFICULT EXAM

Looping = the most common reason

Supine for a difficult sigmoid; right lateral or prone to get from ascending to cecum

Abdominal pressure – for the sigmoid start superior & right of the umbilicus, direct to the LLQ

- apply gradually. Effectiveness depends entirely on the assistant

Rotate the whole scope 180° to change the angle of approach

Failing all this, withdraw to the rectosigmoid and start again

INCOMPLETE COLONOSCOPY

Rate 4-25%. Poor prep → re-prep and repeat. Imaging is useless if the prep was inadequate

Failed cecal intubation → alter sedation, different caliber scope, CO2 or water, fluoroscopy or scope guidance, balloon endoscopy

CT colonography has replaced DCBE

- sens 96% CA, 67-94% advanced adenoma ≥ 1 cm, 73-98% ≥ 6 mm

- costs another prep. Repeat scope by an EXPERT endoscopist beats it
- ABANDONING a difficult scope is NOT a failure – perforation is worse than an incomplete exam

COMPLICATIONS

PERFORATION

Pooled prevalence 0.05%; 0.08% with polypectomy

- older series 0.012-0.8% diagnostic, up to 5% therapeutic

Direct trauma: sigmoid = 64-74% of perforations

- slide-by, forcing a diverticulum, or “bowing” the shaft against the wall proximal to the tip

Barotrauma: up to 35%. Thin-walled cecum & ascending.

Also after stenting an obstructing lesion

Risk: severe diverticular dz, severe IBD, ischemia, prior abdominopelvic surgery

Upright film FIRST – an overdistended colon mimics peritonitis. CT if equivocal

Early recognition → better prognosis, less often needs surgery

Management:

- diffuse peritonitis → emergent exploration. Laparoscopy is safe
- good prep + minimal contamination + stable → primary repair or resection & anastomosis
- stable, no peritonitis, after therapeutic scope → NPO + IV abx, success 33-90%
- endoclip if recognized intra-procedurally: success 59-100%
- delayed surgery after failed nonop mgmt = worse complications & LOS than up-front surgery

BLEEDING

Post-polypectomy 0.3-6.1%. After diagnostic scope only 0.3%

Watabe: polyps > 10 mm carry 4.5x the risk

- < 10 mm 0.4%, 10-19 mm 1.6%, 20-29 mm 3.8%, > 29 mm 5.3%

Risk: size > 2 cm, thick stalk, RIGHT colon, resumed anticoagulation, CV dz, HTN

- risk is with anticoagulants, NOT ASA, NSAIDs, or antiplatelets

Immediate: clips ± cautery ± dilute epinephrine

Delayed: up to a week or more out – warn the pt in writing

- “hot biopsy” does NOT prevent it – cautery drives into submucosal arteries → delayed bleed

Rx: resuscitate, rapid prep, repeat scope.

Angioembolization if it fails. Surgery almost NEVER

POST-POLYPECTOMY SYNDROME

Transmural burn – pain, fever, leukocytosis, localized peritonitis, NO free air

0.003-0.1% of all scopes; ~1% after EMR; 7-8% after ESD

Presents 1-5 days out. CT: mural thickening, stratified enhancement, no extraluminal air

Rx: IV fluids, broad-spectrum abx, bowel rest. Surgery almost never

SPLenic INJURY

~1 in 100,000, under-reported. Traction avulsing the splenocolic ligament

Subcapsular hematoma most common; rupture with shock possible

70.8% female, 63.8% prior abdominopelvic surgery

MOST ultimately require operation – splenic artery embolization gaining favor if stable

INFECTIOUS

Bacteremia 2-4%. Transmission via a contaminated scope ~1 in 1.8 million

- Salmonella, Pseudomonas, Mycobacterium – biofilm on the inner channels

Preclean IMMEDIATELY after the case to prevent biofilm, then leak test, high-level disinfection, visual check

QUALITY MEASURES

Cecal intubation rate, photodocumentation, intervals, prep quality, withdrawal time, ADR

Withdrawal time:

- USMSTF 2002: ≥ 6 min for a screening scope without biopsy
- newer data: 9 min ↑ detection of adenomas AND serrated lesions
- easily gamed – parking in the rectum, re-examining the same segment

Adenoma Detection Rate:

- fraction of pts ≥ 50 on a FIRST screening scope with ≥ 1 adenoma
- benchmarks ≥ 30% men, ≥ 20% women, ≥ 25% mixed
- BEST metric – hard to manipulate, measures the endoscopist not the pt
- polyp detection rate is gameable with hyperplastics; CA detection too rare to track
- drawback: rewards “one-and-done” – only one polypectomy is reimbursed
- adenomas per colonoscopy (APC) proposed to fix this

Training:

- GI fellows: 90% independent completion NOT reached until 500 colonoscopies
- ACGME minimums 50 general surgery, 140 colorectal (30 interventional) – numbers ≠ proficiency
- ABS Flexible Endoscopy Curriculum + FES (SAGES) required since 2017-2018

5 PERIOPERATIVE/ENDOSCOPY: ENDOSCOPIC

MANAGEMENT OF POLYPS AND ENDOLUMENAL

SURGERY

POLYPECTOMY

FORCEPS

- Cold biopsy forceps open to 6 mm; JUMBO cold forceps 8.6 mm – jumbo superior for complete resection
- Hot biopsy forceps: ABANDONED – ESGE recommends against them
- 17% persistent polypoid tissue at repeat scope 1-2 wks later
 - ↑ delayed hemorrhage, and it distorts the histology

CAST – Cold-forceps Avulsion with Snare-Tip soft coagulation

- for the non-lifting laterally spreading lesion the snare slides off of
- avulse all visible polypoid tissue cold, then soft-coagulate the submucosa & margins with the snare tip

SNARE

Incomplete resection by ANY method → interval colorectal CA

Polyps < 6 mm: cold snare 93% complete vs. 76% for cold forceps

- Cold snare = hot snare for complete resection, with less delayed bleeding and less time
- dedicated thin-wire cold snares better still, esp. 8-10 mm and sessile

ESGE 2017:

- < 5 mm: cold snare PREFERRED
- 6-9 mm: cold snare strongly favored
- sessile 10-19 mm: hot snare AFTER submucosal injection
- pedunculated: hot snare – lowers bleeding

Polypectomy required in 30-40% of all colonoscopies

- Incomplete resection 10% for 5-20 mm polyps – risk with larger size and SESSILE SERRATED histology
- residual tissue → repeat snare or cold forceps avulsion

Snaring technique:

- put the polyp in the LOWER HALF of the screen – the working channel exits at 5 o'clock
- if you can't, back the tip away for a better angle of attack
- cold snares grip better – thinner wire
- angulated (hexagonal) snares grasp better and open wider than oval or round

POST-POLYPECTOMY BLEEDING

1-2 per 1000 – 10x the rate of colonoscopy without polypectomy

Cold snare bleeding is IMMEDIATE – handle it at the same sitting

Hot snare bleeding is DELAYED, 0.1-0.7%, up to 30 days out

Risk: large size, PROXIMAL location, anticoagulant/antiplatelet, multiple comorbidities

Routine prophylactic clipping after ordinary polypectomy does NOT help – cost without benefit

SUBMUCOSAL LIFTING

Separates the target from the muscularis – prevents full-thickness and thermal injury

Saline lift lasts ~3 min. Equal to everything else for complete resection, bleeding, perforation

- longer cases: hetastarch, or proprietary Eleview / ORISE – over 40 min

Add DYE (methylene blue, indigo carmine) – delineates margins and deep injury

Technique:

- inject TANGENTIAL to the wall, never perpendicular
- start injecting before the needle enters, or enter the wall and withdraw back into the submucosa
- correct submucosal injections are focal and TALL; subserosal/intramuscular are broad and flat
- to extend a lift, aim at the BORDER of the existing cushion
- dynamic technique: small test injection to confirm the plane, then rapid large volume while redirecting
- lesion on a fold: start on the proximal (oral) side to tip it toward the scope
- same technique for TATTOO placement – keeps the ink out of the peritoneum

Failure to lift = wrong plane, scarring from prior attempts, or SUBMUCOSAL INVASION

ENDOSCOPIC MUCOSAL RESECTION

Sessile or flat lesions confined to the superficial wall.

Routinely 20-50 mm

Lift-and-cut: submucosal injection then snare, in the FEWEST pieces possible

2-3 mm margin of normal mucosa

Piecemeal: snare sequentially along the margin of the prior resection

Safe en bloc for lesions < 20 mm; > 2 cm usually piecemeal → limits pathologic assessment

Recurrence 17% overall in a 1000-case trial (5% for 20 mm lesions) – earlier series 30%

- risk: size (OR 8.2 for > 40 mm), APC use (OR 2.4), bleeding (OR 1.6)
- APC ablates superficially and does NOT eradicate – avulsion 10% recurrence vs. APC 59%

Cold snare piecemeal after lift works up to 55 mm with low recurrence

Underwater EMR:

- evacuate air, fill with 500 mL – 1 L sterile water. Mark the edges with APC, piecemeal snare on cutting current
- water flattens the colon and keeps the muscularis out of the snare
- ↑ complete macroscopic resection, ↓ recurrence. Good for SALVAGE of recurrent polyps

- costs time to instill

Clips:

- NOT routine. Selectively to reapproximate after EMR, or onto a bleeding vessel
- RCT, non-pedunculated polyps > 20 mm: clipping ↓ post-polypectomy bleeding
 - benefit is PROXIMAL – 9.6% → 3.3% (ARR 6.3%). NO benefit in the distal colon
- helps even when the defect cannot be completely closed

ENDOSCOPIC SUBMUCOSAL DISSECTION

For lesions > 2 cm WITHOUT features of malignancy – en bloc, full histology

vs. EMR: 7x LOWER recurrence, but ~5x higher perforation (6.2% vs. 1.3%)

Only 20% of polyps called “endoscopically unresectable” and sent for colectomy are actually malignant

- large polyps that LOOK benign: < 10% CA rate

vs. laparoscopic colectomy: shorter LOS, lower cost, same complication rate but less severe

Technique:

- mark 2-3 mm outside the lesion edge, then inject OUTSIDE the marks
- incise the DISTAL (anal) border semicircularly – a full circumferential incision leaks the lift out
- dissect tangential to the submucosa with the knife. Clear cap distal attachment for traction
- position the pt so gravity drops the lesion away from the wall
- blue dye makes vessels obvious – coagulate as you go, and re-inject ahead of the dissection
- hybrid (ESD borders + snare the center): same en bloc rate, faster, but HIGHER recurrence
- inspect the bed – clips or endoscopic suture for small defects, over-the-scope clip for large
- pin the specimen to a board for orientation

Complications:

- post-ESD electrocoagulation syndrome up to 40%
- bleeding 2-7%; perforation 5-20% – risk rises with size and fibrosis
- perforating a malignant lesion can SEED the peritoneum – 9% in the gastric series
- local recurrence ~1%, none invasive, all managed endoscopically

Learning curve:

- ~30 supervised cases for technical proficiency, years to master
- start in the RECTUM – the mesorectum covers a full-thickness injury
- single center, 200 cases: perforation 12% → 2%, en bloc 80% → 92%

ESD vs. EMR: ESD is curative only for Sm1 – upper 1/3 of submucosa, < 1000 µm. A narrow window

- of 1100 colorectal ESDs, 19% had invasive CA and half of those were Sm2 needing resection
- so only ~10% actually got the benefit of avoiding an operation
- EMR recurrences are usually caught on surveillance and retreated endoscopically

POST-OP CARE

EMR – home same day. ESD – consider overnight

NO prophylactic antibiotics, NO dietary restriction

KUB after a difficult dissection to look for free air

LESION ASSESSMENT

Paris classification:

- polypoid vs. non-polypoid
- non-polypoid graded by protrusion: slightly elevated, flat, slightly depressed, excavated
- INVERSE relationship b/n protrusion and submucosal invasion – DEPRESSED carries the highest CA rate
- high inter-observer variability – pedunculated / elevated / depressed may be more usable

Kudo pit pattern:

- arrangement of the glands – hyperplastic vs. adenomatous vs. malignant

NICE (needs NBI):

- Type 1: same or lighter color, no vessels or lacy vessels, dark/white spots → HYPERPLASTIC
- Type 2: browner, brown vessels around white structures, tubular or branched pattern → ADENOMA
- Type 3: brown to dark brown, disrupted or missing vessels, amorphous surface → DEEP SUBMUCOSAL CA

Non-lifting sign:

- accuracy 95% for invasive CA – sens 62%, spec 98%, PPV 80%, NPV 95%
- endoscopic appearance is MORE sensitive than the non-lifting sign for flat & depressed lesions
- non-lifting also comes from fibrosis or a too-deep injection – ESD still reasonable if morphology is benign
- inadequate lift dramatically ↑ full-thickness injury – there is no buffer

Flat & depressed adenomas: not true polyps, best seen with chromoendoscopy

- high rate of associated cancer – ALL should be removed

Surveillance after piecemeal or large sessile (> 3 cm) resection:

- 3-6 months through year 1, then 6-12 months in year 2, then yearly to year 5

PLATFORMS, CLOSURE & REFERRAL

Endoscopic suturing (OverStitch):

- dual-channel scope. Grasper holds the oral mucosa, suture is passed to the anal side
- partial OR full-thickness bites, interrupted or running

- closes large and full-thickness defects without laparotomy – may avoid the overnight stay

Stabilization platforms:

- DiLumen – double balloon over any scope. Aft balloon inflated, fore balloon beyond the target
 - creates a flat therapeutic zone; fore balloon + clipped suture circles gives counter-traction for ESD
- ORISE TRS – stabilization cage + 2 working channels for angled graspers. 40 cm working length
 - lesion to 6 o'clock, then expand the cage

Referral for the “unresectable” polyp:

- historically 80% of colectomies done for polyp were OVERTREATMENT
- scrutinize the color photos and the path – ulceration, mucosal contour, vascular pattern
- features of malignancy → colectomy. Poor photos → repeat scope with EMR/ESD
- STEP-UP in the OR: endolumenal surgery → CELS → laparoscopic colectomy
 - consent for all three up front. One sitting for the pt

COLONIC STENTING

Self-expanding metal stents, UNCOVERED – ingrowth prevents migration

Two indications: (1) BRIDGE to surgery in left-sided obstruction, (2) PALLIATION

- NO role for prophylactic stenting

Bridge to surgery:

- 5x less likely to end with a permanent stoma, more primary anastomoses, fewer wound infections
- perforation 5%, migration 4-10%, reobstruction 30%.
Two RCTs closed EARLY for stent complications
- ESGE 2014: NOT preferred in an otherwise healthy pt
 - consider if ASA ≥ 3 or age > 70
- newer meta-analyses: oncologically safe – same 5-yr survival, DFS, and local recurrence

Palliation:

- ESGE PREFERRED method. Same mortality as emergent surgery, shorter LOS, fewer stomas
- 95% avoid a stoma long term
- reserve for INTRINSIC lesions – extrinsic compression migrates and perforates, no ingrowth

Technique:

- water-soluble contrast enema or CT with rectal contrast
FIRST – location, length, caliber
 - no contrast across the lesion → the wire will not pass, and false passage risks perforation
- wire across, exchange for a catheter, inject contrast AND air – haustra confirm intraluminal position
- LARGEST diameter, LONGEST stent. Shorter in the rectum – stay > 5 cm off the anus or tenesmus
- clip 5 cm distal to the lesion as a radio-opaque landing marker
- do NOT balloon dilate the stent, do NOT pass the scope through it afterward

- KUB in recovery. Expands over 48 hrs. Stool softeners so it doesn't clog

Anastomotic leak:

- COVERED esophageal stents for a contained leak – blocks stool, lets the sinus heal
- high migration rate. Left 20-50 days, then repeat contrast study. Success 80-100%

6 PERIOPERATIVE/ENDOSCOPY: PREOPERATIVE

EVALUATION IN COLORECTAL PATIENTS

RISK STRATIFICATION

SURGICAL RISK BY PROCEDURE – 30-DAY MI & DEATH

Low < 1% (superficial, breast, eye) / Intermediate 1-5% (intraperitoneal, CEA, EVAR) / High > 5% (aortic, esophagectomy, transplant)

ALL elective abdominal colorectal work = INTERMEDIATE at a minimum. Perforated viscus = HIGH

Laparoscopic is treated the same as open for cardiac risk

Emergency – do NOT delay for a cardiac workup

FUNCTIONAL CAPACITY

1 MET = resting O₂ consumption of a 70 kg, 40-yr-old

- < 4 METs: ADLs, vacuuming, walking 2 mph
- 4 METs: 2 flights of stairs, heavy housework, walking 4 mph, golf without a cart

≥ 4 METs and otherwise healthy → NO further cardiac workup, regardless of risk factors

< 4 METs = poor capacity → further evaluation and risk-benefit analysis

Can't rise from the chair and walk briskly to the table = a FRAILITY marker

REVISED GOLDMAN INDEX (RCRI)

- S3 or JVD 11 – MI within 6 mo 10 – non-sinus rhythm or APCs 7 – > 5 PVCs/min 7
- age > 70: 5 – emergency operation: 4 – AS 3 – poor medical status 3 – intraperitoneal/thoracic/aortic 3

Class I 0-5 / II 6-12 / III 13-25 / IV ≥ 26 – cardiac death 0.2% / 2% / 2% / 56%

ACS-NSQIP calculator: more inputs, but procedure-specific and captures noncardiac risk

PREOPERATIVE TESTING

Routine labs are LOW YIELD – 60% of preop labs are not indicated, and only 0.2% of those show anything actionable

Get:

- Hgb: everyone ≥ 65 having abdominal surgery, or younger if major blood loss expected or Hx of anemia
- Creatinine: everyone ≥ 50 – independent predictor of postop cardiac complications AND mortality
- Platelets: if spinal or epidural is planned
- Type & screen: ALL major abdominal surgery
- Pregnancy test: any woman of childbearing age if it would change management
- A1c within 4-6 wks in diabetics

Do NOT get routinely:

- coags & bleeding time – they do not correlate with intra- or post-op bleeding
- electrolytes, glucose, LFTs, UA in the healthy asymptomatic pt
- asymptomatic bacteriuria: treat empirically and PROCEED

ECG – ACC/AHA:

- only for known cardiac, peripheral arterial, or cerebrovascular disease. Abnormal ECG is rare under 65

CXR:

- known cardiopulmonary disease, or age ≥ 50 for major abdominal surgery. BMI ≥ 40 (AHA)

Imaging:

- Crohn's – MR enterography, which has REPLACED small bowel follow-through
- distinguishes ACTIVE inflammation from chronic fibrosis
- rectal CA – pelvic MRI is the STANDARD for locoregional staging
- colon & rectal CA – CT chest/abd/pelvis for distant disease and T4 planning

CARDIAC

EVALUATION

Clinical risk factors: angina, prior MI, CHF, stroke/TIA, renal dysfunction, IDDM

Echo NOT required if asymptomatic – consider for high-risk surgery or with cardiac risk factors

Imaging stress test: multiple risk factors + medium-to-high risk surgery + poor or unknown capacity, IF it changes management

Cath for unstable symptoms, abnormal stress test, severe CAD, or refractory to medical therapy

Do NOT revascularize solely to lower perioperative risk – beta blockade and statins instead

OPTIMIZATION

CONTINUE long-standing beta-blockers. Do NOT start them de novo

- started within 24 hrs of surgery → ↑ nonfatal stroke, MI, and hypertensive morbidity & mortality

Decompensated CHF: ACEi, aldosterone antagonist, digoxin for at least 1 week preop

CONTINUE statins. No data supports starting them de novo for colorectal surgery

ANTICOAGULATION

Warfarin:

- stop 5 days out; 3-4 days if INR 1.5-1.9. Recheck INR within 24 hrs of the operation
- most abdominal surgery is safe at INR ≤ 1.4
- restart 12-24 hrs postop once hemostasis is assured
- urgent: vitamin K 2.5-5 mg PO or IV. Emergent: FFP

DOACs – apixaban, dabigatran, edoxaban, rivaroxaban:

- onset 1-3 hrs. No monitoring, and no bridging
- stop 4-5 half-lives out: 3 days for high-risk procedures (CrCl > 50), 2 days for low risk
- resume 2-3 days postop after high risk, 1 day after low risk
- reversal: idarucizumab for dabigatran, andexanet alfa for the Xa inhibitors
- BOXED WARNING for spinal/epidural hematoma – do NOT use if neuraxial anesthesia is planned

Others:

- clopidogrel: stop 5-7 days, restart ASAP postop.
Reverse with platelets
- UFH: half-life 45 min, hold 6 hrs. Protamine
- LMWH: half-life 3-5 hrs. Hold the evening dose before;
half dose if once daily

CORONARY STENTS

DAPT for at least 12 months. Stent thrombosis risk is highest in the FIRST 4-6 WEEKS

Nonemergent surgery: wait ≥ 30 days for bare metal, > 6 months for drug-eluting (3-6 mo case by case)

Continue low-dose ASA throughout, resume the P2Y12 inhibitor ASAP

DES needing emergent surgery < 3 months out: bridge with tirofiban

- start within 24 hrs, stop 4 hrs preop, restart 2 hrs postop until clopidogrel resumes

BRIDGING

Bridging is for VKA – DOACs do not need it

CHA2DS2-VASc for thrombotic risk; HAS-BLED ≥ 3 = high bleeding risk

- LOW ($< 5\%/yr$, score < 4 , no prior stroke/TIA/SE): interrupt, do NOT bridge
- MODERATE (5-10%/yr, score 5-6, or embolism > 3 mo ago): consider bridging
- HIGH ($> 10\%/yr$, score 7-9, or stroke/TIA/SE within 3 months): bridge

Restart VKA within 24 hrs, parenteral heparin at 24-72 hrs depending on bleeding risk

PULMONARY

Pulmonary complications reported 2-70%; 6% in NSQIP for major abdominal surgery

COPD is the SINGLE MOST IMPORTANT risk factor – up to 25% complication rate in the elderly, mortality $\sim 7\%$

Other risks: age, ASA ≥ 2 , functional dependence, high Goldman index, CHF. OBESITY IS NOT one

Routine PFTs NOT indicated – clinical findings predict better than spirometry

Screen for OSA (STOP-Bang) – cardiopulmonary complications and unplanned ICU admissions

METABOLIC & NUTRITIONAL

STEROIDS

Prednisone 20 mg/day or equivalent for > 3 weeks = the classic HPA-suppressing dose

- suppression does not persist beyond 1 year off steroids

LOW risk: any dose for < 3 weeks, prednisone ≤ 5 mg/day, or 10 mg every other day – no stress dose needed

Stress dose by operation – hydrocortisone ON TOP of the usual daily dose:

- superficial (biopsy, dental): usual daily dose only
- minor (hernia, colonoscopy, ANORECTAL surgery): + 25 mg IV

- moderate (COLON RESECTION, chole, joint): + 50-75 mg IV, taper 1-2 days

- major (TOTAL PROCTOCOLECTOMY, esophagectomy): + 100-150 mg IV, taper 1-2 days

Newer data: the usual dose alone is likely enough, with RESCUE dosing only for refractory hypotension

- but stress dosing carries minimal risk against an adrenal crisis – still reasonable

DIABETES

Preop: ECG, CXR, creatinine, glucose, A1c within 4-6 weeks

Many centers POSTPONE elective surgery for A1c > 6.5

OBESITY

$\uparrow$ VTE, SSI (4% vs. 3% for open abdominal), wound disruption, sepsis, respiratory & renal complications, UTI

$\uparrow$ operative time, $\uparrow$ anastomotic and POUCH-ANAL leak, and inability to reach the low rectum or anus

Mark the stoma HIGH – the pt has to see it and reach it.

Enterostomal therapist preop

Obesity does NOT exclude malnutrition – sarcopenia and protein-calorie malnutrition are common

Delay for optimization in malignancy does NOT hurt disease-specific survival

MALNUTRITION

Albumin < 4.0 g/dL defines the malnourished state and is a negative prognostic marker

- albumin, prealbumin, transferrin, RBP all fluctuate – use TRENDS, not single values

Sarcopenia: psoas cross-sectional area at L3 normalized to height

- $\sim 1/3$ of colorectal CA pts. LOS 13 vs. 7 days, 1-yr mortality 13.9% vs. 0.9%
- any complication 85.2% vs. 34.5%, major 30.4% vs. 8.9%

TPN: 1.5 g protein/kg, 25 kcal/kg, glucose < 150

IMMUNOSUPPRESSION & CHEMOTHERAPY

STEROIDS

Crohn's: overall complications 15.4% vs. 6.7%

Left-sided resection: steroids $\uparrow$ anastomotic leak more than 7-fold (OR 7.52)

Meta-analysis: leak 6.77% on steroids vs. 3.26% off

HIGHEST RISK: prednisone > 20 mg/day, > 2 months of steroids, or biologics within 12 weeks

- delay the pouch or anastomosis (modified 2- vs. 3-stage), DIVERT, wean to ≤ 20 mg for 2 weeks first

IMMUNOMODULATORS & BIOLOGICS

Azathioprine / 6-MP: no difference in anastomotic complications (10% vs. 14%) – may continue to surgery

Anti-TNF (infliximab, adalimumab, certolizumab) – conflicting:

- Krane: no difference in leak, 2.1% vs. 1.3%, despite MORE steroid use in the infliximab arm

- Wong meta-analysis: consistent ~20% ↑ in infectious complications (OR 1.5)
- PUCCINI: anti-TNF within 12 wks NOT an independent risk factor for postop infection

Vedolizumab (anti-α4β7): use within 12 weeks independently predicts 30-day SSI – worse than anti-TNF

NSQIP Crohn's colectomy: immunosuppression → infection OR 1.25, SSI 1.40, organ space 1.47, leak 1.41

Practice: hold biologics 3.5 half-lives – 6-8 wks for anti-TNF, 12 wks for vedolizumab. Divert if you can't

TRANSPLANT RECIPIENTS

CONTINUE immunosuppression through the perioperative period

mTOR inhibitors (sirolimus, everolimus) impair wound healing – 47% vs. 8% wound complications vs. tacrolimus

- switch to tacrolimus 6 weeks before elective surgery
- Early elective resection for diverticulitis in the immunosuppressed is NO LONGER recommended

CHEMOTHERAPY

Conflicting data on leak – one series OR 2.85 for preop chemotherapy

Bevacizumab (anti-VEGF-A): impaired healing and leak, including LATE leaks

- excluded from trials within 28 days of major surgery
- hold all antiangiogenics – also pazopanib, aflibercept – for ≥ 6 WEEKS. Consider diversion

Diverticulitis on chemo: manage NONOPERATIVELY.

Chemo can be resumed after it resolves

- recurrences are more severe – emergent surgery 75% vs. 23.5%, all diverted, all complicated

SUBSTANCE USE

Alcohol: dose-dependent ↑ SSI, cardiopulmonary complications, LOS, ICU stay, reoperation

- 1 month abstinent before colorectal surgery: MI 23% vs. 85%, arrhythmia 33% vs. 86%, overall 31% vs. 74%
- recommend 3-8 weeks alcohol-free. AUDIT-C to screen

Tobacco: the old teaching that quitting < 8 wks out is worse has been DISPROVEN

- OR for pulmonary complications 2.92 if still smoking or quit < 2 wks, 0.98 if quit 4-8 wks, 1.42 if ≥ 8 wks
- encourage cessation regardless of timing. Ideally ≥ 4 weeks from the quit date

Opioids: ask the indication, duration, side effects, wean plan, and WHO prescribes – then verify it

- dependency means higher perioperative requirement. Involve acute pain service preop

THE ELDERLY

Chronologic age is a POOR surrogate for physiologic reserve – ASA and Lee/Eagle miss it entirely

FRAILTY

Comprehensive Geriatric Assessment = GOLD STANDARD, but takes hours

- each additional CGA impairment = 40% ↑ in 6-month mortality. ASA class was NOT associated

Fried criteria / Hopkins Frailty Score: shrinking

(unintentional ≥ 10 lb in a year), weak grip, exhaustion, low physical activity, slow walk (15 feet)

- 4-5 frail, 2-3 intermediate, 0-1 not frail
- frail → complications OR 2.54, LOS OR 1.69, discharge to a facility OR 20.48

Modified Frailty Index (11 NSQIP variables) and Risk Analysis Index (14 questions) are the quick tools

Deficits in ADL/iADL, fatigue, and cognition predict complications. AGE DOES NOT

COGNITION & DELIRIUM

Cognitive dysfunction 5-15% overall, up to 60% in high-risk groups

Mini-Cog preop – 3-item recall + clock draw.

Recommended by ACS & AGS

Delirium after abdominal surgery in pts > 70: incidence 60%, and 30-day mortality 20% in those pts

Modifiable: visual & hearing impairment, cognitive impairment, sleep deprivation, immobility, dehydration

- also glucose > 150, > 4 new medications, bladder catheters, BUN/Cr > 17, restraints, uncontrolled pain

Rx: NOT drugs first – reorient, avoid triggers, music, family at bedside. Haldol only if you must. AVOID restraints

PREHABILITATION

Multimodal: exercise (aerobic + resistance + flexibility), nutrition, anxiety reduction

Carli RCT in FRAIL colorectal CA pts, mean age 78: NO difference in any outcome

Systematic review, major abdominal surgery: ↓ overall complications OR 0.61, pulmonary 0.41, cardiac 0.46

7 PERIOPERATIVE/ENDOSCOPY: OPTIMIZING OUTCOMES

WITH ENHANCED RECOVERY

CONCEPT & ORIGINS

DEFINITION

Standardized, evidence-based intervention at every phase of care to blunt surgical stress

- goal: avoid complications, faster recovery & discharge WITHOUT ↑ readmission, ↓ cost
- enhanced recovery / ERP / ERAS used interchangeably

Peri-op stress → sympathetic & endocrine activation, insulin resistance, acute phase reaction, cytokines, leukocytosis, thrombosis

ORIGINS

Wilmore (Boston) & Kehlet (Copenhagen) – define the MODIFIABLE sources of peri-op stress

Kehlet: immediate diet + immediate activity + multimodal analgesia → earlier discharge readiness after OPEN colectomy

- pts on protocol never dip below their baseline health

COST trial 2004 – lap non-inferior for colon CA → MIS uptake in North America

ERAS Society named 2007. NHS Enhanced Recovery Partnership Programme after the 2008 recession

- first MANDATED and first truly multidisciplinary program – began with colorectal

ASER 2014, American chapter of ERAS Society 2017, Enhanced Recovery Canada 2017

BUILDING A PROGRAM

Step 1: define the outcomes to improve. Step 2: build the evidence library

- do NOT start by writing order sets

Champions from surgery, anesthesia & nursing are ESSENTIAL

- then pharmacy, IT, nutrition, administration

Phases: preop → intraop → postop, or 5-phase (ACS Quality Red Book): preop / periop / intraop / postop / post-discharge

- elements repeat across phases – the pt and the surgeon are the only two players in every phase

PREOPERATIVE ELEMENTS

EDUCATION

Simplest appropriate literacy level – written, spoken, and video

Common language at every touch point ↓ variability & ↓ stress

SMOKING CESSATION

a/w leak, impaired microcirculation, post-op pulmonary complications, IBD recurrence

Even 2-3 weeks of pre-op cessation is beneficial

Addiction-trained local team ↑ success

NUTRITION

PONS score (pre-op nutrition screening):

- BMI < 18.5 (< 20 if age > 65)

- unplanned weight loss > 10% in 6 months
- eating < 50% of normal diet in the preceding week
- any YES, and/or albumin < 3.0 → pre-op nutrition clinic or dietician

Protein calories, routine minerals & vitamins, directed supplements for measured deficiency

Wait for UPTRENDING weight & prealbumin – normalize before operating

ANEMIA

Algorithm:

- Hb < 12 g/dL female, < 13 male → serum ferritin
- ferritin < 100 µg/L = iron deficient → operation > 6 wks out: PO iron; < 6 wks: IV iron
- ferritin > 100 → Cr & CrCl. Abnormal → nephrology. Normal → B12 & folate (folate PO, B12 SC)

HYPERGLYCEMIA

Degree of hyperglycemia is LINEARLY a/w severity of complication

- the risk is NOT seen in pts well managed with insulin around the operation

HbA1C screening (ADA):

- age > 45, hx DM (incl. gestational), PCOS, high fasting glucose
- BMI ≥ 25 with inactivity, HTN, hyperlipidemia, or 1st-degree relative w/ DM
- normal < 5.7%. Prediabetic 5.7-6.4%. Diabetic ≥ 6.5%

HbA1C takes 2-3 months to move – our pts realistically get 2-4 weeks

- 2 weeks of BS < 150 mg/dL is a measurable risk reduction
- BS check in holding, q2h intraop, PACU, then qAC & HS

FASTING & CARBOHYDRATE LOAD

Clear liquids up to 2 HOURS before induction of general anesthesia (ASA guideline)

Ljungqvist: pre-stress MALTODEXTRIN carb loading ↓ post-op insulin resistance & complications

- plain carbohydrate WITHOUT the maltodextrin source does not do it
- mediated by AMP-activated protein kinase activation

BOWEL PREP

Mechanical prep + ORAL ANTIBIOTICS for any planned lower bowel resection

- early ERAS guidelines did not endorse it – evidence since is robust, most programs include it now

PERIOPERATIVE & INTRAOPERATIVE ELEMENTS

PONV PROPHYLAXIS

Risk score – 1 point each:

- female, non-smoker, hx PONV, post-op opioids
- baseline 10%, any one 20%, two 40%, three 60%, all four 80%
- identify in pre-op clinic → multimodal prophylaxis by anesthesia

MULTIMODAL ANALGESIA (MMA)

Recognize the cost of EVERY narcotic dose – as little as 10 morphine equivalents a/w ↑ ileus

Pick ≥ 1 from each category:

- adjuncts: acetaminophen, NSAIDs, gabapentinoids
- single-shot regional: TAP, rectus sheath, SAB ± opioid
- continuous: thoracic epidural, TAP / RS catheter, wound catheter
- other: lidocaine infusion, dexamethasone, ketamine bolus or infusion
- rescue: tramadol, then opioid – PO if tolerated, IV if needed

Epidural still recommended for OPEN CRS if locally supported

- REMOVE shortly after bowel function returns
- badly run epidurals ↑ time to mobility, catheter removal & discharge
- for MIS, non-catheter blocks are more appropriate than an epidural

*Cautions: gabapentin – drowsiness & resp. depression.
PO acetaminophen before gastric decompression is less effective than IV at fascial closure*

Infusional lidocaine – center reports positive, Cochrane found no convincing evidence

Revisit the MMA scheme every 6-9 months

Rescue plan for suboptimal analgesia:

- focused H&P → assess severity → determine pain type (neuropathic / inflammatory / visceral / somatic)
- EXCLUDE a surgical or medical complication BEFORE treating
- confirm every non-opioid option used, incl. tramadol, then add opioid

FLUID MANAGEMENT

3 tenets:

- liberal fluid during bowel prep and until 2 h before induction
- ZERO-BALANCE intraoperative management, based on weight
- normotension & urine output maintained with REACTIVE IVF until PO intake adequate

Myles et al (> 3000 pts): ↑ AKI in the RESTRICTIVE arm – but no analysis of ERAS elements or pre-op fluid allowance

Mayo observational: no ↑ AKI; their chief finding was ↑ ILEUS with greater fluid volumes

OTHER INTRAOPERATIVE

MIS ↓ surgical stress – the benefit is ADDITIVE with an ERP

Minimize drains, tubes and lines

- abdominal drains: no benefit
- NGT: harm, except in obstruction
- CVL: ↑ infection & complications

VTE and antibiotic prophylaxis – see Ch 6

- timing, in-patient plan and discharge plan must be visible to the WHOLE team

POSTOPERATIVE ELEMENTS

Post-op elements carry the GREATEST impact on optimal recovery (iERAS, Canada)

- but they only succeed where the earlier phases had high compliance

EARLY DIET, MOBILIZATION & ORAL MEDS

Immediate diet and immediate mobilization – safe and beneficial

LOW RESIDUE beats clear liquids – earlier return of bowel function, earlier d/c, fewer complications

Routine post-op NGT is NOT beneficial

Adoption of early feeding remains poor – the argument for protocolized care

DISCHARGE CRITERIA

International consensus, 5 items:

- tolerating a solid diet
- adequate liquid intake
- pain controlled on oral medication
- adequate activity
- objective ward data: BP, HR, urine output, temperature, spontaneous voiding

McGill: readiness for discharge AND actual discharge both at ~3 days after colorectal resection

POST-DISCHARGE

Continued MMA, continued activity, VTE prophylaxis, frequent contact and PROs

SPECIAL POPULATIONS

STOMA

Diverting ileostomy = frequent source of delayed discharge and readmission

- high output / dehydration readmission up to 15%
- ERAS gets them out sooner – LESS time to learn stoma care

Norway RCT: ERAS + dedicated stoma nurse → shorter LOS, no ↑ readmission or stoma complications

Younis (UK): pre-op stoma teaching in an ERP nearly HALVED LOS. Readmission 2.5% → 0%

EMERGENCY SURGERY

vs. elective ERAS: longer LOS, higher unplanned reoperation, NO difference in leak or readmission

- overall compliance lower, but comparable to elective in the operating room

vs. emergency WITHOUT ERAS: safe, and confers the same benefits as in the elective setting

Meta-analysis, 6 protocols / 1334 pts: ↓ complications, faster bowel function, shorter LOS, no ↑ readmission

ELDERLY

Bagnall systematic review, 16 studies / 5965 pts – 2 RCTs: shorter LOS & fewer complications in > 65 and > 70

No difference in morbidity or mortality vs. younger pts – but longer LOS and more ER visits/readmissions

Feroci: > 75 adhere poorly to post-op items – poor compliance was the GREATEST predictor of poor outcome

Octogenarians on MIS: met release criteria at a median of 5 d, actual discharge 6 ± 4.2 d

- READINESS for discharge is a better outcome measure than LOS in the very elderly

Do NOT read ineligibility as non-compliance – the anticoagulated pt who cannot have an epidural is not non-compliant

IBD

Malnutrition, immunosuppression, anemia, abscess, fistula, obstruction – and often less suitable for lap

Ban (NSQIP): IBD dx → prolonged LOS, ↑ readmission, ↑ morbidity/mortality vs. non-IBD colectomy

Dai: post-op ileus 28.8% in IBD vs. 14.8% in colorectal CA

D'Andrea pre/post-ERP in IBD: ↓ SSI, ↓ ileus, ↓ LEAK, with trends down in LOS, readmission, reoperation, sepsis

- Crohn's → longer LOS, but the post-ERP group was still shorter

OUTCOMES & ECONOMICS

Mean saving \$3101 USD per patient (systematic review & meta-analysis)

Savings come from shorter LOS and fewer complications, without ↑ readmission

Roulin: specific gains in medication, laboratory and radiology costs

- standardization prevents unnecessary diagnostics without ↑ complication rate

Quadruple Aim: better outcomes, lower cost, better patient satisfaction, AND provider satisfaction

No worse patient satisfaction, QOL, fatigue or return to activities under ERAS

Barriers: lack of nursing staff, lack of financial resources, resistance to change, poor communication

AHRQ “Safety Program for Improving Surgical Care and Recovery” – planned for ~750 US hospitals

Future: wearables & telemedicine for recovery-centric outcomes, PROs integrated into the EHR

8 PERIOPERATIVE/ENDOSCOPY: GENERAL

POSTOPERATIVE COMPLICATIONS

OVERVIEW

Colorectal = ~25% of all general surgery operations, complication rates exceed 35%

Complications → ↑ mortality, ↑ LOS, ↑ readmission, ↑ cost, and WORSE long-term oncologic outcome

CLAVIEN-DINDO CLASSIFICATION

7 grades – I, II, IIIa, IIIb, IVa, IVb, V

- I = any deviation from the normal post-op course needing no major intervention
- V = death of the patient

Others: Comprehensive Complication Index (Clavien & Dindo), Accordion scale, ACS-NSQIP

FREQUENCY – ACS-NSQIP TARGETED COLECTOMY, 2012-2017

- ileus 17.1% > bleeding/transfusion 10.3% > sepsis 5.4% > organ-space SSI 5.1% > superficial SSI 5.0%
- septic shock 4.1%, vent > 48 h 3.8%, LEAK 3.4%, pneumonia 3.4%, UTI 2.5%
- DVT 1.5%, wound disruption 1.3%, deep incisional SSI 1.3%, C. diff 1.3%
- ARF 0.8%, MI 0.8%, cardiac arrest 0.8%, PE 0.7%, stroke 0.3%

RISK FACTORS

NON-MODIFIABLE

Age – ↓ physiologic reserve, worse organ function, cognitive impairment, frailty

- octogenarians & nonagenarians: complication rates often exceed 25%

MALE sex: ↑ overall complications and ↑ anastomotic leak

ASA 3-5 a/w leak. High Charlson Index a/w mortality & morbidity

- comorbidities CLUSTER – never read an index in isolation

Prior abdominal surgery predicts conversion, enterotomy, ileus, reoperation, longer OR time

MODIFIABLE

Malnutrition:

- up to 50% of surgical pts. Screen with NRS or MUST
- immunonutrition (arginine + fatty acids) ↓ LOS and ↓ complications
- oral supplementation ≥ 7-10 days before an elective operation. Parenteral only as needed

Smoking:

- abstinence 4 weeks pre-op returns wound infection rates to those of nonsmokers
- 6-8 week cessation program: complications 31% → 5%
- meta-analysis of 11 RCTs: 44% pooled risk reduction in 30-day complications

Anemia:

- NSQIP 23,348 elective colorectal: independent risk factor for complications and LOS

- correction with IV/PO iron → 0% post-op transfusion vs. 38% in the uncorrected

Sarcopenia:

- ↑ 82% odds of post-op complication after colorectal resection
- meta-analysis, 29 studies: RR 1.40 major, 1.35 overall complications

Obesity:

- BMI > 30, DOSE-DEPENDENT relationship b/n BMI class and SSI

Functional capacity:

- Carli: prehab improved walk test scores – but no measurable ↓ in complications
- Carli 2020: no improvement in the Comprehensive Complication Index

Open approach a/w ↑ complications. MIS ↓ SSI, VTE and pneumonia – use it whenever possible

Risk calculators: POSSUM, APACHE, and the ACS-NSQIP Risk Calculator (now with geriatric factors)

GASTROINTESTINAL – #1

ILEUS

THE single most common complication – 12-17% after colorectal surgery

Functional SB obstruction – disturbed peristalsis from anesthetic & surgical manipulation

- the large bowel equivalent is pseudo-obstruction, Ogilvie's

Slowed by opioids, electrolyte abnormalities, inflammation, pain, degree of manipulation

Distension & tympany, no flatus or BM, N/V, bloating, hiccups

- imaging: dilated loops, NO transition point

Severe vs. non-severe – even under an ERP:

- non-severe (“primary”) 6.1% – bowel rest, NGT, IVF, mobilize, AVOID opioids
- severe 9.3% – driven by an intra-abdominal complication (abscess) → treat the underlying insult

Rules: (1) exclude a mechanical component, (2) start nonoperative, (3) address the underlying cause

EARLY POST-OP SBO (ESBO)

Up to 9.5% of abdominal operations, adhesive. Higher risk after OPEN

Mimics ileus – but CT shows a TRANSITION POINT. Strangulation risk 8-9%

NG decompression succeeds in up to 87%

Safe waiting times reported from 24 h to 7 days – individualize

- reoperation carries added risk, especially after an index open case

HEMATOLOGIC – #2

BLEEDING

Reoperation is rare; transfusion in up to 10% of colorectal operations

Risk: intraop factors, hemophilia A (VIII) & B (IX), vWD, warfarin, platelet disorders

Mild and self-limited → withhold anticoagulants and NSAIDs first

196 reoperations for bleeding: 77% of takebacks within 24 h, mortality 28%

- do NOT delay for CT angiography if the pt is exsanguinating
- finding no active source is common – evacuating clot still helps achieve hemostasis

Presacral venous plexus:

- easily disrupted during proctectomy – can be massive
- packing + resuscitation FIRST
- then suture ligation, sterile thumbtacks, rectus muscle welding, hemostatic agents
- damage control: pack, ICU, correct acidosis / coagulopathy / hypothermia
- second operation at 24-48 h to restore continuity, remove packing, close

Abdominal compartment syndrome:

- pressure > 20 mmHg → decompressive laparotomy
- multi-organ dysfunction and mortality approaching 50%

VTE

Colorectal VTE rates 1.1-2.5%. CA and IBD create hypercoagulable states

Risk: obesity, steroids, sepsis, reoperation, ASA 3, any other post-op complication

Caprini and Roger scores (ACCP) → ambulation vs. mechanical vs. LMWH / LDUH

Dx: US + D-dimer for DVT, CT-PA for PE. V/Q only for contrast allergy or renal impairment

IVC filter if anticoagulation is contraindicated

INFECTIOUS – #3

SURGICAL SITE INFECTION

500,000 US cases/yr – 3.7 million excess hospital days, \$10 billion excess cost

2-11x higher risk of death. MOST are preventable
Colorectal has one of the highest rates: 15% to over 30%

Superficial incisional (skin/subcut) / deep incisional (muscle/fascia) / organ space

5 core themes across the WHO, ACS-SIS and CDC guidelines:

- appropriate antibiotic within 1-2 h BEFORE incision
- alcohol-based skin prep combined with chlorhexidine
- normothermia $\geq 36^{\circ}\text{C}$
- glycemic control – CDC target < 200 mg/dL
- FiO₂ > 80% intraop and immediately after extubation
- + mechanical prep with oral antibiotics (ASER)

CDC reviewed 42 processes: only 12 had high-quality evidence, and 25 got NO recommendation at all

Rx = source control. Superficial: open, explore, irrigate, debride, wound care

- deep and organ space: percutaneous drainage, else back to the OR for washout

- infected implanted mesh MUST come out – antibiotics alone will not address the nidus

WOUND DEHISCENCE

1-3% of colorectal operations. Evisceration = IMMEDIATE operation

Late manifestation is the incisional hernia

Systemic: DM, uremia, immunosuppression, steroids, malnutrition, CA, obesity, smoking

Local: inadequate closure, ↑ intra-abdominal pressure, poor healing

Swedish, 30,050 pts: reoperation for dehiscence in 2.9% after colorectal CA surgery, adjusted mortality ↑ 26%
STITCH trial – small bites (5 mm of fascia every 5 mm) vs. large (1 cm every 1 cm): incisional hernia 13% vs. 21%

Retention sutures in high risk (RCT, 300 pts): dehiscence 4% vs. 13.3%, evisceration 0.7% vs. 2.7%

UTI

Most frequent nosocomial infection – ~4% after colorectal operations

Risk: pre-existing contamination, retention, indwelling catheter

NSQIP: LOS +5 days, reoperation 11.9% vs. 5.1%, 30-day mortality 3.3% vs. 1.7%

C. DIFFICILE

Cause of 10-20% of antibiotic-associated colitis & diarrhea. Post-op incidence 0.2-8.4%

Risk: antibiotics, PPI, low albumin, prior hospitalization

Rx: PO vancomycin → fecal transplant → urgent/emergent surgery

ANASTOMOTIC LEAK

3.4% in NSQIP – covered in Ch 10

PULMONARY – #4

RESPIRATORY FAILURE

Ventilation > 48 h or reintubation. 3.3-4.3%, reintubation ~2.3%

Risk: poor pre-op lung function, age, OSA, pulmonary HTN, CVD, smoking, aspiration

Rx: early tracheostomy, protective ventilation, peak < 30 mmHg, PEEP 10-20 mmH₂O, mobilize, bronch. ECMO if critical

ERP with > 70% compliance + MIS prevented pulmonary complications

- a pulmonary complication costs +15 days of LOS

PNEUMONIA

The most common pulmonary complication among pts who DIE after surgery

Mortality 20-40%. Colorectal incidence 1-4%

Atelectasis, aspiration and secretions are the predisposing factors

- pain control is what allows coughing, deep breathing and mobilization

Subglottic secretion suctioning ↓ ventilator-associated pneumonia

ASPIRATION

1-2% of surgical cases, mortality > 30%

Risk: depressed mental status, NGT or ETT, older age, pulmonary dz, intraop transfusion, dementia, malignancy

Prevention: fasting, positioning, careful intubation, H2 blockers, bedside swallow evaluation

Rx: reestablish airway patency, bronchoscopy for solid matter, fluids + antibiotics

RENAL – #5

ACUTE KIDNEY INJURY

NSQIP definition: Cr rise > 2 mg/dL or need for acute renal replacement therapy

Up to 40% of all surgical pts, 14% of colorectal

Risk: hypovolemia, bleeding, nephrotoxins, cardiovascular failure

a/w short- AND long-term mortality, CKD, and dialysis

POST-OP URINARY RETENTION (POUR)

Up to 25% of colorectal pts. Still 14% under an ERP

Independent risk factors: MALE sex and THORACIC EPIDURAL

a/w slower mobilization, more pain, more UTI

Tamsulosin 3 days before and 3 days after: POUR 25% → 6.7%

CARDIAC – #6

MYOCARDIAL INFARCTION

~1.5% after a colorectal operation, mortality > 28.5%

Myocardial INJURY in ~16% of all surgical pts – mortality still 8.9%

Risk: CHF, CKD, age > 70, PVD, cancer, valvular disease, HTN

2014 ACC/AHA stepwise assessment:

- low risk < 1% MACE, elevated > 1% MACE
- functional capacity in METs: < 4 poor, 5-10 moderate/good, ≥ 11 excellent
- pharmacologic stress test if elevated risk with poor or unknown capacity
- revascularize if the stress test is abnormal

POST-OP ATRIAL FIBRILLATION

6.6% within 30 days. Closely a/w pneumonia, abdominal fluid collections, sepsis

In-hospital mortality 9.1% vs. 2.6%. 1-year mortality 33.3% vs. 8.8%

Prevention: continue beta-blockers, fluid optimization, prevent the other complications

NEUROLOGIC – #7

STROKE

0.3%. Mostly thromboembolic, within the first 72 h

Risk: intraop hypotension, prior stroke, AF, valvular/mechanical valve, HTN, PVD, age, CA, smoking

Non-contrast head CT FIRST – ischemic vs. hemorrhagic

SEXUAL DYSFUNCTION

Pudendal nerves are NOT typically damaged during proctectomy

Hypogastric nn. (T10-L2, sympathetic) – descend at the sacral promontory. Injured on posterior TME dissection or high IMA transection → EJACULATORY dysfunction

Nervi erigentes (S2-S4, parasympathetic) – anterolateral rectal wall. Traction avulsion, or injury near the seminal vesicles & prostate → ERECTILE dysfunction

Reported male dysfunction 5-90% – and many are dysfunctional BEFORE the operation

- Adam et al: ED 71% post-op vs. 24% pre-op, ejaculatory dysfunction 78% vs. 32%
- T3/T4 and low rectal tumors are independent risk factors
- Dulskas: ED 63.9% post-op vs. 41.7% pre-op
- APR worse than LAR

Sildenafil RCT after proctectomy: 80% improved vs. 17% placebo

- psych evaluation and partner support improve the response to drug therapy

Female infertility:

- 3x after total proctocolectomy in UC – 48% vs. 15%
- meta-analysis: nearly 4x after IPAA
- lap vs. open: NO difference in infertility rates

DELIRIUM

Rates reported as high as 87%

Risk: dementia, age, sensory impairment, dehydration, substance abuse, drug withdrawal (anticholinergics, benzos), sleep-wake disturbance, prolonged hospital or ICU stay

a/w ↑ mortality, ↑ LOS, discharge to a nursing facility

Prevention: repeated orientation, EARLY mobilization, keep hearing aids and glasses on

9 PERIOPERATIVE/ENDOSCOPY: ANASTOMOTIC

CONSTRUCTION

PHYSIOLOGY OF ANASTOMOTIC HEALING

LAYERS

Submucosa = highest collagen content, greatest tensile strength – MUST be incorporated (Halsted)

Serosal apposition w/ inversion of the mucosa (Lembert)

No serosa: esophagus & low rectum → higher leak rates

PHASES

Inflammatory / "lag" (day 0–2): collagenolysis predominates

- anastomosis is **WEAKEST** the first 2 days
- integrity depends **ENTIRELY** on suture/staple until collagen synthesis starts
- collagenolysis liberates proline & lysine for later synthesis

Proliferative (day 2–4): collagen synthesis

- fibroblasts AND smooth muscle cells (muscularis mucosae & propria) – unique to gut

Remodeling: collagen maturation & cross-linking

Tensile strength at 1 wk: small bowel ~100% of expected, colon only ~50%

Mucosal restitution complete by day 3 if mucosa is apposed

- crypt goblet cell mucus layer separates the microbiome from the healing anastomosis

MICROBIOME

Collagenolytic bacteria undermine tensile strength

- *E. faecalis*: degrades collagen, activates MMP9
- *P. aeruginosa*: mutates to ↑ collagenase

The 5 things to minimize: tension, necrosis, air leak, missed submucosa, poor perfusion

FUNDAMENTAL PRINCIPLES

Healthy, well-perfused ends. Airtight. NO tension

Resection extent is dictated by the DISEASE – never compromised by the anastomosis you would prefer

Preop localization: endoscopic description is unreliable, tattoo does not help preop planning

- endoscopic CLIP + plain x-ray = accurate preop localization

The anastomosis is built supine – gravity changes the tension when the pt stands up

MOBILIZATION

SMALL BOWEL

Rarely needs mobilization – intraperitoneal w/ mobile mesentery

Extracorporeal ileocolic: mobilize the ENTIRE hepatic flexure, divide the gastrocolic, off the duodenal sweep

- omental bulk, obesity, large specimen, short mesentery, thick abd wall → dictate extraction site & incision length

Ileoanal reach:

- dissect the root of the mesentery to the pancreas and proximal SMA
- "step ladder" relaxing incisions ant. & post., or windows in the mesentery
- transilluminate to find the arcades before dividing anything
- cadaveric/angiographic study validated the step ladder technique

SPLenic FLEXURE

A left-sided anastomosis has one **FIXED** end – flexure mobilization is the critical skill

Left & transverse mesentery off the retroperitoneum back to the aorta

- divide attachments to kidney, stomach, spleen, inferior border of pancreas

High ligation of the IMA: up to 10 cm of additional length vs. low tie

IMV divided at the inferior border of the pancreas – **ESSENTIAL** for low pelvic reach

Push further: dissect past the IMV to the origin of the middle colic, right of the ligament of Treitz

Take the omentum off the transverse colon – divide the gastrocolic ligament

Release the embryologic hinge of the splenic flexure mesentery

- "unhinges" the 90–180° conformation → straight descent from the middle colics
- allows reach well below the symphysis pubis

Kream et al: consistently low leak rates irrespective of anastomotic height – the key is COMPLETE flexure mobilization

SPECIAL MANEUVERS

Retroileal / Ileal Mesenteric Window (Toupet):

- transverse colon passes **UNDER** the small bowel through a window b/n the SMA and ileocolic a.
- requires full splenic flexure mobilization to the root of the middle colic
- bare area b/n SMA & ileocolic – a 4–5 cm defect accommodates the colon
- cut edge of the transverse mesentery points **LEFT** as the bowel descends right of the aorta

Right Colon De-Rotation (Deloyers):

- right colon as the conduit, based on the **ILEOCOLIC a.**
 - dissect to its origin so it rotates w/o kinking
- cecum & TI rotated in the sagittal plane – cecum to the RUQ, ascending colon caudal
- dorsal surface of the mesentery becomes ventral
- reaches the low pelvis and even the anal canal
- preserves reservoir, water absorption, ileocecal valve – 67% of 48 pts had < 3 BM/day
- **MUST** do an appendectomy – the cecum now lives in the mid-right abdomen

PERFUSION

Clinical assessment: color, motility, mucosal bleeding

Skeletonize & transect the marginal artery at the distal descending colon

- look for BRISK PULSATILE arterial bleeding
- no pulsatile flow → walk proximally until you get it.
THAT is the transection point

ICG Fluorescence Angiography:

- absorbs NIR at 800 nm, protein-bound, stays intravascular → marks microperfusion
- subjective, not yet quantitative
- PILLAR II: altered the level of proximal resection in 8% of cases
- NOT yet shown to reduce leak

CONFIGURATION

Small bowel: end-to-end, or side-to-side (antiperistaltic functional E-E, or isoperistaltic)

Size mismatch after right colectomy → Cheatle slit on the antimesenteric side of the smaller bowel, or go side-to-side

End ileum to side of transverse colon: circular stapler w/ NO intersecting staple lines

Extended right colectomy: isoperistaltic side-to-side is advantageous

Subtotal colectomy: transpose sigmoid to the RLQ, ileosigmoid side-to-side w/ ileum in its native position

Colocolonic after splenic flexure resection: end-to-end lays neatest – wall too rigid for side-to-side

Kono-S (Crohn's):

- divide bowel PERPENDICULAR to the mesentery w/ a linear stapler
- mesentery divided right at the bowel edge – preserves blood supply & enteric nerves
- the two staple lines are sutured together as a “supporting column” – excludes the anastomosis from the mesentery
- antimesenteric longitudinal incisions 7–8 cm, starting 0.5–1 cm off the staple line
- closed TRANSVERSELY, Heineke-Mikulicz fashion
- safety comparable to side-to-side, LOWER recurrent dz

LOW PELVIC ANASTOMOSIS

Narrow pelvis – male, obese – limits the conduit that can cross the pelvic floor

Upper rectum & ileorectal: end-to-end, or side-to-end for size mismatch

LAR syndrome is the functional problem that drives reservoir reconstruction

Colonic J-Pouch:

- 5–6 cm limb, antimesenteric colotomy, 1–2 linear loads, EEA to the anorectum
- ↓ frequency & urgency vs. straight
- some say straight catches up by 2 yrs – others show benefit persisting at 5 yrs
- 24 months of LAR syndrome is enough to make a pt ask for a stoma back

- 30% still get LAR syndrome despite the pouch
- NSQIP: J-pouch had FEWER reoperations and less organ space infection than straight
- side-to-end configuration → better perfused than the cut end; mesenteric bulk fills presacral dead space

Side-to-End (“Baker type”):

- efferent limb 5–6 cm
- function equivalent to J-pouch, SUPERIOR to straight. No difference in morbidity
- FASTER to build than a pouch

Poor function is second only to leak as the reason a low anastomosis gets converted to permanent colostomy

HAND-SEWN ANASTOMOSIS

Suture:

- monofilament: less tissue trauma, less bacterial adherence – but memory, and knots slip
- SLOWLY absorbable (PGA or PDS) – strength persists into remodeling. Permanent NOT needed
- avoid silk & linen – more local inflammation

Technique:

- INVERTING (Lembert). Everting abandoned – more inflammation & adhesions, leak equivalent
- bite 3–4 mm, MUST catch submucosa. Mucosa contributes no strength
- tie secure, not “anemic” – leave room for the edema to come
- continuous is faster. No outcome difference vs. interrupted

Single vs. Two Layer:

- Czerny 2-layer: interrupted Lembert post., continuous full-thickness inner, Connell ant., interrupted Lembert ant.
- 2 layers → longer, aperture stenosis, more ischemia & necrosis
- Cochrane: single = two layer for leak, complications, mortality, LOS
- SINGLE LAYER preferred in most instances

Hand-sewn is the FAIL-SAFE when staplers fail

- after mucosectomy or intersphincteric resection, the anal anastomosis is hand-sewn

STAPLED ANASTOMOSIS

Hultl 1917 established the concepts still in use: tissue compression, B-shaped staples, two overlapping staggered rows – airtight, w/ gaps that let blood through

Titanium = lowest tissue reaction, and greater tensile strength than suture when properly formed

TYPES

Linear noncutting / transverse: staples, then you divide.

Contour® – curved head for pelvic transection, 3 staple lines, closes the specimen side

Linear cutting: side-to-side anastomoses. Lap/robotic versions give 6 rows (3 each side) vs. 4

Circular: end-to-end low pelvic, also end-to-side & side-to-end. Staples AND cuts on ONE firing, 2–3 rows, mucosa inverted

STAPLE HEIGHT – OPEN / CLOSED

- Grey 2.0 / 0.75 mm – mesentery
- White 2.5 / 1.0 mm – vascular
- Blue 3.5 / 1.5 mm – standard
- Gold 3.8 / 1.8 mm – standard/thick
- Green 4.1 / 2.0 mm – thick

Staple too SHORT for the tissue → serosal laceration up to complete staple line failure

COMPRESSION – NAKAYAMA

Pre-compression improves staple form; longer compression = more consistent form

GROSS mismatch (blue load on the pylorus) can NOT be overcome by any firing technique

The TIP of the stapler forms staples less reliably than the base

Specimen-side staple line mirrors the patient side – inspect the specimen to infer the in vivo line

Circular stapler forms reliable B-staples REGARDLESS of pre-compression or degree of closure – parallel closure mechanism

PRACTICAL RULES

Wait longer than the recommended 15 sec – up to 1 minute – before firing

Pause b/n strokes on lap staplers. Powered devices (Sureform, Signia) sense thickness and modulate the stroke

Multiple firings to transect the rectum = RISK FACTOR FOR LEAK

- hunt the aberrant “migratory”/crotch staple at the tip and REMOVE it before the next firing

Open rectal transection: usually a single 30–45 mm firing

Inspect donuts for all layers and intact rings

Air leak test w/ videoendoscopy is a NECESSARY adjunct for any pelvic anastomosis

- also worth doing after rectal transection, BEFORE double stapling

On lap/robotic platforms the end effector sits at a distance from the surgeon – a danger point. Automation must NOT reduce vigilance

COMPRESSION RING

Denans → Murphy Button. Not used in North America

Ring compresses, tissue necroses slowly while healing regains strength, ring passes per anus

No retained foreign body – less inflammation, fewer adhesions, higher burst pressure in pigs

Meta-analysis, 10 RCTs, ~2000 pts: NO difference in leak, stricture, or mortality

- faster return of bowel function, but MORE obstruction (OR 1.87). No advantage over conventional

Cochrane 2012, colorectal, 9 RCTs / 1233 pts: no difference except stricture MORE frequent w/ stapling, and hand-sewn takes longer

Cochrane, ileocolic, 7 RCTs / 1125 pts: stapled leak 2.5% vs. hand-sewn 6%. Cancer subgroup 1.3% vs. 6.7%.

NO difference in the noncancer / Crohn's subgroup HASTA (ileostomy closure, 337 pts): leak 3.0% stapled vs. 1.8% hand-sewn (NS). Obstruction ≤ 30 d 10.3% vs. 16.6% (NS). Stapled faster

German registry, 4062 open right colectomies: leak 3.9% stapled vs. 3.0% hand-sewn – NS. Stapled faster

Danish nationwide, 1414 right colectomies: leak 5.4% stapled vs. 2.4% hand-sewn (adj OR 2.91). 30-d mortality 15.6% w/ leak vs. 2.1% without

European multicenter, 2515 right colectomies: leak 7.4%, morbidity 38%, mortality 2.6% – 10.6% w/ leak vs. 1.6%. Leak predictors: longer op (OR 1.007/min), OPEN (OR 1.9), STAPLED (OR 1.5)

Bottom line: no clear winner. MUST master both

HAND-SEWN VS. STAPLED – THE DATA

10 PERIOPERATIVE/ENDOSCOPY: ANASTOMOTIC

COMPLICATIONS

ANASTOMOTIC LEAK

DEFINITION & GRADING

NO consensus on the clinical criteria – extravasation of retrograde contrast on CT has the highest sens. & spec.

Graded by what it forces you to do:

- A: radiologic leak only, NO change in management
- B: active intervention WITHOUT relaparotomy – abx, percutaneous or transanal drainage
- C: relaparotomy

INCIDENCE

Overall 2–21%

Risk tracks the LEVEL of the anastomosis

- lowest: small bowel & ileocolic
- highest: ileorectal & distal colorectal

RISK FACTORS

Patient:

- DM, hyperglycemia, high HbA1c
- MALE sex, high BMI, tobacco
- IBD, chronic immunosuppression, radiation enteritis
- malnutrition, hypoalbuminemia, active infection

Rectal CA specific:

- more distal anastomosis, neoadjuvant XRT, advanced T stage

Intraop:

- tension, poor blood supply to the ends
- the operating SURGEON is a risk factor – which traits matter is unknown

Bowel prep:

- MBP + oral abx TOGETHER → lower leak. Consistent across multiple NSQIP analyses

Drains:

- unclear whether routine closed-suction drainage lowers leak
- rarely controls the sepsis when a leak actually happens
- but they do NOT cause leaks – IR can reposition one, and it controls a pelvic hematoma

Proximal diversion:

- cited as risk, as protective, and as neutral – UNCLEAR
- does NOT prevent leak. DOES ↓ septic complications, reoperation, and mortality
- may keep a leak from ever declaring itself clinically

Microbiome:

- E. faecalis degrades collagen and activates MMP9 – some strains far more than others
- P. aeruginosa mutates to ↑ collagenase activity
- standard oral/IV abx do NOT eliminate either
- explains the leaks in pts with no other risk factor

PRESENTATION & TIMING

Early: fever, leukocytosis, ↑ pain, suspicious wound or drain output, prolonged ileus

- or nonspecific ileus & low-grade fever, or frank peritonitis & sepsis

Up to HALF present AFTER discharge – a significant number > 1 month out

- LAR for rectal CA: a THIRD become evident > 1 month post-op

Late leaks are insidious – pelvic pain and failure to thrive

DIAGNOSIS

CT w/ rectal contrast is the test

CT before POD 4–5 is unreliable – takes to day 5 for infected fluid to rim enhance

- rim-enhancing collections & specks of free air are frequently equivocal

CRP – NPV 97%:

- POD 3 < 172 mg/L, POD 4 < 124, POD 5 < 144
- open: POD 3 > 209 and POD 4 > 123.5 most predictive
- lap: POD 2 > 146.7 most predictive
- POD 3 CRP < 145 → 93% NPV for 30-day readmission

Procalcitonin – use w/ CRP:

- POD 3 < 2.7 ng/mL → NPV 96.9%
- POD 5 < 2.3 ng/mL → NPV 98.3%

Biomarkers over cutoff in a well-appearing pt → weigh discharge against readmission risk

MANAGEMENT OF LEAK

Driven by: clinical condition, timing, LOCATION, and whether it is CONTAINED

Fecal peritonitis / sepsis:

- emergent OR, washout, source control
- can't safely expose it (obliterative adhesions) → lay drains at the leak + proximal stoma
- small bowel or ileocolic w/ viable, mobile ends → resect & redo
- marginal pt or marginal bowel → end ostomy + mucous fistula, or divided end-loop stoma
- colorectal w/ a significant defect → TAKE IT DOWN, end colostomy
- protect the rectal stump – oversew the staple line, rectal tube, drain over it. Dehiscence is likely
- expect to need a laparotomy for an adequate washout even after a MIS index case

Contained, abscess < 3 cm:

- broad-spectrum abx alone

Larger abscess:

- percutaneous drainage + abx. Diversion by severity
- colorectal/coloanal: drain TRANSRECTALLY through the anastomotic defect into the cavity

Non-op management preserves the primary anastomosis in HALF of leaks

Reoperate at ≥ 3 months so the inflammatory adhesions resolve – waiting longer often heals it without an operation

Transanal repair:

- endorectal advancement flap, dermal flap, or primary suture repair
- TAMIS / TEMS described, ± lap washout

- STRONGLY consider a diverting loop ileostomy if you do this
- often NOT feasible – tissue around a leak is too fibrotic

CHRONIC PRESACRAL SINUS

An unhealed leak off the POSTERIOR aspect of a colorectal anastomosis

9.5% after LAR w/ neoadjuvant chemoradiation

Sx: pelvic pain, fever, rectal discharge, tenesmus

Septotomy:

- divide the wall b/n lumen and tract – unroofs the sinus and makes the cavity part of the lumen
- transanal if distal enough; TEMS/TAMIS for a more proximal septum
- often needs repeating over weeks to months

Fibrin glue:

- transanal or endoscopic injection
- IR can inject as they pull the transrectal drain, once the cavity has narrowed to a tract

Frequently fail → resect the anastomosis, debride, then redo or completion proctectomy

ENDOLUMINAL VACUUM THERAPY

Endoscopic sponge into the presacral cavity – heals the MAJORITY

Outpatient, changed every 2–3 days. Weeks to months to full resolution

May PREVENT presacral sinus formation. Well tolerated and safe

START WITHIN 15 DAYS of diagnosis – salvage use later does far worse

Diversion is commonly part of the strategy, but not universal

Also described: covered intraluminal stents, over-the-scope clip. Small series only – sepsis must already be controlled

OUTCOMES AFTER LEAK

Peri-op mortality 3–14%

Rectal CA: 5-yr overall survival 44–53% vs. 64%, cancer-specific 42% vs. 67%

- traditionally blamed on delayed adjuvant chemo
- also: post-op infection alters peritoneal & serum cytokines → ↑ residual tumor cell migration & invasion, pro-angiogenic genes upregulated

Risk of a PERMANENT ostomy rises the more distal the leak

Worse function – frequency, poorer continence, pad use. Pelvic fibrosis ↓ rectal compliance

Sexual & urinary dysfunction go unreported – ASK, don't wait for it to come up

ANASTOMOTIC FISTULA

Immediate post-op = TECHNICAL error

- posterior vaginal wall caught in the circular stapler → stapled colovaginal fistula
- ureter or urethra incorporated if the rectal stump was not mobilized off them

- reoperation + diversion. Permanent colostomy is the likely end result

Later = a leak that failed to heal

- intra-abdominal → enterocutaneous. High output or persistent low output → resect & redo
- pelvic → abd wall, buttock (persistent transgluteal drain tract), vagina (usually s/p hysterectomy), rarely fallopian tube
- s/p resection for colovaginal or colovesical diverticular fistula – the fresh vaginal or bladder suture line is where an abscess will necessitate
- OMENTAL FLAP in the pelvis b/n the anastomosis and the other suture lines

Rare: fistula to the epidural space → epidural abscess.

Weight loss, low-grade fever, malaise. Source control first, then decompression & debridement

BLIND LOOP SYNDROME

Blind sac of an ANTIperistaltic side-to-side anastomosis dilates over time

Presents YEARS later – bloating, nausea, weight loss, poor appetite, vague pain unrelated to meals

Complications: SIBO, pseudo-obstruction, volvulus, ulcer, bleeding, perforation

Workup is unrevealing – the anastomosis reads as widely patent and within normal limits

- capsule endoscopy or double balloon enteroscopy makes the dx

Rx: resect the anastomosis, redo it END-TO-END

ANASTOMOTIC BLEEDING

Reported in up to 5% of colorectal anastomoses – clinically significant far less

- 2.3% by the definition of Hgb ↓ 2.0 + hematochezia. 0.5–0.8% need an intervention

Most bleed within the FIRST 24–48 hrs and stop on their own

DELAYED bleed → think anastomotic breakdown. Scope or image it

Left-sided: bright red. Right-sided: dark clots. NO abdominal pain

Tagged RBC scan and CTA are NOT needed – the source is not a dilemma

Rx: resuscitate, correct coagulopathy, hold NSAIDs & anticoagulants, transfuse by hemodynamics

If a pouch or coloanal anastomosis bleeds: rectal tube and a 1:100,000 solution of saline + epi for 15 minutes

Endoscopy = PREFERRED intervention:

- minimize insufflation. Disruption is rare
- prep usually unnecessary – rapid purge if too much intraluminal blood
- CLIPPING is safe and effective
- epi injection for non-focal bleeding – but risks ischemia of the rest of the anastomosis
- electrocautery works, but risks anastomotic fistula

Angiography (vasopressin or embolization): SELECTIVE – real risk of anastomotic ischemia & breakdown
Surgical revision stops it in most cases – reserved for endoscopic failure

Prevention:

- Cochrane: hand-sewn 3.1% vs. stapled 5.4% – NOT significant, favors neither
- use the ANTIMESENTERIC border. Clear the mesentery for an end-to-end
- inspect INSIDE a functional E-E before closing the transverse opening
- pulsatile bleeding: figure-of-eight suture, NOT cautery – then use it to evert and inspect the whole staple line
- left-sided: scope checks bleeding and integrity in the same pass

ANASTOMOTIC STRICTURE

Definition: will not admit a 12-mm colonoscope or a rigid proctoscope

Incidence < 10% by that definition – up to 30% if the clinically insignificant ones are counted

LOWER the anastomosis, HIGHER the stricture rate – IPAA and coloanal are the worst

Sx: constipation, cramping, narrow caliber. Overflow diarrhea & incontinence in the extreme

Dx usually within 12 months of resection unless it is recurrent dz

ETIOLOGY

Ischemia, infection, LEAK, radiation, recurrent dz

Proximal DIVERSION – no stool passing to dilate it

Cochrane: left-sided end-to-end STAPLED 8% vs. hand-sewn 2%

- confounded – staplers get used for the low ones. Authors did NOT favor hand-sewn
- ileocolic: stapled = hand-sewn, both low

EVALUATION

After cancer resection, MUST rule out recurrence – biopsy, CEA, PET-CT. Re-biopsy if suspicion stays high

Define the anatomy first – scope through it, or fluoroscopy if you can't

The end-to-side / side-to-side “dog ear” sits at 90° to the lumen and gets misread as a pinpoint stricture

TREATMENT

Digital dilation:

- anal canal, low pelvic, IPAA. Usually under anesthesia
- DRE before reversal after a hand-sewn coloanal – a contrast study misses it if the catheter tip sits proximal
- pt can self-dilate w/ Hegar dilators

Endoscopic Balloon Dilation – FIRST LINE:

- success 67–100%. Best for SHORT strictures, < 2 cm
- Suchan, 94 pts: 67% overall – 88% if the original dz was benign, only 59% if malignant
- Biraima, 76 pts: recurrence 11% / 22% / 25% at 1 / 3 / 5 yrs. Half done with 1–2 dilations

- secondary success 97% w/ repeat dilation – only 2 ever needed surgery
- recurrence predictors: lumen < 10 mm, HAND-SEWN anastomosis, > 2 dilations required
- complications minor – bleeding, one perforation

Endoscopic Electrocautery Incision (EECI):

- radial incisions through the fibrotic mucosa at the most resistant point, then dilate
- via endoscope, TEMS, or TAMIS. Success 90–100% in small series
- for the short fibrotic stricture that recurs after balloon

Not worth it:

- SEMS – they migrate
- transanal circular stapler resection – only for mild strictures that admit the anvil. Not adopted

Surgical Revision:

- for LONG (> 2 cm), irregular, angulated strictures
 - ureteral stents – the left ureter is adherent to colon and mesentery
 - KEY: get BELOW the fibrosis to soft, pliable colon or rectum
 - perforation at the anastomosis during takedown is common
 - > 11 cm from the verge → new stapled colorectal. < 11 cm → hand-sewn coloanal, almost universally
 - straight coloanal usually; J-pouch if the pelvis allows. Immediate or delayed (Turnbull-Cutait)
 - can mucosectomize and leave a rectal muscular tube – Soave-like
 - DIVERT
 - success 57–100%, pooled 79%. Continuity restored in ~80%
 - BUT 17% incontinent and ~60% w/ some LAR syndrome. Mean age 58 – older pts do worse
- A well-constructed permanent colostomy restores quality of life and is a legitimate answer, not a failure

11 ANORECTAL DISEASE: HEMORRHOIDS

~4% of the US population; > 3 million office visits/yr, > \$770 million

Peak age 45-65, M = F. Higher socioeconomic status and whites

Only 40% of those with enlarged hemorrhoids are symptomatic

Only 5-10% will need operative hemorrhoidectomy

ANATOMY

- Treitz Muscle: anal submucosal muscle
- Vascular Cushions: arteriovenous, submucosal. Contribute up to 20% of baseline continence, engorge on Valsalva
- 3 main cushions: L. Lateral, R. Ant, R. Post
 - most have additional small accessory cushions
- inflow from terminal branches of the superior & middle rectal aa.
- outflow via sup/mid/inf rectal vv. → internal pudendal v. → IVC

Internal: proximal to dentate, columnar mucosa,

VISCERAL innervation – pressure, no pain

External: distal to dentate, anoderm, SOMATIC innervation – exquisitely painful

Mixed: both components

GRADING (INTERNAL)

- I: engorged, no prolapse below the dentate
- II: prolapse on Valsalva, reduce SPONTANEOUSLY
- III: prolapse, require MANUAL reduction
- IV: prolapsed, NOT reducible

Strangulated: grade IV, edematous to the point of compromised supply → necrosis / gangrene

Skin tag: redundant anoderm, the residue of a resolved thrombosis

ETIOLOGY

Straining – hard stool or pelvic outlet obstruction. Western commode vs. squatting

Repetitive Valsalva – weightlifters, COPD, chronic cough

Impaired venous return – late pregnancy, pelvic outlet dysfunction

Tissue level: MMPs, VEGF, nitric oxide synthase, neovascularization. GWAS mutations described

- Anemia from hemorrhoids is RARE – always scope these patients to rule out malignancy

SYMPTOMS

Internal: painless BRIGHT RED bleeding, protrusion, pressure, mucus discharge, seepage

- PAIN should make you question the diagnosis – think thrombosis, fissure, abscess
- internal hemorrhoids hurt only when prolapsed and strangulated – not a subtle finding

External: itching, irritation, moisture, hygiene difficulty.

Painless UNLESS thrombosed

- thrombosis: blue/purple firm nodule at the anal verge, clot may erode through the skin

EXAMINATION

- prone jackknife – if not tolerated, Sims (left lateral)
- inspect, DRE for sphincter tone and other pathology, then ANOSCOPY – side-viewing best
- document anterior-posterior / right-left, not o'clock
- strain on the commode – grades the prolapse and excludes full-thickness rectal prolapse
- no labs or imaging needed for diagnosis

Hematochezia blamed on hemorrhoids = the most common MISSED colorectal cancer diagnosis

- colonoscopy if age > 45, change in bowel habits, anemia, weight loss, or FHx / Lynch

MEDICAL MANAGEMENT

Trial conservative therapy 6-8 weeks, then reassess in the office

Stool habits:

- no prolonged sitting – defecation should take a few minutes; get up and return with the urge
- foot stool to approximate squatting

Stool texture:

- soluble fiber, goal 25-50 g/day – diet alone won't do it, supplement
- water ≥ 64 oz. Fiber is a sponge – it fixes hard AND loose stool

Hygiene:

- warm soaks / sitz 15 min – relaxes the floor, reduces prolapse and edema. No salts or emollients

Topicals:

- no quality data. Acceptable to continue if the pt perceives benefit
- lidocaine, hydrocortisone, pramoxine
- limit to 7 days – dermatitis, and steroids thin the anoderm

Oral:

- NSAIDs
- Phlebotonics (flavonoids): ↓ pruritus and bleeding, ↑ vascular tone. Europe & Asia – NOT FDA approved

OFFICE-BASED PROCEDURES

For grade I-II and SOME grade III with BLEEDING as the dominant symptom

Large prolapsing grade III → operation, or downgrade first with conservative measures

Cannot tolerate office anoscopy = not a candidate

Rubber Band Ligation:

- band the redundant mucosa ≥ 2 cm ABOVE the dentate line
- tissue necroses and the band sloughs at 5-7 days, leaving an ulcer that scars in
- McGown suction ligator – no assistant needed
- more than one column can be banded safely, but ↑ pain and urinary retention
- pressure is expected for 1-2 days; PAIN means the band is too distal
- bleeding peaks POD 5-7. ~20% need repeat banding

- hold anticoagulants (ASA 80 mg is OK) – though clopidogrel case-control showed no difference, 3.75% vs. 2.78%
- PELVIC SEPSIS: fever, urinary retention, swelling, pain – rare, rapidly progressive, FATAL if missed

Infrared Photocoagulation (IPC):

- tungsten-halogen lamp, polymer probe tip converts light to heat
- grade I-II. Apply to the APEX 3-4x, 0.5-2 sec pulses, 2.5-3 mm depth
- can treat multiple columns in one sitting

Bipolar Diathermy:

- grade I-III. 20 watts pulsed, 2.2 mm depth, at the apex
- RCT of 122: bipolar ≈ IPC
- IPC vs. RBL crossover: less analgesic use and bleeding with IPC at 24 hrs

Sclerotherapy:

- oldest technique, 1869. Grade I-III
- 1-1.5 mL into the SUBMUCOSA at the base, 21-ga spinal needle
- hypertonic saline or 5% phenol in oil
- too superficial → mucosal damage; too deep → pain, infection, abscess
- SAFE on anticoagulation. Comparable to RBL in trials

Historic / abandoned: Lord's dilation (sphincter injury), cryotherapy, direct-current electrotherapy

THROMBOSED EXTERNAL HEMORRHOID

Pain peaks ~48 hrs and improves by day 4 – spontaneous improvement starts within 72 hrs

- within 72 hrs and painful → EXCISE
- improving / beyond 72 hrs → OBSERVE. Excising then only prolongs the pain
- persistent lump and pain for days-weeks with no improvement → also excise
- not a candidate for excision → topical nifedipine, ↓ pain by relieving sphincter spasm

Technique – office, does NOT need the OR:

- betadine, 1% lidocaine ± epi 1:200,000 by 27-ga at the base
- EXCISE the whole hemorrhoid and clot in an ellipse, superficial to the sphincter – do NOT just incise and evacuate
 - excision → less recurrence and less pain than incision
- stay in the plane – minimal blood loss. Don't go wide on the anoderm
- pressure, silver nitrate. Leave the wound to close secondarily

OPERATIVE HEMORRHOIDECTOMY

For failed medical / office therapy, grade III & IV, large external component, extensive disease, or incarcerated / strangulated / gangrenous

No bowel prep, no pre-op antibiotics. Enema optional

Position and anesthetic per surgeon preference. Always start with inspection, DRE, anoscopy

Preserve mucocutaneous BRIDGES b/n excision sites – this is what prevents stenosis

1-3 columns per operation. Incidental pathology finding ~1%

Ferguson (Closed): most common operation in the US.

Elliptical incision from perianal skin to the anorectal ring, dissect off the sphincter, suture ligate the pedicle, amputate, close running/locking with the same suture. Small bites of sphincter close the dead space. Leave a few mm of the anal margin open for drainage

Milligan-Morgan (Open): UK. Identical excision, wounds left to heal by secondary intent over 4-8 weeks

- meta-analysis of 11 RCTs: CLOSED had less pain, faster healing, less post-op bleeding
- complications, recurrence, infection the same. Individual RCTs show little difference. Both appropriate

Whitehead: circumferential excision of hemorrhoidal tissue and anoderm just proximal to the dentate.

ABANDONED – stenosis, mucosal ectropion ("Whitehead deformity"), incontinence

Energy devices:

- bipolar (LigaSure) vs. conventional: less EARLY pain, gone by day 14. Faster operation (Cochrane)
- ultrasonic shears similar. Head-to-head RCT: pain scores and outcomes the SAME
- laser and diathermy: no pain benefit, higher cost

Stapled Hemorrhoidopexy (PPH):

- circular stapler excises submucosa ABOVE the dentate → pexy, not excision. Purse-string 2 cm above the dentate
- does NOT address external disease
- inspect and palpate the VAGINA before firing
- FALLEN FROM FAVOR
 - RCT of 777: less pain, but WORSE QOL, recurrence 32% vs. 14% at 24 months
 - Cochrane 12 trials / 1097 pts: more reoperation, less likely to be asymptomatic
 - systematic review 14,232 pts: median complication rate 16.1%, 5 deaths
 - 40 published rectal perforations 2000-2009 – 35 needed laparotomy + diversion, 4 died
 - unique complications: rectovaginal fistula, staple line bleeding, staple line stricture

THD / HAL-RAR (Doppler-guided dearterialization):

- Morinaga 1995. Doppler anoscope identifies and suture ligates the 4-6 feeding arteries
- add a running mucopexy from the ligation point down to just above the dentate for prolapse
- no excision, so less pain in theory
- systematic review 28 studies / 2904 pts: pooled recurrence 17.5%, highest in grade IV. Bleeding 5%, reintervention 6%

- RCT vs. RBL for grade II-III: recurrence, symptoms, complications, QOL and continence all EQUAL
- MORE early pain with HAL, more expensive, NOT cost-effective vs. RBL
- meta-analysis vs. stapled: MORE recurrence with HAL
- vs. excisional: no difference in recurrence, but long-term data lacking

Bottom line: excisional hemorrhoidectomy remains the GOLD STANDARD – the less painful alternatives all recur more

PAIN CONTROL & POST-OP CARE

Pudendal block for every case, regardless of GETA vs. MAC

- 1:1 mix of 1% lidocaine and 0.25% bupivacaine, 40-60 mL total
- epi 1:200,000 ↑ max dose and duration
- liposomal bupivacaine: time to first opioid 19 vs. 8 hrs
 - volume-expand 20 mL with 20 mL of 0.25% bupivacaine
 - NEVER expand in lidocaine – it drives bupivacaine out of the liposomes → toxicity

Non-narcotic mainstay: ibuprofen 600 mg TID, acetaminophen ≤ 4 g/day, diazepam for spasm

- narcotic for breakthrough only. A 5-10 day supply is optimal
- prescribe an opioid WITHOUT acetaminophen
- metronidazole: mixed data, benefit disappears on sensitivity analysis

Topicals post-op:

- 5% baclofen: ↓ pain and analgesic use at 1 and 2 weeks
- GTN: ↓ pain POD 3 and 7 (not day 1), OR 3.57 for healing at 3 weeks. Headache
- lidocaine helps when combined with diclofenac or nifedipine

AVOID constipation – fecal impaction post-op is devastating. But avoid diarrhea too – frequent loose stool delays healing

COMPLICATIONS

Urinary retention:

- 1-15%, the most common. #1 reason for failed same-day discharge
- higher after spinal and after HAL
- limit IV fluid to < 500 cc, use local liberally

Post-hemorrhoidectomy hemorrhage:

- delayed bleed 0.9-10%
- passing a whole BM of clots → EUA
- culprit vessel often not found; if found, figure-of-eight absorbable suture
- evacuate residual clot with rigid proctoscopy so the picture stays clear
- male sex and the individual surgeon are independent risk factors

- no difference open vs. closed, or with bipolar

Anal stenosis:

- from excess anoderm removal, most often after EMERGENCY hemorrhoidectomy with inadequate bridges
- bulk laxatives, dilation, anoplasty

Fecal incontinence:

- rare. Sphincter stretch, direct injury, or loss of cushion bulk. Proper technique avoids it

SPECIAL POPULATIONS

Strangulated:

- if not necrotic: circumferential local, reduce, bed rest
- BEST option unless prohibitive risk = expeditious excisional hemorrhoidectomy
- NECROSIS → excision is mandatory; leave wounds OPEN if tissue is edematous or devitalized
- reduction + banding: 13.5% still went on to hemorrhoidectomy

Pregnancy:

- 3rd trimester prolapse 14.4%, postpartum thrombosis 14.6%
- almost always resolve after delivery – operate only for strangulation or a very symptomatic thrombosis
- LOCAL anesthesia, LEFT LATERAL position to keep off the IVC

Portal hypertension:

- rectal VARICES and hemorrhoids are DISTINCT – hemorrhoid symptoms occur at general-population rates
- varices bleed far less than esophageal varices
- if they bleed: decompress the portal system FIRST – medical, TIPS, shunt, transplant
- sclerotherapy and suture ligation only after that, or when all else is exhausted

Crohn's:

- distinguish from Crohn's perianal skin tags
- rectal inflammation or active anorectal Dz → conservative management, do NOT operate
- well controlled, no proctitis: Wolkomir & Luchtefeld 90% healing
- Karin: 13 pts, grade III, THD – 10 of 13 symptom-free at 18 months

Immunocompromised:

- transplant, steroids, chemo, HIV. Poor healing and infectious complications
- if the immunocompromise will resolve, wait it out
- ongoing immunosuppression: medical management first, intervene only after failure
- RBL and excisional hemorrhoidectomy are SAFE in HIV on HAART with acceptable CD4

12 ANORECTAL DISEASE: ANAL FISSURE AND ANAL

STENOSIS

ANAL FISSURE

EPIDEMIOLOGY

Linear tear, dentate line → anal verge

90% posterior midline

- ant. midline: 10-25% of women, 1-8% of men
- 3% both ant & post

Lateral / Atypical: think IBD, TB, HIV, syphilis

Acute: < 6 weeks

Chronic: > 6 weeks – indurated edges, visible IAS at base, hypertrophied papilla, sentinel tag

- pain persists even after local anesthetic

ETIOLOGY

Hard stool, or frequency from diarrhea

- only 13% actually report constipation

High resting pressure → ischemia

- anoderm supplied by inf. rectal aa. traversing the IAS
- perfusion inversely related to IAS pressure
- paucity of arterioles in the posterior midline
- NOS deficiency found in chronic fissure specimens

Postpartum: 11% of chronic fissures

- anterior midline, and NORMAL or LOW pressure
- do NOT use pressure-lowering treatment in these

SYMPTOMS & DIAGNOSIS

Pain during & after defecation, bleeding

- razor blades vs. glass shards, fear bowel movements

History & inspection – DRE and anoscopy not advised if fissure seen

If multiple or lateral → EUA + biopsy

Differential: abscess, fistula, TB, STD, IBD, CA

MEDICAL MANAGEMENT

Conservative:

- stool softeners, psyllium, sitz baths, topical lidocaine
- healing 16-31%
- lidocaine = pain relief only, no healing benefit
- maintenance fiber ↓ recurrence

Calcium Channel Blockers:

- FIRST LINE for chronic fissure
- nifedipine or 2% diltiazem gel tid x 3 months
- healing close to 90%
- equivalent to nitrates with fewer side effects

Topical Nitrates:

- ~50% healing, 13.5% over placebo
- ~50% of those healed will recur
- largely abandoned – headache

Botulinum Toxin:

- SECOND LINE, when topicals fail
- 20-100 IU, healing 27-96%
- works better as 2nd line than as 1st
- temporary incontinence to flatus up to 18%
- meta-analysis: no clear advantage over nitrates or placebo
- vs. LIS: lower healing, but lower incontinence

No medical therapy beats surgery for healing – all beat it for continence

OPERATIVE TREATMENT

Lateral Internal Sphincterotomy (LIS):

- Gold Standard for chronic fissure with HIGH tone
- healing 88-100%, incontinence 8-30% (persistent in 3-7%)
- Tailored (to apex of fissure) PREFERRED over traditional (to dentate)
 - less incontinence, healing comparable
- open vs. closed: no difference
- 1st line surgical if no obstetric injury, IBD, prior anorectal surgery, or weak sphincter
- recurrence 0-15%, from insufficient division
 - can repeat on the contralateral side

Advancement Flaps:

- FIRST LINE for fissure with NORMAL or LOW tone
- typically women after a difficult vaginal delivery
- dermal V-Y or house flap (see Anal Stenosis)
- 98% healing regardless of tone, but 6% get a fissure at a new site
- RCT: LIS 84% / V-Y 48% / LIS + V-Y 94% healing

Anal Dilatation:

- historic only
- less effective than LIS, higher incontinence (OR 4.03)
- *Pneumatic balloon (40 mm, 20 psi x 6 min): healing 83% vs. 92% LIS, incontinence 0% vs. 16% at 2 yrs*

Fissurotomy & Fissurectomy:

- NOT standard treatment
- fissurotomy (open the tract running to the sentinel tag): 98% resolution
- fissurectomy worse than sphincterotomy (OR 8.07 for failure)
- fissurectomy + Botox, or + isosorbide dinitrate: near 100% healing

SPECIAL SITUATIONS

Crohn's:

- deep, painful ulcerations
- medical therapy resolves > half
- avoid surgery – poor healing, perianal deformity

HIV & STD:

- biopsy for the causative organism
- deep broad-based ulcers OR normal appearance
- NOT associated with hypertonicity
- debridement + intralesional steroid short term
- optimize antiretroviral therapy long term

BOTTOM LINE

Acute → conservative

Chronic → CCB + fiber + sitz baths. Fails → Botox

High tone → LIS. Low tone or shaky continence → advancement flap

ANAL STENOSIS

CAUSE

Narrowing of the anorectal junction, canal, or margin

- pliable tissue replaced by fibrosis
- usually loss of anoderm DISTAL to the dentate line

Hemorrhoidectomy = 90% of all cases

- rate 1.5-4%, up to 19% in recent reports
- classic: excising acute swollen grade IV disease without preserving anoderm

Others: pull-through (16%), stapled hemorrhoidopexy, mucosectomy/IPAA, coloanal, Crohn's, radiation, chronic laxatives, STD/TB, Paget's & Bowen's excision, congenital

SYMPTOMS

Difficult, painful, or incomplete evacuation

Impaction, narrow caliber, tenesmus, overflow diarrhea, seepage

EVALUATION

Office exam often diagnostic – findings don't always track symptoms

EUA to define extent and biopsy

- relaxes under anesthesia = FUNCTIONAL
- stays tight = ANATOMICAL

CLASSIFICATION (MILSOM & MAZIER)

Severity:

- Mild: exam with finger or medium Hill-Ferguson
- Moderate: needs dilation to examine
- Severe: can't admit little finger or small Hill-Ferguson

Level:

- Low: ≥ 0.5 cm distal to dentate (65%)
- Mid: within 0.5 cm either side (18.5%)
- High: ≥ 0.5 cm proximal (8.5%)

Extent: *localized / diffuse / circumferential*

Morphology: *diaphragmatic (IBD), ringlike (< 2 cm), tubular (> 2 cm)*

TREATMENT

Asymptomatic stenosis needs no correction

Treat any inflammatory or infectious cause, and any associated fissure, first

NONOPERATIVE

For mild, sometimes moderate

Fiber + softeners – a soft bolus stretches the canal naturally

Dilation:

- office Hegar + local gel then home dilations, or first dilation under anesthesia
- 2 rules: no deep fissures in the anoderm, no disruption of the IAS
- aggressive dilation = brief relief, then worse anoderm & sphincter fibrosis
- better for SHORT strictures
- check anastomoses on DRE within 2 weeks post-op – catch it before it fibroses

- Crohn's: ~50% respond, ~50% go on to proctectomy or diversion

SURGICAL

For severe, and persistent moderate

Access with a small Hill-Ferguson + gentle dilation. Lone-Star retractor

Biopsy anything suspicious – chronic wounds can transform

Must address BOTH components

- anoderm repair fails if IAS fibrosis is left alone
- LIS fails if the anoderm is left stenotic

Transverse Closure after Scar Excision:

- short strictures, Heineke-Mikulicz
- simplest, but high restenosis. Convert to Y-V if tension

Rectal Advancement Flap:

- PROXIMAL & MID canal
- modification of Martin's anoplasty
- include muscularis propria or the flap dies
- advance only to the intersphincteric groove – further gives ectropion

Y-V Flap:

- MID & DISTAL. Limbs ≥ 5 cm
- healing 64-100%. Tip ischemia if under tension

V-Y Flap:

- triangular island, all edges free, donor site closed

Diamond (Rhomboid) Flap:

- Caplin & Kodner. Donor site closed

House Flap:

- Christensen. Wider flap, healing > 90%

U Flap (Island Anoplasty):

- Pearl. Donor site left OPEN to granulate

Rotational S-Plasty:

- circumferential or large defects, e.g. after Paget's or Bowen's excision

Farid RCT, house vs. rhomboid vs. Y-V: house had the largest caliber at 1 yr and the best QOL. Y-V fastest (35 vs. 62 min)

Last resort = diverting colostomy

PERIOPERATIVE

Bowel prep + oral antibiotics – the flap creates a suture line

Prone jackknife

2-0 Vicryl for strength, 3-0 for approximation. No dead space

Do NOT inject epinephrine into the flap – tip ischemia

- Aftercare: don't sit on it, dry dressings, seen in office by day 5
- PREVENT diarrhea – liquid stool is worse for flaps than bulky stool
- localized dehiscence is common, manage in the office

PREVENTION AT THE HEMORRHOIDECTOMY

Preserve mucocutaneous bridges > 1 cm

Don't chase every abnormal cushion – band or ligate the third pedicle instead of excising it

Caution with acute grade IV – anoderm is swollen, and
local anesthetic inflates it further → over-resection
Anchor rectal mucosa to the IAS at the dentate line rather
than closing each wound vertically
Avoid anterior and posterior midline resection. Minimize
thermal injury

13 ANORECTAL DISEASE: CRYPTOGLANDULAR

ABSCESS AND FISTULA

ANORECTAL ABSCESS

EPIDEMIOLOGY & PATHOPHYSIOLOGY

Incidence 8.6-20 per 100,000. M:F 2.4-3:1. Mean age 40

90% cryptoglandular – glands at the dentate line b/n the anal papillae & columns of Morgagni

- extend into submucosa, IAS, intersphincteric space, EAS to varying degrees
- inspissated debris → infection tracks along the gland or by path of least resistance
- Eisenhammer, 1950s

10%: Crohn's, obstetric injury, fissure, TB, sarcoid, HIV

POTENTIAL SPACES

Perianal, ischiorectal, intersphincteric, supralelevator, submucosal, deep & superficial postanal

Ramanujam (n=1023): perianal 42.7%, ischiorectal 22.7%, intersphincteric 21.4%, supralelevator 7.3%

- perianal + ischiorectal = 65-80% of all

Horseshoe: posteriorly through the deep postanal space → bilateral ischiorectal spaces

Supralelevator: more often from an INTRAABDOMINAL source (divertic, CA, Crohn's) than cephalad cryptoglandular

DIAGNOSIS

Hx & physical sufficient for perianal & ischiorectal –

imaging NOT indicated for the classic presentation

Intersphincteric: intense anal pain, NO external findings, won't tolerate DRE

Image only if: unclear Dx, isolated intersphincteric, prior CA / XRT / Crohn's / anorectal surgery / trauma, or concern for horseshoe or supralelevator extension

TREATMENT – I&D

Expedient I&D. Antibiotics are NOT a substitute for drainage

Office or ER drainage PREFERRED – expedites sepsis control

- wheal of local, decompress the cavity through the wheal w/ a large needle, more local, then small incision
- take to OR if: deep / nonpalpable, tissue necrosis, or pt won't tolerate bedside

Incise over the fluctuance. Large cavity → incise the portion CLOSEST to the anal verge

- keeps the tract short if a fistula follows

Intersphincteric: internal drainage at the dentate line via sphincterotomy if no external fluctuance

Supralelevator – route dictated by ORIGIN

- intraabdominal → transabdominal (IR) or transrectal
- cephalad from intersphincteric → TRANSRECTAL
- cephalad from ischiorectal → TRANSCUTANEOUS
- wrong route creates an iatrogenic suprasphincteric fistula

Horseshoe: bilateral transcutaneous ischiorectal drainage + division of the anococcygeal ligament to enter the deep postanal space

POST-DRAINAGE CARE

Do NOT pack – unpacked wounds are less painful and heal faster

Mushroom-tip catheter for large or deep cavities – keeps the external opening, shrinks the incision

Sitz baths 10-15 min bid-tid + external gauze. Nothing else

POST-DRAINAGE ANTIBIOTICS

Guidelines still recommend AGAINST routine antibiotics

- reserve for extensive cellulitis, sepsis, immunocompromise
- augmentin, or Bactrim, or quinolone + flagyl. Prior MRSA → Bactrim or clinda

New data that abx ↓ fistula formation:

- Ghahramani RCT (n=307): cipro + flagyl ↓ fistula 30% → 14.1%
- Lohsiriwat: 48% → 17%
- Mocanu meta-analysis: 36% ↓
- NOT reproducible – other series show no protective effect

Culture only if antibiotics are planned or a resistant organism is suspected

FISTULA-IN-ANO

PROGRESSION

100% of cryptoglandular abscesses have a path from the dentate line to the cavity

30-70% have an identifiable tract at drainage – only ~30-35% become a true fistula

- ↑ in nondiabetics and age < 40
- no difference by sex, smoking, HIV, or periop antibiotics

Do NOT perform primary fistulotomy at the time of abscess drainage

- inflammation obscures the degree of muscle involvement → unnecessary sphincter division

PARKS CLASSIFICATION

Intersphincteric 24-45% – through IAS only, then the intersphincteric space to skin

Transsphincteric 30-60% – through IAS & EAS

- Low: ≤ 30% of EAS. High: > 30% of EAS

Suprasphincteric 2-20% – IAS, cephalad in the intersphincteric space, then over the EAS into the ischiorectal space

Subcutaneous / submucosal 2-3% – no sphincter involvement

Extrasphincteric – NOT cryptoglandular. Intraabdominal source, Crohn's, trauma, or a mis-drained supralelevator abscess

Neither the abscess location nor the external opening predicts the degree of sphincter involvement

GOODSALL'S RULE

External opening POSTERIOR to the transverse anal line
→ curves medially to a posterior midline internal opening

ANTERIOR → radial, straight to the nearest crypt

Correct ~80% overall

- posterior 91%, intersphincteric 93%
- anterior 69%, transsphincteric 68%

Exception: posterior tracts that swing anteriorly before reaching skin

SIMPLE VS. COMPLEX – ASCRS SPTF

Complex: > 30% of EAS, anterior in a female, multiple tracts, recurrent, preexisting incontinence, prior XRT, Crohn's

Simple → sphincter-dividing surgery at initial presentation is acceptable

All others → sphincter-preserving, usually beginning with a draining seton

IMAGING

MRI = GOLD STANDARD when imaging is indicated

- body coil now surpasses endoanal coil at 1.5T & 3.0T; no tract cannulation needed
- Konan, 3T: 83.1% concordance w/ OR findings, but changed management in only 33.8%
- changed management in complex 54.4% vs. 5.2% simple; external opening > 2 cm from verge 47.1% vs. 10.2%; horseshoe 66.7%

EAUS: internal opening 80-90%, equal to MRI. Secondary extensions 67-80% vs. 90% for MRI

- dilute H₂O₂ as contrast ↑ internal opening detection, classification, supralelevator extension
- highly operator dependent – can be done intraoperatively and change the operation

CT alone not helpful. CT fistulography inferior to MRI

- classification 73.1% vs. 92.7%; internal opening 68.2% vs. 85.3%

Fistulography largely obsolete

NO imaging for straightforward cryptoglandular Dz

EUA & INTRAOPERATIVE IDENTIFICATION

EUA is the first step – MAC sedation, prone jackknife

Blunt probe from the external opening, guided by the opposite hand at the internal opening

- must pass smoothly – force creates a false passage

Do NOT “pop through” the last layer of mucosa – a false internal opening → recurrence

H₂O₂ or methylene blue via the external opening: internal opening found in 83%

ABANDON the case if the internal opening is not identified – better than injuring the sphincter

Rare malignant degeneration of a chronic fistula – biopsy anything suspicious, follow long term

SPHINCTER-DIVIDING OPTIONS

Fistulotomy:

- the ONLY surgical option recommended for the simple fistula

- recurrence 0-9%, incontinence 0-37%
- lay open incl. secondary tracts, or lay open the primary tract + counter-drain the secondaries
- Marsupialization: healing 6 vs. 10 wks, post-op bleeding 36% vs. 46%
- Fistulectomy NOT better – same recurrence & incontinence, bigger wound, bigger sphincter defect, slower healing
- role only for a blind-ending sinus that does not cross the sphincter

Modified Hanley:

- posterior midline fistulotomy into the deep postanal space + counter-drainage of bilateral horseshoe tracts
- reserve for FAILED sphincter-sparing repair
- incontinence low despite the amount of EAS divided

Cutting Seton:

- serially tightened, cuts through leaving scar behind. Weeks to months
- recurrence 0-10%, incontinence 0-67%
- Ritchie (n=1460): 12% – rises to 32% excluding papers that never reported continence, 22% excluding medicated setons
- incontinence ↑ with a more proximal internal opening
- Vial: IAS preserved vs. divided – recurrence 5% vs. 3%, incontinence 5.6% vs. 25.2% → do NOT divide the IAS
- last resort only, when other options are exhausted

Fistulotomy + Primary Sphincter Reconstruction:

- fistulotomy ± fistulectomy, then end-to-end sphincteroplasty
- healing 91-96%, incontinence 2-13% – post-defecation soiling the MC type
- worse w/ recurrent fistula, complex tract, secondary tracts, prior seton

SPHINCTER-SPARING OPTIONS

Draining (Loose) Seton:

- sepsis control + tract maturation. Very well tolerated, minimal risk
- also viable as DEFINITIVE Rx – internal opening migrates distally out of the high-pressure zone
- Emile: recurrence ~10%, incontinence 3%. Worse w/ recurrent, supralelevator, anterior
- Kelly (n=200): 100% initial clearance, 6% recurrence, 96% tolerance. Seton exchanged q3 months, median 3 changes
- Omar RCT: EAS-sparing rerouting seton vs. conventional – recurrence 3% vs. 13% (NS), healing 46 vs. 103 days

Endorectal Advancement Flap (ERAF):

- FIRST LINE for complex fistula. Success 60-100%
- wide-based mucosa/submucosa ± circular muscle. Adequate mobilization, NO tension
- more muscle → better flap viability but MORE incontinence, up to 35%

- recurrence a/w smoking, recurrent Dz, Crohn's, prior horseshoe abscess, ↑ BMI
- Contraindications: Crohn's, undrained sepsis, persistent secondary tracts, fistula > 3 cm, malignancy or XRT etiology, anorectal stricture
- Yellinek: rhomboid 64% vs. elliptical 62% – no difference. Curettage = fistulectomy of the tract
- repeat ERAF feasible, higher recurrence than the first
- adding fibrin glue or a plug makes it WORSE. PRP may help
- Anocutaneous (dermal) flap – house, diamond, or V-Y. Avoids ectropion. Success 50-70%, incontinence 10-20%

Ligation of Intersphincteric Fistula Tract (LIFT):

- Rojanasakul, 2007. FIRST LINE for complex fistula
- intersphincteric incision, dissect to the mature tract, divide & doubly ligate. Wounds left open to drain
- initial series 94.4% healing / 0% incontinence; 251 pts 87.7%
- overall 61-94%, change in continence rare
- failure usually converts to an INTERSPHINCTERIC fistula → salvage w/ simple fistulotomy
- recurrence a/w a SHORTER tract
- Madbouly RCT, LIFT vs. ERAF: 94% / 91% initial, falling to 74% / 66% at 1 yr – late failure is real, follow ≥ 12 months
- Variants: BioLIFT 80.2% vs. 81.9% (cost, no gain), LIFT-PLUG 94.0% vs. 83.9% (22 vs. 30 d), LIFT plus 85% vs. 81%. None yet recommended over standard LIFT

Fibrin Glue:

- debride the tract, then inject. Repeatable, no continence risk
- success 14-94%, falls as complexity ↑
- NOT first line – reasonable 2nd line or when nothing else is feasible

Fistula Plug:

- Johnson 2006: 87%. Subsequent series 24-88%
- variable results + cost → not a first-line option
- no benefit added to ERAF

NOVEL THERAPIES

FiLaC – laser tract closure:

- radial diode laser fiber after mechanical debridement
- initial 77-82%, subsequent series 33-71% primary success
- better w/ intersphincteric type, tract < 30 mm, prior seton
- incontinence 1.7%

VAAFT:

- fistuloscope w/ glycine-mannitol irrigation, fulgurate the walls & secondary tracts, brush debride, close the internal opening
- Garg meta (786 pts): 76% success, 16.2% complications, no continence change

- Emile meta (788 pts): 86.8% at 9 months, 4.8% complications
- recurrence by closure method: staple 15.3% < suture 17.7% < advancement flap

Stem Cells:

- Garcia-Olmo phase II: adipose-derived + fibrin glue 71% vs. glue alone 16%, RR 4.43 – but fell to 62.5% at 1 yr
- FATT phase III (n=200): NO difference b/n stem cells, stem cells + glue, and glue alone (~40% at 6 mo, ~50% at 1 yr)

OTSC Proctology Clip:

- nitinol clip placed endoscopically over the internal opening
- small series 60-93%, worse w/ prior fistula surgery
- usually requires the clip cutter for removal. Not yet routine

BOTTOM LINE

Abscess → drain NOW, in the office if you can. No routine antibiotics, no packing

Do NOT lay open a tract at the time of abscess drainage

Simple fistula → fistulotomy. Complex → draining seton, then ERAF or LIFT

Priorities: control sepsis, cure, continence – but CONTINENCE may outrank cure

- a pt lives better with an indwelling loose seton than with incontinence

When one sphincter-sparing method fails, move to another. Consent for failure up front

14 ANORECTAL DISEASE: RECTOURETHRAL AND

COMPLEX FISTULAS

RECTOURETHRAL FISTULA (RUF)

ETIOLOGY

Vast majority are acquired & iatrogenic, from treatment of prostate CA

- congenital RUF is a neonatal problem, separate entity
- Radical prostatectomy: RUF 1-6%, the same open, lap, or robotic
- prostatic urethra separated from the ant. rectal wall only by Denonvilliers' fascia & the prostatic capsule
- typically at the vesicourethral anastomosis, from unrecognized rectal injury or a failed rectal repair
- rectal injury at prostatectomy 0.1-9%; 54% of RUF pts had an overt rectal injury
- other RF: age, prior TURP, bacterial prostatitis, prior hormonal Rx, perineal approach

XRT drives both incidence and complexity

- ionizing radiation → microvascular injury, mucosal ischemia, fibrosis
- pre-1997 < 4% of RUF had XRT; 1998-2012 > 50% did
- EBRT alone ~1%, brachytherapy alone ~3%.
- Combined ↑ regardless of order or isotope
- cryosurgery & HIFU ~2%

SALVAGE therapy after EBRT – prostatectomy, cryo, HIFU, or BT: RUF 3-6%, reported up to 60%

- the most complex – large fibrotic tracts in poor tissue, w/ urethral & rectal strictures, sexual & urinary dysfunction

Also: low rectal resection, salvage rsxn for anal CA, pelvic fracture, penetrating trauma, IBD, pelvic infection

- rarely fistulotomy or stapled hemorrhoidopexy

Anterior rectal biopsy in an irradiated field is often the final precipitating event – biopsy with great care

- 2ndary rectal CA after EBRT at 5-15 yrs

PRESENTATION

Post-prostatectomy: within the first 2-4 weeks

Post-XRT: up to 14 YEARS after the last dose

Passage of urine per rectum on voiding is the MC complaint

- fecaluria, pneumaturia, pelvic or bladder pain, recurrent UTI
- > 80% have preexisting erectile dysfunction

EVALUATION

Defect in the ANTERIOR rectal wall 5-6 cm from the verge
Cystoscopy + colonoscopy – location, size, tissue quality, biopsy anything suspicious

VCUG or retrograde urethrogram. Gastrografen enema

CT / MRI as adjuncts and to exclude an undrained abscess

PSA – exclude recurrent CA

Urodynamics: severe incontinence or voiding dysfunction will NOT improve with repair

- these pts are better served by permanent urinary diversion

CLASSIFICATION

Rivera staging – not universally adopted:

- 1: < 4 cm from verge, non-irradiated
- 2: > 4 cm from verge, non-irradiated
- 3: < 2 cm diameter, prior radiation, any distance
- 4: > 2 cm diameter, prior radiation, any distance
- 5: ischial decubitus fistula

Simple vs. Complex – what most authors use:

- Simple: < 1 cm, non-irradiated, minimal Sx, no sepsis, no prior repair attempt
- Complex: > 1 cm, prior XRT or cryo, urethral stricture, sepsis, or prior failed repair

MANAGEMENT

Multidisciplinary – colorectal + urology + reconstructive/plastics

Neoplasm as the cause → treat the neoplasm FIRST.

Crohn's → optimize medical Rx first

A significant number heal WITHOUT an operation

- fecal diversion alone: spontaneous closure 14-46%
- small RUF: urethral catheter drainage alone

Simple RUF:

- urethral catheter drainage x 2-3 months
- healed → pull the catheter
- unhealed → local flap repair, transanal. Fecal diversion NOT required
- flap fails → fecal diversion, reassess at 2-3 months
- still open → repeat local flap, or transperineal repair w/ gracilis or dartos interposition

Complex RUF:

- stoma ± suprapubic catheter ± abscess drainage
- reassess at 3 months, endoscopically AND radiographically, from BOTH the rectal and urinary side
- healed → close the stoma
- unhealed w/ positive oncologic margin, nonfunctioning bladder, severe urethral stricture, or high fistula → ABDOMINAL operation
- otherwise → transperineal repair with an interposition flap
- poor candidate or refuses → permanent diversion is a legitimate endpoint

NO data that fecal diversion raises the closure rate – it is symptom control and surgeon judgment

- loop ileostomy favored: easy to make & close, and leaves the colon fallow for a later proctectomy

OPERATIVE APPROACHES

Transanal – Endorectal Advancement Flap:

- for the SIMPLE, small, low, non-irradiated RUF
- prerequisite: NO anal or rectal stricture. Fecal diversion not required
- divide the fistula, dissect rectum off urethra, debride the urethral opening

- interpose pararectal fat or biologic mesh over the urethral side, 3-0 absorbable
- flap secured w/ interrupted 3-0 polyglactin. Catheter 4-6 weeks before assessing healing
- TEM platform can be used
- Garofalo (n=12): 67% primary healing, 83% final after 2nd repair

Transperineal – PREFERRED for complex RUF:

- allows interposition of healthy, well-vascularized tissue. Closure ~90% regardless of XRT or ablation
- good exposure for LOW and MID rectal RUF
- prone jackknife, U-shaped perineal incision from lateral perineum onto posterior scrotum
- dissect the rectoprostatic plane ANTERIOR to the sphincters, separate the fistula, continue 3-4 cm cephalad
- augment the urethra w/ buccal mucosa or biologic mesh if the tissue is thin
- urethra closed 3-0 absorbable, imbricate bladder over it if possible
- close the rectal defect HORIZONTALLY – avoids narrowing the lumen
- flap parachuted in on pre-placed sutures, wound closed in layers over a drain

Dartos flap: low, small RUF. Skin removed, rotated up.

Varma: 6/8 healed – both failures were XRT and HIV

Gracilis flap: larger, higher, or irradiated RUF. 3 small longitudinal incisions ~4 cm posterior to adductor, divide the insertion at the medial knee, clip the SFA perforators, PRESERVE the neurovascular bundle within 10 cm of the pubic symphysis, tunnel subcutaneously into the perineum

Posterior – York-Mason & Kraske:

- success ~88%, similar to transperineal – but the data are nearly all NON-irradiated
- York-Mason: posterior sagittal division of the sphincters, levators, and posterior rectal wall. Repair urethra then rectum, close each layer meticulously
 - major complications: rectocutaneous fistula, sphincter compromise
- Kraske: parasacral incision, coccygeal resection, divide the anococcygeal ligament, posterior proctotomy
- USE HAS FALLEN OFF over the last 15 yrs – limited exposure, cannot address urethral stricture or bladder neck, hard to interpose tissue
- Dafnis (n=20): 90% initial. Failure a/w DM, smoking, XRT
- Van der Doelen (n=28): 64% initial, 75% ultimate. Non-irradiated 89% vs. irradiated 50%
 - irradiated w/ gracilis 100% (3/3) vs. 29% (2/7) without

Transabdominal:

- for concomitant complex intrapelvic problems, positive oncologic margins, nonfunctional bladder, strictured urethra, prior failed repair

- do the URINARY procedure first, then primary rectal repair, then interpose omentum (preserve the L gastroepiploic) or rectus abdominis
- rectal defect too large or tissue too poor → proctectomy ± coloanal anastomosis
 - very low fistula → mucosectomy or intersphincteric dissection from below, handsewn anastomosis
- sphincter preservation not indicated → leave the stump, end colostomy, and AVOID the perineal incision
- options: cystectomy + urinary diversion w/ rectal repair, proctectomy w/ coloanal, APR, pelvic exenteration
- a 2nd low pelvic dissection has a MUCH higher failure rate than the first

Others:

- Puborectalis interposition: 1-cm bilateral strips released posteriorly, rotated medially, overlapped over the closed fistula. Solomon 4/4 healed. Muscle bulk is the limitation
- fibrin glue, cauterization, over-the-scope clips – case reports, short follow-up, NOT mainstream

OUTCOMES

No RCTs – rarity means all series. Overall closure after repair 68-100%

Transperineal + muscle interposition:

- systematic review: healing non-irradiated 89% vs. irradiated 90% – essentially the SAME
- but PERMANENT fecal diversion 25% irradiated vs. 4% non-irradiated
- permanent urinary diversion 42% vs. 4%
- Kaufman (n=98): healed after one procedure 98% non-XRT vs. 86% XRT; GI continuity restored 94% vs. 65%
- Hampson (n=21): initial success 95%, 30-day morbidity 19%. Long term: 53% perineal pain, 43% residual gracilis-harvest problems
- Cleveland Clinic (n=36 gracilis): 78% initial, 97% after redo, complication rate 47%
- non-irradiated w/ a prior failed repair: 9/9 healed w/ transperineal + gracilis

Gracilis harvest morbidity is NOT trivial – consent for it
Radiation is the single variable that predicts failure and permanent diversion

BOTTOM LINE

Prostate CA treatment causes it. Radiation makes it complex

Simple, small, non-irradiated → catheter, then transanal flap. No stoma needed

Complex → divert, drain the sepsis, wait 3 months, reassess from BOTH sides

Definitive repair = TRANSPERINEAL with gracilis interposition

Bad bladder, positive margin, high fistula, or a dead rectum → abdominal operation

Warn the irradiated pt: good chance the stoma and the urinary diversion become permanent

15 ANORECTAL DISEASE: RECTOVAGINAL FISTULA

RECTOVAGINAL FISTULA

CLASSIFICATION

Abnormal connection b/n the anal canal or rectum and the vagina

Height – Daniels:

- Low: distal anal canal (dentate line or below) to the vaginal fourchette → perineal approach
 - “anovaginal” and “low rectovaginal” used interchangeably
- High: upper vagina at the level of the cervix → abdominal approach
- Middle: everything in between

Simple vs. Complex – reflects the local tissue:

- Simple: low, < 2.5 cm, obstetric or infectious. Local repair
- Complex: high, larger, or from XRT / CA / IBD / pelvic surgery. Resection, interposition, &/or diversion

ETIOLOGY

Obstetric:

- pressure necrosis of the rectovaginal septum in prolonged labor, laceration of the sphincter complex, or episiotomy
- presents 7-10 days after delivery, MC after a 3rd or 4th degree lac – inadequate repair, breakdown, or infection
- 0.06-0.1% of US vaginal deliveries. Far higher where labor is prolonged
- long called the MC cause – recent series say otherwise
 - Mayo 23%, Cleveland Clinic Florida 24%
 - Norwegian population study: 42/182 enterogenital fistulas obstetric, only 24 (13%) truly RVF
- injury recognized AT delivery → repair immediately, optimal conditions, experienced team

Crohn's:

- RVF in ~1% of women w/ CD. 10-yr cumulative probability 3%
- FALLING in the biologic era – IBD-South Limburg 5-yr rate 5.7% (1991-2005) → 1.7% (2006-2011)
- risk ↑ w/ disease duration and more distal luminal disease. Highest w/ Crohn's proctitis

Cryptoglandular: 2-40% of surgical series

Anastomotic & iatrogenic:

- up to 10% of women after LAR – posterior vaginal wall caught in the staple line
- also IPAA, hysterectomy, stapled hemorrhoidopexy, STARR
- Kosugi: 6/16 (37%) of anastomotic-vaginal fistulas healed with DIVERSION ALONE within 6 months

Radiation: after XRT for cervical and other pelvic

malignancy

- Zelga: onset 5 months to 20 years out, median 20 months

Malignancy: direct extension of rectal, cervical, or vaginal CA

EVALUATION

Two goals: identification, then assessment of the sphincter

Identification:

- dark red rectal mucosa vs. pink vaginal mucosa.
 - Dimple in the anterior midline of the rectum
- Tampon / Methylene Blue Test: tampon in the vagina, methylene blue enema 20-30 ml, prone → inspect the tampon
 - in-clinic version: gauze in the vagina, dilute Betadine per rectum by 60 cc syringe
- Vaginography: contrast per vagina through a Foley w/ the balloon occluding the orifice. 79-100% sensitive
 - more useful for colovaginal & enterovaginal fistula
- MRI = PREFERRED imaging – trajectory, number of tracts, undrained collections. CT inferior for soft-tissue contrast
- Endoluminal US: best for the SPHINCTER, poor view beyond it
- Rectal contrast enema for anastomotic patency.
 - Endoscopy if Crohn's or XRT suspected
- Awake exam inadequate → EUA

Assessment:

- incontinence Sx in 48%, and ~100% of post-obstetric pts
- ALWAYS assess the sphincter – if damaged, address it at the same operation
- quantify: pad use, ability to defer stool & flatus, day & night stool frequency, volume of drainage
- pneumaturia or fecaluria → look for an associated enterovesical or rectourethral fistula
- mark the stoma site pre-op if an ostomy is on the table

TREATMENT

Control infection FIRST – drainage, draining seton, antibiotics. Then decide

Asymptomatic or minimally symptomatic → NO intervention, especially in Crohn's

Multiple attempts are the RULE

- Pinto (n=125, 184 procedures): 57% healed after the first repair, 87% after multiple
- recent series (n=79): 72% ultimately healed, mean 3.6 ± 2.4 procedures (range 1-10)

No single technique is superior – healing 50-85% across series

Preop: smoking cessation, weight loss, DM control, bowel regimen. Mech prep + single dose abx

Post-op: stool softener or mineral oil, peri-bottle, NO tub soaks, no intercourse / tampons / strenuous activity x 6 wks

MEDICAL MANAGEMENT – CROHN'S

Non-cutting seton + infliximab. Remove the seton before the last dose

- ACCENT-2 subset: 16/25 (64%) w/ ≥ 50% closure at induction; at 52 wks 45% infliximab vs. 43% placebo

- duration of closure longer w/ infliximab (45 vs. 25 wks)
 - SHORT-term benefit only
- 2016 systematic review, 137 CD RVF: 63% any response, 38% complete
- “worth a try,” far from a panacea

NEVER operate before the proctitis is treated

Stem cell injection: 60% healing in 10 CD pts – early data

Anti-TNF, steroids, or immunomodulators at the time of surgery do NOT worsen healing (Narang)

PERINEAL APPROACH

PREFERRED for sphincter injury, deficient perineal body, or a gaping introitus

Episioproctotomy:

- convert to a 4th degree laceration – longitudinal incision over the probe to create a cloacal defect
- excise the tract & all granulation tissue, mobilize the rectovaginal septum to healthy pliable tissue
- rectal mucosa: running 3-0 absorbable
- levators & rectovaginal septum approximated – SHALLOW bites, no tension (deep bites → levator spasm & dyspareunia)
- OVERLAP the sphincters w/ mattress sutures. Align the hymenal edges, repair the transverse perineal muscles, skin loosely
- success 64-100%

Transverse Perineal Repair:

- transverse incision through the perineal body, dissect proximal to the tract
- excise the tract edges, close the rectal wall in TWO layers, approximate septum & levators, close the vaginal epithelium

TRANSRECTAL APPROACH

The traditional colorectal preference – the repair sits on the HIGH-PRESSURE side

Endorectal Advancement Flap (ERAF):

- curvilinear incision ~180° just distal to the fistula opening
- flap of mucosa, submucosa & rectal wall off the rectovaginal septum, mobilized PROXIMAL to the fistula – no tension
- base 2-3x the apex. Excise the tract, close the opening, trim the flap, sew to the distal cut edge
- vaginal / perineal side left OPEN to granulate
- success 43-93%. FI after repair 21-40%

Rectal Sleeve Advancement:

- distal rectum diseased, proximal rectum normal – Crohn's, XRT, prior surgery
- mucosectomy from the dentate line cephalad to healthy tissue, full thickness above the anorectal ring
- mobilize 90-100%, bring down without tension, sew to the neodentate line
- the alternative when the only other option is proctocolectomy or permanent diversion

Noble-Mengert-Fish: full-thickness anterior wall mobilized 180°, flap secured to the EAS

VAGINAL APPROACH

Least reported in the colorectal literature – but consider it

- avoids mobilizing a diseased rectum AND avoids sphincter stretch from anal retractors
- FAVORED with active Crohn's proctitis. Endovaginal flap outcomes = endorectal flap
- posterior vaginal flap raised, both orifices closed, levators approximated in the midline, flap advanced over the repair
- flapless variant – core out the tract and freshen the edges: 67% in 15 pts

TISSUE INTERPOSITION

For prior failed repair, Crohn's, and radiation. Adds bulk, obliterates dead space

Martius – labial fat pad & bulbocavernosus:

- longitudinal incision over the labia majora
- preserve the POSTEROLATERAL pedicle off a branch of the internal pudendal
- tunneled and interposed b/n the closed rectal and vaginal repairs
- success 60-100%

Gracilis: greater bulk of vascularized tissue. Tunneled from the proximal thigh into the perineum. Success 53-92%

Bioprosthesis: plugs and biologic mesh. Success only 20-35% – the early enthusiasm has faded

ABDOMINAL APPROACH

For MIDDLE-third rectal or upper vaginal fistulas, and for severely damaged post-surgical or irradiated tissue

Healthy tissue → anterior dissection b/n rectum & vagina, excise the tract, interpose OMENTUM

Most need resection of the diseased rectum w/ a lower anastomosis

- normal distal rectum → immediate anastomosis
- other fistulous connections, or an internal opening near the suture line → Turnbull-Cutait DELAYED coloanal
- circumferential sutures at the neodentate line, prolapse the bowel out and wrap in gauze x 5-7 days, return to OR to amputate and fix
- Bricker-Johnston: sigmoid divided w/ Hartmann's, distal end sewn to the rectum at the level of the fistula, later side-to-side colo-colic

DIVERT for any abdominal repair and for any radiation fistula

Severe radiation injury → permanent colostomy

CHOICE OF TREATMENT

Obstetric → high rate of spontaneous closure at 6-9 months, wait it out

- ~100% have a sphincter defect. Goal is closure AND continence
- sphincteroplasty + episioproctotomy. Martius or advancement flap also good
- deliver by C-SECTION after a repair

Cryptoglandular → EUS to exclude occult sphincter disease, then advancement flap

Crohn's → medical first, seton, then flap. If you flap,

DIVERT

- per-procedure success 44% Crohn's vs. 78% non-Crohn's
- RF for permanent diversion: complex fistula, anal stenosis, prior rectal resection, FI, prior temporary diversion

Radiation → divert $\geq$ 6 months. Low: muscle interposition.

High: abdominal interposition or resection

Iatrogenic → high: redo resection. Low: advancement flap

Persistent → 3 months minimum b/n attempts. The 2nd often works, the 3rd usually doesn't

- go to interposition graft + sphincteroplasty

No RCT on diversion – divert for redo repairs, technically difficult repairs, and suboptimal tissue

16 ANORECTAL DISEASE: PILONIDAL DISEASE AND HIDRADENITIS SUPPURATIVA

PILONIDAL DISEASE

EPIDEMIOLOGY & ETIOLOGY

Chronic suppurative dz of the sacrococcygeal natal cleft – quiescent to frankly purulent

~70,000 cases/yr in the US

Once quoted M:F 3.5:1 – now affects the sexes

EQUALLY, up to 55% female in recent series

- “jeep disease” – WWII drivers, prolonged sitting
- risks: obesity, hirsutism, poor hygiene, sitting/driving, sweating, deep cleft, FamHx

ACQUIRED, not congenital – the embryologic-remnant theory is dead

- warmth + friction + moisture, plus trauma to skin & hair follicles
- → granulomatous foreign-body reaction → sinus, then infection

DIAGNOSIS

Physical exam alone – midline pits in the cleft, loaded with hair & debris

- clear the debris & free hair first, then examine the ENTIRE cleft
- pull hair from the pits with a hemostat

Abscess: erythema, fluctuance, induration – typically just LATERAL to midline

- may sit well away, but always communicates with a midline pit

Pts report “rectal bleeding” – but spontaneous, unrelated to bowel movements

Pits low near the verge get mistaken for a fistula external opening

NO formal classification, no gold standard, no guideline for procedure choice

The treatment MUST NOT be more debilitating than the disease

NONOPERATIVE TREATMENT

Quiescent disease needs NO invasive treatment

Risk Factor Modification:

- hygiene, weight loss, hair removal, avoid prolonged sitting

Hair Removal:

- shaving, waxing, depilatory, or laser
- laser: recurrence 0-28%, lower than non-laser. Needs dark hair / light skin
- NOT depilatory or laser over active infection – shave, weekly in clinic if needed
- post-op hair removal ↓ recurrence even after definitive surgery

Antibiotics:

- NOT routinely indicated, and NEVER sole therapy for an abscess
- reserve for extensive cellulitis + systemic signs, DM, immunosuppression, prosthesis

- 3rd-gen cephalosporin + metronidazole

Phenol:

- office, local anesthesia. Success 62-95%, complications 0-2%
- debride tract, enlarge cavity, instill 1-3 mL crystallized phenol, ointment to shield skin
- 1-4 sessions. Better w/ laser or depilatory. Works for recurrent dz

Fibrin Glue:

- curette hair & debris, then instill. Recurrence 0-17%, success 96%, repeatable
- Cochrane, 4 RCTs / 253 pts: LOW-quality evidence. Utility unclear

SURGICAL TREATMENT

Incision & Drainage:

- MANDATORY for acute abscess
- incision OFF THE MIDLINE, over the point of maximum fluctuance
- unroof the cavity – edges must NOT re-oppose or it heals prematurely
- nearly always clears the acute infection, and is often definitive
- recurrence = failure to clear retained hair, debris, granulation, epithelium

Lay Open (Unroofing):

- probe the tract, incise over the probe, excise overlying skin to keep it SHALLOW
- curette the tract, marsupialize edges with running absorbable
- success up to 97%
- recurrence 8.8% if incised & left open, 20.8% if excised & left open
- infection rate = primary closure, but recurrence significantly LOWER
- cost: bid dressing changes; hypertrophic granulation needs silver nitrate

Excision + Primary Closure:

- excise cavity to sacral fascia, close in layers, skin vertical mattress
- tie-over pressure dressing several days to prevent seroma
- faster healing, less time off work – but infection may force reopening

Cochrane, 26 trials / 2530 pts: primary closure heals faster, SSI no different. Off-midline closure had FEWER SSI and faster healing than midline

No winner open vs. closed – but IF you close, keep the closure OFF THE MIDLINE

FLAP PROCEDURES

Goal: excise the disease, cover with healthy tissue, and FLATTEN the natal cleft

Indications: chronic sinus w/ epithelialized granulation failing hygiene + hair removal, or multiple prior drainages. Elective timing

Karydakis Flap:

- elliptical excision to sacral fascia, superior & inferior corners ~2 cm off midline
- skin + subQ flap raised on the OPPOSITE side, advanced across the bed
- deepest layer MUST catch sacral fascia. Vertical mattress skin, sutures out 10-12 d
- Karydakis, 6545 cases: wound complications 8%, recurrence < 2%
- modern series: recurrence 1.2-2.7%, wound complications 8-19%, morbidity up to 21%
- for RECURRENT disease: 1-yr recurrence 3%, dehiscence 10%

Rhomboid (Limberg) Flap:

- rotational. Excise a diamond to sacral fascia, apices just OFF the midline
- keeps the wound from coming to a point just above the anus – highest-risk site
- include all prior I&D scars in the excision
- from the lateral apex: 5-6 cm horizontal limb, then inferomedial at an acute angle
- undermine for a tension-free closure without making the flap ischemic. Drain optional
- infection 1.5-4%, recurrence 0-4.9%, morbidity 19.7%. LEAST cosmetic of the flaps
- but complications reported as high as 49% – dehiscence 45%, recurrence 13%

Cleft Lift (Bascom):

- prone. "Safety zone" = tissue that touches when the cleft is pulled together
- that is the most LATERAL extent of dissection
- excision crosses the central pits, scimitar-shaped inferiorly
- does NOT require excising ALL the diseased tissue
- RAISE THE FLAP FIRST, toward the side opposite the tissue to be excised
- mastectomy-flap thickness. Preserve subQ toward the anal side
- excise the overlying skin only – leave the subQ. Lance central scar. Drain, subcuticular skin
- Bascom & Bascom: 90% initial success, 100% after re-do. Later series healing 96-97%
- morbidity up to 20%, but least post-op pain and highest satisfaction

Other Flaps:

- V-Y advancement, Z-plasty, rotational S-plasty for large or recurrent defects
- myocutaneous flaps and STSG for severe salvage

MINIMALLY INVASIVE

Endoscopic Ablation (EPSiT / VAAPS):

- saucerize the opening, insert a 4 mm hysteroscope under continuous saline
- adhesiolysis, hair grasped out, ablate w/ 5F bipolar, then iodine. ± phenol
- success 67% at median 52 wks, delayed healing 77%
- vs. Limberg: HIGHER recurrence but FEWER complications

Laser Ablation (diode):

- outpatient, local. Debride & curette the tract, irrigate, then ablate the epithelium
- EVERY PIT must be treated
- success ~85-90%, recurrence as low as 2.9%. Best in LESS severe disease
- immediate return to work in up to 92.8%
- complications: pain, hematoma, abscess, drainage

Trephination:

- probe each pit, core it out with a 2.0-9.0 mm skin trephine (4-5 mm down to a cavity)
- 6.0-9.5 mm for acutely infected areas, left open. Complications < 5%
- healing 89.7% at 4 wks, recurrence 16.2%
- does NOT preclude a later flap

RECURRENCE & WOUND CARE

Recurrence up to 34% at 1 yr and 66% at 5 yrs – no procedure is exempt

Higher after antibiotics alone and after repeated I&D. 45% of recurrences are obese

Nonhealing wounds are 2/2 repeated friction in the cleft with movement

- rule out recurrent sepsis FIRST, then exhaust wound care before re-excising
- silver nitrate to hypertrophic granulation, warm soaks, soap cleansing
- silver gauze, hydrophilic wound cream, methylene blue / gentian violet cream bid

HIDRADENITIS SUPPURATIVA

EPIDEMIOLOGY

Prevalence 0.1% in the US, 98 per 100,000

Women > 2x men. Peak age 30-39. African American prevalence > 3x white

Onset after puberty – only 2% before age 11, 7.7% before age 13

- early onset = stronger genetic susceptibility, more widespread disease

Axilla MC (70%), then perineum & groin. Often more than one site

- smoking: OR 1.9 for developing HS; meta-analysis of 25 studies OR 4.26 in current smokers
- obesity: OR 1.12 for EVERY 1-unit rise in BMI

PATHOGENESIS

NOT an apocrine gland infection – a disease of FOLLICULAR OCCLUSION

- occlusion → rupture → keratin & bacteria into dermis
→ foreign-body immune response

Auto-inflammatory: Th17, IL-17, IL-1β are the targets.

Bacteria/biofilm are secondary

DIAGNOSIS

Delay is the rule – 2.3 yrs to first physician, 7.2 yrs to diagnosis

Requires all three:

- morphology: nodules, abscesses, tunnels, scars
- location: axilla, inframammary, groin, perigenital, perineal
- progression: 2 recurrences in 6 mo, OR persistent lesions ≥ 3 mo

Differential:

- absence of MIDLINE SACRAL PITS distinguishes it from pilonidal
- absence of ANAL CANAL involvement distinguishes it from Crohn's and benign fistula
- no tract to or through the sphincter. Biopsy if uncertain
– chronic dz can go to SCC

HURLEY CLASSIFICATION

I: transient, nonscarring inflammatory lesions

II: recurrent abscesses w/ tunnels & scarring – lesions separated by NORMAL skin

III: coalescent lesions, diffuse tunnels, scarring, inflammation

- refined Hurley: I and II each subdivided A mild / B moderate / C severe. III always severe
- criticism: static, and built for ONE anatomic region while most HS is multifocal

826 pts: Hurley I 45%, II 41%, III 13%

- severity a/w male sex (OR 2.11), duration, BMI, pack-years, axillary (2.24), perianal (1.92)

Other Scores:

- Sartorius: regions counted & weighted, points for inter-lesional distance and Hurley III
- HiSCR: lesion counting. The validated endpoint in the adalimumab trials

Comorbid: obesity & metabolic syndrome MC. Also IBD, spondyloarthritis, skin tumor, acne, pilonidal

MEDICAL THERAPY

Topical:

- clindamycin 1% bid x 3 months – the ONLY topical antibiotic studied, beats placebo
- for Hurley I and mild II. No response at 3 mo → move on

Systemic Antibiotics:

- tetracycline 500 mg bid up to 4 months – widespread Hurley I / mild II
- clindamycin 300 mg bid + rifampin 600 mg qd: FIRST LINE moderate-severe, up to 10 wks
- response 73% at 12 wks → 41% at 1 yr. Relapse a mean of 4.2 mo after stopping

Biologics:

- adalimumab 40 mg SC weekly – FIRST LINE biologic
- RCT of 154 failing abx: ↓ HiSCR & pain, ↑ QOL and work productivity. 3 further RCTs confirm
- infliximab 5 mg/kg – SECOND LINE, only after adalimumab fails. Stop at 12 wks if no response
- anakinra (IL-1 receptor antagonist) and ustekinumab as alternates

Biologics + surgery beat surgery alone – recurrence 19% vs. 38%, new disease 18% vs. 50%, disease-free interval ~1 yr longer. Timing relative to surgery does not matter

Other:

- metformin x 24 wks helps mild-moderate dz. Hormonal therapy: weak evidence
- isotretinoin disappointing – 41% of 207 improved. Only w/ nodulocystic acne
- CO2 laser excision: Hurley II only, HIGHER recurrence than wide excision

SURGERY

Excise the entire PILOSEBACEOUS / hair-bearing region – skin down to fat or fascia

Surgery does NOT alter disease biology. Trade extent of excision against morbidity

Tense painful abscess: drainage only, no medical therapy. Temporizing – recurrence inevitable

Deroofing:

- remove the roof, leave the floor to granulate
- 44 pts / 73 lesions, Hurley I-II: 83% recurrence-free at median 34 mo

STEEP (Skin Tissue-sparing Excision w/ Electrosurgical Peeling):

- successive tangential loop excisions to the epithelialized floor of the tracts

Wide Local Excision:

- small defect → primary closure. Large → secondary intent, or immediate/delayed STSG
- perineal and perianal wounds RARELY need a colostomy
- stage it if circumferential near the anal canal – stricture risk. NPWT before grafting

OUTCOMES

590 pts: drainage HIGHEST recurrence; deroofing ≈ wide excision. Overall recurrence 24%

- risk ↑ w/ younger age (HR 0.8), multiple sites (HR 1.6), drainage (HR 3.5). Extent, gender, severity did NOT matter

Meta-analysis of 22 studies – recurrence: wide excision 13%, local incision 22%, deroofing 27%

- within wide excision: grafting 6%, flaps 8%, primary closure 15%

17 ANORECTAL DISEASE: DERMATOLOGY AND

PRURITUS ANI

PRURITUS ANI

DEFINITIONS & EPIDEMIOLOGY

Pruritus Ani: "itchy anus" – any condition causing itching & irritation of perianal skin

Secondary: an identifiable cause. 75% of cases

Primary (idiopathic): diagnosis of EXCLUSION. 25%, and the hardest to treat

Affects 2-5% of the population – most never seek care
4th to 6th decade. M:F 4:1

Lesion terms: macule (flat) / papule (raised solid) / vesicle (fluid) / bulla (> 1 cm) / pustule (pus) / ulcer (loss of continuity)

- intertrigo: inflammation b/n 2 opposing skin surfaces, e.g. obesity

PATHOPHYSIOLOGY

Unmyelinated C fibers in anoderm & perianal skin → dorsal horn → contralateral spinothalamic → thalamus → cortex

THE VICIOUS CYCLE: C fiber stimulation → scratching & wiping → excoriation → more C fiber stimulation

Histamine is the MOST POTENT pruritogen

- also ACh, substance P, CGRP, opioid peptides, proteases, bradykinin, serotonin, PGE

Two pathways – and this is why antihistamines so often fail:

- histaminergic: Gq → PLCβ3 → DAG/IP3 → PKCε → TRPV1 opens → depolarization
- non-histaminergic: PAR-2 and 5-HT act directly on sensory neurons

FECAL SOILAGE

The dominant mechanism in idiopathic disease

Manometry vs. controls:

- greater rise in rectal pressure during IAS relaxation – 29 vs. 18 mmHg
- prolonged IAS relaxation – 29 vs. 8 seconds
- saline infusion: leaks at 600 mL vs. 1300 mL in controls
- volume at first leak inversely related to symptom severity

Manometry only if workup negative AND no response to intensive therapy

Pelvic floor PT if pelvic floor dysfunction is present

Treat the primary diarrheal disease (UC, Crohn's, IBS) and the pruritus usually resolves

DIET & MEDICATIONS

Offenders: coffee (caffeinated AND decaf), tea, cola, beer, chocolate, tomatoes/ketchup, citrus, milk & dairy, peanuts, grapes, spices

- elimination → remission within 2 weeks, then reintroduce one at a time to find threshold
- mechanism: lower sphincter tone + exaggerated anal reflex → looser, faster stool → seepage

Meds implicated: tetracycline, colchicine, quinidine, peppermint oil, local anesthetics, neomycin

DERMATOLOGIC CAUSES

Contact Dermatitis:

- MOST COMMON perianal dermatologic condition
- macular erythema, hyperkeratosis, radial fissuring
- irritant (soaps, cleansers, alcohol, feces – or one hit of bleach/formalin) vs. allergic
- Rx: remove the agent, keep dry, stop the trauma. Patch testing if refractory

Atopic Dermatitis:

- thickened, leathery, lichenified patches. Onset < age 10, strong FamHx
- look elsewhere: neck, antecubital & popliteal fossae, keratosis pilaris, Morgan-Dennie lines
- Rx: barrier (petrolatum, zinc oxide), anti-inflammatories, antihistamines
- AVOID combined topical anesthetic + steroid – it masks the inciting compound

Psoriasis:

- erythema with SHARPLY defined borders – but perianal scaling is usually ABSENT
- "inverse" / intertriginous psoriasis – paler, non-desquamating plaques in flexural folds
- symptoms are cyclical, mostly at NIGHT
- check groin, genitalia, cleft, axilla, umbilicus
- anti-TNF for IBD can cause PARADOXICAL perianal psoriasis
- Rx: low- to mid-potency topical steroid. Tacrolimus or dapsone if severe

Lichen Simplex Chronicus:

- from chronic diarrhea. Thickened, cracked, excoriated skin
- Rx: control BM frequency – psyllium, loperamide, silver sulfadiazine ± low-dose hydrocortisone

Lichen Sclerosus (Atrophicus):

- almost always WOMEN. Thin, wrinkled, "cigarette-paper" skin w/ discoloration. Involves labia
- a/w SQUAMOUS CELL CA – examine at least ANNUALLY, biopsy anything suspicious
- Rx: clobetasol propionate 0.05% x 6-8 weeks. Tacrolimus as alternative

Seborrheic Dermatitis:

- rare. Malassezia furfur – antidandruff shampoo or any topical antifungal

Neoplastic – biopsy MANDATORY for anything failing first-line therapy:

- Bowen's (SCC in situ): well-demarcated plaque, crusting & scaling
- Paget's: 7th decade, slowly expanding sharply demarcated erythematous eczematous plaque
 - if found → COLONOSCOPY to rule out an underlying carcinoma
- WLE with frozen sections if noninvasive; radical resection if invasive

- SCC of the anal margin and cloacogenic CA can present as refractory pruritus

Also: scleroderma, erythema multiforme, dermatitis herpetiformis, lichen planus, radiation dermatitis, Darier disease

Anorectal causes: prolapsing internal hemorrhoids, fissure, fistula, abscess, proctitis

INFECTIOUS CAUSES

Risks: DM, immunocompromise, obesity, hyperhidrosis, tropical climate

Fungal – the MOST COMMON perianal infections:

- Candida albicans: normal gut saprophyte. Moist, red, macerated skin
 - 20% KOH prep of scrapings → mycelia & spores
 - Rx: nystatin powder/ointment or imidazole several times daily + fix the precipitant
- Dermatophytes: Epidermophyton floccosum, Trichophyton mentagrophytes, T. rubrum
 - their presence is ALWAYS associated with pruritus. Topical or systemic antifungal

Erythrasma:

- Corynebacterium minutissimum. Perianal, axilla, thighs, toe webs
- large pink-red patch that turns brown
- WOOD'S LAMP: CORAL-RED fluorescence – the diagnostic test
- Rx: erythromycin 250 mg q6h, or tetracycline
- also β-hemolytic strep and S. aureus – topical fusidic acid or mupirocin

Parasitic:

- Pinworm (Enterobius vermicularis): worms emerge at NIGHT → nocturnal itch
 - cellophane TAPE TEST for worms & eggs. Rx: mebendazole, or albendazole 100 mg x 1
- Pediculosis pubis: malathion 0.5% lotion. Treat all partners, hot-wash all linens
- Scabies (Sarcoptes scabiei): dark punctate lesions, finger webs & volar wrists
 - KOH prep. Rx: permethrin – cure > 90% – or oral ivermectin. Hot-wash all linens

Viral:

- HSV: painful scattered ulcers & vesicles. Culture the ulcer base or vesicle fluid
 - self-limited in 1-3 weeks. Acute: acyclovir 800 mg tid x 2 d, or valacyclovir 500 mg tid
 - suppression: acyclovir 400 mg bid, or valacyclovir 500 mg daily
- Condyloma (HPV): large lesions cause pruritus – excision and/or fulguration

SYSTEMIC CAUSES

DM, cholestasis, lymphoma, leukemia, renal failure, thyrotoxicosis, hypothyroidism, HIV, pellagra

- deficiencies of vitamin A, vitamin D, iron

- these give GENERALIZED pruritus – treat the systemic disease

Psychiatric: stress, anxiety, depression – association only, never proven. Treat concurrently

DIAGNOSTIC APPROACH

H&P + ANOSCOPY on everyone. H&P alone finds the cause in 75%

Full-body skin exam BEFORE the focused exam – dermatologic disease is rarely limited to one site

Palpate INGUINAL NODES before positioning – palpable nodes = neoplasia or STD

Prone jackknife. Inspect for color change, nodules, fistula opening, tags, ulcers, rash

- Valsalva to exclude mucosal prolapse
- swab for STD BEFORE the digital exam
- biopsy anything suspicious for malignancy – punch or full thickness

CLINICAL STAGING

- 1: *no lesion at the verge, but palpation & anoscopy painful. Other anal lesions excluded*
- 2: *red dry skin, at times weeping, superficial round splits & longitudinal fissures*
- 3: *reddened weeping skin, superficial ulcers & excoriations, pale whitish areas, NO hair*
- 4: *pale, thickened, dry, leathery, scaly, hairless. NO ulcers – the chronic end state*
 - rarely used clinically, but standardizes communication and research

TESTING

Swabs & cultures at the first visit only if infectious risk is high

- viral cultures ON ICE. Tzanck smear if zoster suspected. KOH prep for hyphae
- stool O&P x 3 if diarrhea. Nasal/throat swabs are USELESS – don't send them

Labs only if systemic symptoms, or after first-line failure

- CBC w/ diff, bilirubin/LFTs/alk phos, TSH, BUN/Cr, HIV antibody

Colonoscopy if systemic or refractory symptoms, change in bowel habits, diarrhea, pain, or hematochezia

Biopsy at the SECOND visit if first-line fails and no cause found – send to micro AND path

- histology: epithelial intercellular edema & vesiculation; hyperkeratosis & acanthosis if chronic

TREATMENT – FIRST ENCOUNTER

Set expectations up front: chronic condition, INCREMENTAL improvement, not cure

General principles alone work in up to 90% of idiopathic cases

Hygiene:

- sitz baths WITHOUT additives after defecation. Bidet or handheld shower head

- NO soaps, scrubbing, or aggressive wiping. NO perfumes, dyes, or deodorized baby wipes
- BLOT with damp toilet paper – not a moist wipe. Hair dryer on the lowest setting
- cornstarch or talc to keep the fold dry – but AVOID cornstarch if fungus is suspected
- light cotton underwear, non-perfumed detergent, dry cotton ball at the anus
- dilute white vinegar 1 tbsp in 8 oz water, applied with a cotton ball

Barriers:

- AVOID creams initially – they trap moisture
- if skin changes are significant: zinc oxide or petrolatum after washing
- ± menthol, phenol, camphor. Ostomy powder tid if barrier creams fail
- topical gentamicin / clinda / bacitracin, or clotrimazole / nystatin, if infection suspected

Berwick's solution (crystal violet 1% + brilliant green 1% + 50% ethanol + water) for severe office cases – air dry, seal with tincture of benzoin. Lasts up to a week, long enough to re-epithelialize

Bowel Habit:

- psyllium 2 tbsp every AM with 8-10 glasses of liquid – absorbs water, ↓ seepage
- loperamide or diphenoxylate/atropine if stool stays loose

Acute itch = a marker of FECAL SEEPAGE. Immediate cleansing is the remedy, not a drug

TREATMENT – SECOND ENCOUNTER (3-6 WEEKS)

Repeat the history, review the food diary, and biopsy + scope if not already done

Topical Steroids:

- 1% hydrocortisone bid x 2 weeks, then taper onto a zinc oxide barrier
- RCT vs. placebo: 68% reduction in itch, 75% improved QOL
- MAX 8 weeks – longer causes skin atrophy
- a BRIDGE to break the itch-scratch-itch cycle, NOT curative
- clobetasol 0.05% for chronic or severe disease
- ORAL steroids are NOT indicated

Antihistamines:

- no RCTs in pruritus ani. Sedating agents work by aiding SLEEP, not locally
- diphenhydramine 25-50 mg or hydroxyzine 12.5-25 mg at bedtime
- TOPICAL antihistamines are not potent enough and sensitize the skin – do NOT use

Neuromodulators:

- doxepin 10-25 mg up to 75 mg – anti-H1 AND anti-H2
- amitriptyline 25 mg up to 100 mg – best for neuropathic stinging or burning
- gabapentin and SSRIs for intractable disease

Capsaicin:

- 0.006% cream tid – for pruritus > 3 months
- RCT crossover vs. 1% menthol: 31 of 44 (70%) responded
- brief burn gives inhibitory feedback, suppresses histamine, depletes substance P

Tacrolimus 0.1%:

- 2 RCTs / 53 pts: significant improvement out to 6 weeks
- best in atopic dermatitis. Good substitute when tapering off steroids to avoid atrophy

Anal Tattooing – LAST RESORT:

- 10 mL 1% methylene blue + 5 mL NS + 7.5 mL 0.25% bupivacaine w/ epi (1:100,000) + 7.5 mL 0.5% lidocaine
- intradermal & subQ, prone jackknife, sedation or GA – perianal skin to the entrance of the canal
- methylene blue is directly toxic to the cutaneous nerves. Tattoo fades in 3-4 weeks
- > 80% complete or partial response, but systematic review graded the evidence WEAK
- complications: ↓ perianal sensation, transient fecal incontinence, local inflammation

Local anesthetics (lidocaine 2-5% gel): temporary relief only, NO disease effect – they sensitize the skin

18 ANORECTAL DISEASE: SEXUALLY TRANSMITTED

INFECTIONS OF THE COLON AND RECTUM

GENERAL PRINCIPLES

EPIDEMIOLOGY & IMMUNOLOGY

2.4 million reported cases of chlamydia, gonorrhea & syphilis in 2018, and climbing

50-60% of chlamydia & gonorrhea occur at NON-URETHRAL sites

85% of rectal STIs in MSM are ASYMPTOMATIC

- urogenital-only screening misses 12-30% of chlamydia & gonorrhea in women
- untreated extragenital STI = a reservoir, and a risk factor for acquiring HIV

Symptoms mimic hemorrhoids, fissure, fistula, hidradenitis, or CA. LGV proctitis mimics IBD – IBD failing therapy → think infectious proctitis

Receptive intercourse denudes both the mucous coat and the protective cell layers

HPV ↑ Langerhans cells in the anal mucosa – HIV DROPS them when coinfects. Latex allergy also ↑ risk

- → HPV persists and progresses. HIV+ pts are at greater risk of every secondary organism

SCREENING – CDC

Everyone 13-64: HIV at least once in a lifetime

Sexually active women < 25, or older w/ multiple partners: chlamydia & GC ANNUALLY

MSM: chlamydia, GC & syphilis at least annually, q3-6 months if high-risk behavior

- test urethra, rectum AND pharynx, regardless of condom use. HIV annually

Reportable in all 50 states: gonorrhea, chlamydia, syphilis, chancroid, HIV

PREVENTION

Vaccines: Gardasil-9 from age 9 – originally MSM to 26, FDA now approves to 45. HepA, HepB

PrEP:

- tenofovir disoproxil fumarate 300 mg + emtricitabine 200 mg daily. MUST know HIV status first
- 86% reduction in transmission, 99% if taken consistently
- on-demand: 2 pills before sex, then 1 daily x 2 days after. Screen q3 mo: HIV, renal, STIs

PEP:

- within 72 HOURS, do not delay for testing: tenofovir 300 + emtricitabine 200 daily + raltegravir 400 bid or dolutegravir 50 daily x 28 d

INITIAL EVALUATION & EMPIRIC THERAPY

Anal exam + anoscopy ± endoscopy – erythema, friability, exudate, discharge

SWAB BEFORE THE LUBRICANT – most medical lubricants are bacteriostatic

- two swabs: one viral (HSV), one bacterial (GC, chlamydia)

Serology for SYPHILIS and HIV in every suspected STI

Proctocolitis: stool for Giardia and Entamoeba. Biopsy for CMV if AIDS

Empiric: ceftriaxone 250 mg IM x1 + azithromycin 1 g PO x1 + valacyclovir 1 g bid x 7-10 d ± doxycycline 100 mg bid x 7 d

Universal rules: abstain 7 days after therapy, RETEST AT 3 MONTHS, treat partners from the last 2-3 months

BACTERIAL INFECTIONS

CHLAMYDIA TRACHOMATIS

MOST COMMON reportable STI in the US – ~2 million new cases/yr. 1 in 20 women aged 14-24

Obligate intracellular. ≥ 15 serovars

D-K: urethritis, cervicitis, PID, proctitis. L1-L3: lymphogranuloma venereum

> 50% of chlamydia in MSM is RECTAL, and rectal disease is more often symptomatic

Incubation days to 2-3 wks. Untreated → ectopic pregnancy, infertility, PID

Diagnosis:

- NAAT ~97% sens/spec. Men first-catch urine, women vaginal swab. Extragenital tests FDA-approved 2019

Treatment:

- azithromycin 1 g PO x1, OR doxycycline 100 mg bid x 7 days
- DOXYCYCLINE PREFERRED for RECTAL disease – azithromycin fails in up to 20%
- pregnancy: retest at 3-4 weeks AND at 3 months

LYMPHOGRANULOMA VENEREUM

Serovars L1-L3, L2 driving the surge. Rare in the developed world before 2003 – now MSM, esp. HIV+ Severe proctitis w/ ulcers → abscess, fistula, stricture, chronic pain – MIMICS CROHN'S

Tender femoral & inguinal LAD, often UNILATERAL – the classic BUBO. Uncommon w/ genital infection

Diagnosis:

- positive C. trachomatis NAAT → REFLEX to LGV testing
- biopsy: lymphocytic infiltrate, crypt abscesses, sometimes granulomas – reads as Crohn's

Treatment:

- doxycycline 100 mg bid x 21 DAYS. Aspirate or I&D buboes to prevent fistulization
- HIV+ MSM w/ proctitis + positive rectal chlamydia → treat EMPIRICALLY for LGV

NEISSERIA GONORRHOEAE

Intracellular diplococcus. 2nd most common reportable STI – > 1 million cases/yr

50% of men and up to 95% of women ASYMPTOMATIC – the reservoir

Proctitis: tenesmus, hematochezia, mucopurulent discharge from the crypts. Disseminated: arthritis, dermatitis, endocarditis

- cervicitis in 35-50% of women with rectal GC – DO the vaginal exam

Diagnosis:

- NAAT: sensitivity 100%, specificity > 95%
- gram stain + culture on THAYER-MARTIN for failures – NAAT gives NO susceptibility data

Treatment:

- DUAL therapy: ceftriaxone 250 mg IM x1 + azithromycin 1 g PO x1
- dual even for isolated GC – resistance to fluoroquinolones, cefixime, tetracyclines
- failure = positive test or symptoms 3-5 days after therapy with no interval contact

SYPHILIS

Treponema pallidum. ~35,000 cases in 2018 – up > 70% from 2014. MSM and HIV+ MSM

Primary: ulcer at inoculation 1-21 days. Usually painless – but EXTRAGENITAL ulcers HURT

- resolves in 3-6 weeks with or without treatment, so most are never caught here
- HIV+ more often asymptomatic → they present in the secondary stage

Secondary: weeks to months. Fever, malaise, arthralgia, diffuse LAD, rectal mass

- maculopapular rash involving the PALMS AND SOLES = hematogenous spread
- condyloma lata: flat gray-white lesions packed with spirochetes

Latent 1-30 yrs → Tertiary: cardiac involvement and gummas

Neurosyphilis: ANY stage. Meningitis, cranial nerve palsy, paralysis, dementia

- more prevalent in HIV+, and HIV may accelerate its course

Diagnosis:

- nontreponemal FIRST (VDRL, RPR) – false + w/ HIV, autoimmune disease, pregnancy
- confirm w/ a TREPONEMAL test (FTA-ABS, EIA) – these stay reactive FOR LIFE
- dark field is gold standard on tissue. CSF or slit lamp for any CNS/ocular symptom

Treatment:

- benzathine penicillin G 2.4 million U IM x1 for EARLY disease
- latent and tertiary need longer courses – ID consult
- Jarisch-Herxheimer: fever, headache, myalgia within 24 hrs. Supportive
 - in pregnancy it can precipitate PRETERM LABOR
- retest at 6 AND 12 months. Persistent titers = failure or reinfection. Partners from 3 months

CHANCROID

Haemophilus ducreyi. Only 3 US cases reported in 2018 – but notoriously hard to culture

MULTIPLE painful ulcers w/ RAGGED borders and exudate, hours to days after exposure

Unilateral suppurative inguinal LAD. Facilitates HIV transmission

Diagnosis:

- gram stain: gram-negative rods in chains – “SCHOOL OF FISH.” Sensitivity only 40-60%
- PCR most sensitive, but no FDA-approved test in the US
- clinical criteria: ≥ 1 painful ulcer + no syphilis + regional LAD + HSV-negative exudate

Treatment:

- ceftriaxone 250 mg IM x1 OR azithromycin 1 g PO x1
- better by day 3, objective improvement by day 7, full healing up to 2 wks. Slower if HIV+

DONOVANOSIS (GRANULOMA INGUINALE)

Klebsiella granulomatis. Rare in the US – tropical and developing regions

NON-TENDER, beefy red, highly vascular ulcers that bleed

- anorectal: verrucous lesions, deep fissures, fibrotic ulcers, subQ “PSEUDO BUBOES”

DONOVAN BODIES in large mononuclear cells on Giemsa stain. PCR now available

Rx: azithromycin 1 g PO weekly x 3 wks, or 500 mg daily x 3 wks. Add gentamicin if no response

- ulcers heal from the OUTSIDE IN – treat until fully epithelialized

VIRAL INFECTIONS

HERPES SIMPLEX VIRUS

HSV-2 carried by > 20% of US adults. MC cause of

NONGONOCOCCAL PROCTITIS in MSM

Anogenital HSV-1 is RISING – less childhood exposure leaves more susceptible adults

Symptoms 4-21 d after receptive intercourse. Latency in sensory ganglia. Contagious ~3 wks until epithelialized

Proctitis: friable mucosa, diffuse ulceration limited to the DISTAL 10 CM

- vesicles and pustules are LESS common in proctitis than on skin
- lumbosacral radiculopathy: urinary dysfunction, sacral paresthesia, impotence, pain

Recurrence in the first year after seroconversion up to 90% for HSV-2 – but milder and shorter

Diagnosis:

- PCR is the GOLD STANDARD
- culture detects 90% of active vesicles and allows typing & sensitivities
- TZANCK PREP and DFA are insensitive – NOT recommended. Serology = prior infection only

Treatment – no cure, antivirals only shorten episodes:

- initial: acyclovir 400 mg tid x 7-10 d, valacyclovir 1 g bid, or famciclovir 250 mg tid
- suppression: acyclovir 400 bid, valacyclovir 500 mg or 1 g daily, famciclovir 250 bid
- episodic: acyclovir 800 bid x 5 d or 800 tid x 3 d; valacyclovir 500 bid x 3 d. HIV+ needs higher doses
- severe, systemic, or neurologic → IV acyclovir

- acyclovir resistance 5% in HIV+, < 1% if immunocompetent → foscarnet or cidofovir

HUMAN PAPILLOMA VIRUS – GENITAL WARTS

- > 20 million affected in the US, 5 million new cases/yr
- Condyloma in MSM: 57% if HIV-negative, > 90% if HIV+.
- HIV+ MSM at 2x risk
- > 120 serotypes – 6 & 11 benign condyloma, 16 & 18 dysplasia and invasive CA. 3-mo incubation

Diagnosis:

- clinical. Gray or pink fleshy cauliflower growths
- ANOSCOPY on everyone – intra-anal papules
- RARELY extend proximal to the dentate line
- SUBTYPING IS NOT RECOMMENDED – it does not change management
- biopsy only if immunocompromised, uncertain, failing, worsening, or large/atypical/pigmented

Treatment – destroy all visible disease with minimal collateral injury:

- destruction does NOT eradicate the virus. Recurrence 4-29%; a single treatment clears up to 75%
- ↑ recurrence: intra-anal lesions, extensive disease, combination therapy needed, unvaccinated
- patient-applied: imiquimod 5% qhs 3x/wk, wash off at 6-8 hrs – 87% at 16 wks. WEAKENS LATEX
 - podofilox 0.5% bid x3 d then 1 off, up to 4 cycles, max 0.5 mL/d – 37%. Sinecatechins 15%
- provider-applied: cryotherapy, TCA or BCA 80-90% (apply to white frost), surgical excision
- electrosurgery: fulgurate to gray-white, debride w/ curette. NOT into deep dermis or fat
- > 5 warts: 2 months of topical immunotherapy BEFORE ablation

GIANT CONDYLOMA (BUSCHKE-LÖWENSTEIN)

HPV 6, 11, 16, 18. Papillomatosis, acanthosis, thickened rete ridges, ↑ mitoses

The continuum from condyloma to invasive SCC – transformation in up to 56%. MC if immunosuppressed

- Rx: WIDE LOCAL EXCISION with 1 CM MARGINS – recurrence 65%, higher if HIV+. Secondary intent, or flap/graft for large defects
- APR if the sphincter is involved. Chemoradiation if unresectable or a poor candidate

MOLLUSCUM CONTAGIOSUM

Poxvirus. Incubation 1 wk to 6 mo. Contact AND fomites. Gray-white UMBILICATED papules. Self-limited unless HIV

Rx: phenol or TCA, tangential excision, electrocautery, cryotherapy. Podophyllotoxin, imiquimod

HIV & ANORECTAL DISEASE

- Single-stranded RNA retrovirus – reverse transcriptase, attacks the CD4 receptor
- CD4 < 500: immune reserve compromised, HSV and HPV reactivate

- CD4 < 200: AIDS – opportunistic infection and malignancy

Benign anorectal pathology is the MOST COMMON operative indication in HIV. AIDS CAs: anal SCC, lymphoma, Kaposi's

CD4 / T4 count has NO predictive value for wound healing

- the poor-outcome literature is > 20 yrs old and PREDATES HAART
- a well-compensated HIV+ pt on HAART: treat exactly as HIV-negative

Abscess & Fistula:

- abscess: small incision close to the anal verge. Culture. Antibiotics if immunocompromised
- fistula is cryptoglandular – seton or sphincter-preserving. Fistulotomy only for LOW simple tracts
- assess bowel habits, diarrhea, continence before ANY sphincter-sacrificing procedure

HIV Anal Ulcer vs. Fissure – the critical distinction:

- tender, necrotic, exudative, DEEP, with irregular OVERHANGING borders
- typically OFF midline. When midline, MORE PROXIMAL – closer to the dentate line
- a/w a HYPOTONIC sphincter. Most disabling of any anal dz. NOT affected by antivirals
- do NOT perform internal sphincterotomy unless ulcerating disease is EXCLUDED
- intralesional steroid gives significant relief
- ulcer excision + mucosal advancement flap if medical therapy fails

ECTOPARASITES

PEDICULOSIS PUBIS

Phthirus pubis. Incubation < 1 week. Pruritus, crusting, RUST-COLORED fecal flecks

30% RISK OF COINFECTION – screen for HIV and all other STIs

- Rx: permethrin 1% cream or pyrethrins w/ piperonyl butoxide – wash off at 10 minutes
- alternatives: malathion 0.5% x 8-12 hrs, or ivermectin 250 µg/kg PO repeated at 2 wks
 - lindane LAST – aplastic anemia & seizures. Comb out nits, hot-wash linens. Clear at 1 wk

SCABIES

Sarcoptes scabiei. Survives 36 hrs at room temperature – transmitted by fomites

NOCTURNAL pruritus, symmetric papules & excoriations – finger webs, nipples, genitals

Rx: permethrin 5% cream, wash off at 8-14 hrs, or ivermectin 200 µg/kg PO repeated at 2 wks

Crusted (Norwegian) scabies in HIV / immunocompromise – far more transmissible:

- COMBINATION 25% benzyl benzoate or 5% permethrin PLUS oral ivermectin
- AVOID lindane – toxicity. Re-evaluate at 2 weeks

19 ANORECTAL DISEASE: ANAL INTRAEPITHELIAL

NEOPLASIA

TERMINOLOGY

Dysplastic growth of squamous epithelium at the anal transition zone

LAST project – two-tiered system, REPLACES AIN I-III

- LSIL = AIN 1 or anal condyloma – “infection”
- HSIL = AIN 2 & AIN 3 – “precancer”
- Bowen's, CIS, “anal dysplasia” all mean HSIL – abandon those terms

Cytology graded as for the cervix: insufficient / normal / ASCUS / LSIL / HSIL / SCC

- ASC-H = atypical squamous cells, cannot r/o HSIL

Anal & cervical canals share embryology, cytology, histopathology

- oncogenetic pathway is the same as cervical CA

HPV

> 150 serotypes

- High risk: 16, 18, 31, 33, 35, 45 → E6 & E7 inhibit p53 and Rb
- 6 & 11 → 90% of genital warts, low malignant potential
- 79% of anal SCC attributable to type 16 or 18

79 million currently infected in the US – equal prevalence developed & developing world

90% of immunocompetent pts asymptomatic, clear it w/o treatment w/in 2 yrs

- progression & presence of condyloma track IMMUNE STATUS

Subclinical, grossly apparent (condyloma), or latent – migrates genitalia → anus

Risk: uncircumcised, # of partners, partner's # of partners, early first sexual contact

Vaccine (2006):

- prevalence of types 6, 11, 16, 18 on cytology ↓ > 50% in teens & young women
- Oliver: 71% ↓ in ages 14-19, 61% ↓ in ages 20-24
- genital wart cases falling since 2011
- Giuliano quadrivalent RCT (>4000 boys & men): prevents external genital lesions
- Palefsky MSM subset: efficacy vs. AIN 2/3 for HPV 6/11/16/18 = 50% ITT, 77.5% per-protocol
- give BEFORE onset of sexual activity

EPIDEMIOLOGY

Anal CA ~1.8 per 100,000 – new cases rising ~2% per year over the last decade

AIN in 6-22% of women w/ cervical, vaginal, or vulvar intraepithelial dysplasia

LSIL may regress or progress; HSIL typically does NOT regress

- contrast: ~60% of low-grade CERVICAL lesions regress spontaneously

Rate of conversion to invasive CA – controversial:

- prospective: 9-13% for AIN 3 at 5 yrs, untreated

- meta-analysis much lower – 1 in 600/yr HIV+ MSM, 1 in 4000/yr HIV– MSM
- Scholefield, 35 pts w/ AIN III x 63 mo: 8.5% → invasive SCC, ALL on chronic immunosuppression
- Watson, 72 pts w/ AIN I-III x 60 mo: 15% histologic progression, 11% developed SCC

WHO TO SCREEN

HIV+ – rates substantial REGARDLESS of sexual practice; highest risk is HIV+ MSM

MSM

Solid & liquid organ transplant, medically induced immunosuppression

Women w/ prior cervical, vulvar, or perineal dysplasia
Hx of condyloma or other STD

- immunosuppressed cannot clear HPV – that is the whole mechanism

Screening HIV+ MSM is cost-effective, comparable to other accepted preventive care

- cost per HSIL found ~\$809

SCREENING & DIAGNOSIS

AIN is typically ASYMPTOMATIC – may give pruritus, bleeding, d/c, tenesmus, pain

Sensitivity of DRE and standard anoscopy is LOW

- anoscopy may show a lesion that looks like condyloma but returns AIN on bx

Head-to-toe exam for squamous lesions, all node basins

Look for the other HPV dz – oral, cervical, vulvar, penile.

Refer to GYN/urology

Anal Cytology (anal pap):

- unlubricated moistened Dacron swab, insert 3-4 cm, withdraw slowly in a circular motion
- liquid-based PREFERRED over slide + fixative
- NO bowel prep. MUST precede any instrumentation and any lubricant
- do the DRE and anoscopy AFTER the swab
- sens 69-93%, spec 32-59%
- false negative 23% in HIV– MSM vs. 45% in HIV+ MSM
- ASCUS or higher → HRA
- ASCUS carries an 84% rate of biopsy-proven dz; sens 92% for AIN2+
- 28% of low-grade cytology upgraded to high-grade or invasive on histology
- cytology misses ~1/3 of high-risk lesions – it does NOT correlate reliably w/ histology

Repeat Interval, Normal Cytology:

- HIV+: 6-12 months
- HIV–: 12-24 months
- insufficient or ASCUS: repeat at 6 months

High Resolution Anoscopy (HRA):

- office-based, the anal equivalent of colposcopy. No prep, no analgesia

- lateral position, 3-5% acetic acid x 2-5 min, then magnifying anoscope
- dysplasia = acetowhite, vascular disarray, friability – all ↑ w/ grade
- Lugol's optional – normal epithelium takes it up (wood-like), dysplasia stays yellow
 - many AVOID Lugol's – it obscures grading of the dysplasia
- examine the ENTIRE canal and verge, emphasis on the transition zone
- TARGETED BIOPSY makes the diagnosis – tissue beats cytology

Goldstone, 727 MSM s/p ablation of all HRA-identified lesions:

- 1-yr recurrence 53% HIV+ / 49% HIV–; probability of cancer 1.9% at 3 yrs

Crawshaw, HRA vs. expectant mgmt (424 pts): SAME 5-yr progression rate

- those who progressed were the NON-COMPLIANT – compliance is the variable

TREATMENT

Refer to an expert center. Goal = ↓ progression while preserving function

No high-quality RCTs comparing strategies – individualize

Expectant Management:

- LSIL in a compliant pt – f/u q 4-6 months
- Pineda, 10 yrs of HRA + targeted destruction: 78% clearance, 1.2% progression to SCC

Topicals:

- TCA – provider-applied, no systemic SE. 72-79% resolved or downgraded, recurrence 21%
- 5-FU – response 55-57%, complete response 39%, but 50% recur by 6 mo. 73-85% report SEs
- Imiquimod 5% – most studied. RCT 43% vs. 4% placebo; 61% of responders held at 36 mo
- Cidofovir 1% – complete response 63% at 12 wks, 20% recurred by 24 wks. Compounded only
- ALL recur once stopped – use when ablation isn't possible, or as an adjunct to it

Local Ablation:

- targeted destruction – electrocautery, fulguration, or infrared coagulation (IRC), under HRA
- NO margins needed – dz is confined to the epidermis, do NOT destroy dermis
- electrocautery: initial response 75-80%, complete response 33%, recurrence 25%
- IRC: resolution 82% HIV+ / 90% HIV–; 1-yr recurrence 61% HIV+ / 38% HIV–
- IRC less painful, can be done in clinic
- AVOID large field defects – scarring and stenosis.

These pts need re-treatment for life

Richel RCT, 148 HIV+ MSM – 5-FU vs. imiquimod vs. electrocautery:

- complete response 17% / 24% / 39%

- side effects 27% / 43% / 18%
- 72-wk recurrence 58% / 71% / 68% – recurrence is high no matter what you pick

Wide Local Excision:

- HISTORIC. Mapping bx + WLE NO LONGER recommended, even for diffuse dz
- recurrence 9-63%; repeat excision → anal stenosis and fecal incontinence

ANCHOR: treatment of HSIL vs. active monitoring in HIV+ pts, endpoint anal CA incidence

- stopped early – treating HSIL cut progression to anal cancer by 57%
- TREAT HSIL in the HIV+ patient

SURVEILLANCE

Recurrence is high after EVERY modality – surveillance is mandatory

LSIL / AIN I:

- immunocompetent: annual cytology + anoscopy, OR annual HRA
- high risk, prior CIN/AIN, or immunosuppressed: HRA q 6-12 mo + annual cytology

HSIL / AIN II-III, after ablation:

- immunocompetent: HRA q 3-6 mo + annual cytology
- immunosuppressed: HRA q 3-4 mo + annual cytology
- recurrent or too extensive to ablate → topical + HRA q 3 mo

If HRA normal x 2 consecutive years → annual anal cytology

High-grade cytology w/ NEGATIVE anoscopy and pathology → follow closely, do not dismiss

BOTTOM LINE

LSIL → observe. HSIL → treat

Ablation under HRA = most effective in the immunocompetent

Immunosuppressed → topicals + close observation, and expect to re-treat

WLE and mapping biopsies are dead

Compliance with surveillance matters more than which program you choose

20 MALIGNANT DISEASE: ANAL CANCER

ANATOMY & DEFINITIONS

Anal canal 3-4 cm – proximal limit = apex of puborectalis (anorectal ring), distal = mucocutaneous jxn

- includes 1-2 cm of glandular mucosa + transitional mucosa at the dentate line

Anal transition zone (ATZ) – where the transformed squamous cells arise

Anal canal CA: cannot be seen ENTIRELY w/ gentle traction on the buttocks

Perianal CA: at or distal to the squamous mucocutaneous jxn, fully visible w/ traction, w/in 5 cm of the anus

- “anal margin” is the old term for perianal skin

The more distal the tumor, the more keratinizing and the more it behaves like a perianal tumor

Lymphatic drainage – set by the dentate line:

- above: superior rectal → inferior mesenteric; middle & inferior rectal → internal iliac
- below: inguinal and femoral
- at the dentate line: either or both

EPIDEMIOLOGY & RISK

SCC = 85% of anal cancers

Incidence RISING – 2.7% per year 2001-2015; mortality up 3.1%/yr, esp. > 50 yo

- distant dz at dx has TRIPLED over that span
- 5-fold rise in young black men, doubled in whites of both sexes

2020 US: ~8590 new cases (5900 female), 1350 deaths

Risk: HPV, HIV, transplant or chronic steroid immunosuppression, smoking, multiple partners, anoreceptive intercourse, prior cervical / vulvar / anal intraepithelial neoplasia

HSIL (AIN) is the precursor lesion – see ch 19

New dx of anal dysplasia or anal SCC in a woman → cervical screening if none w/in 1 yr

NO association w/ colonic malignancy – colonoscopy NOT indicated

- EXCEPT anal Paget's → colonoscopy to r/o associated adenoCA

EVALUATION & STAGING

Hx: bleeding, pain, mass, irritation, obstructive sx, continence, STD/HPV, anoreceptive intercourse

- blamed on hemorrhoids or fissure – but onset is MONTHS not years, and it worsens

Exam: size, location, distance from verge, laterality, sphincter and adjacent organ (vagina, prostate) involvement, mobility, ulceration, INGUINAL nodes

HIV testing for every newly diagnosed SCC

Bx to confirm – AVOID a large excisional bx, the wound delays treatment

Imaging:

- CT C/A/P for systemic staging

- PET-CT: alters the XRT field; up- or downstages 5-38%; changes nodal staging in up to 40%
- does NOT replace contrast CT. Negative post-treatment PET → good 2-yr DFS
- pelvic MRI for locally advanced dz

AJCC 8th – SCC staged by SIZE and nodes, NOT depth:

- Tis: HSIL (formerly CIS, Bowen's, AIN II-III)
- T1 < 2 cm; T2 2-5 cm; T3 > 5 cm; T4 any size invading adjacent organ (vagina, urethra, bladder)
- N1a inguinal, mesorectal, or internal iliac; N1b external iliac; N1c external iliac + any N1a
- T1N0 I; T2N0 IIA; T3N0 IIB; T4N0 IIIB; T1-2N1 IIIA; T3-4N1 IIIC; M1 IV

PERIANAL SCC

Keratinized squamous epithelium, verge → 5 cm radially
5x LESS common than canal tumors; 15% of anal region tumors; 1-1.5 per 100,000

Different histogenesis – expresses CK 5/6 and CK 13, rarely CK 7, 18, 19

BETTER prognosis than canal SCC. Slow growing, well differentiated, women, 7th-8th decade

Mets uncommon, recurrence is locoregional

- < 2 cm: nodes rare. 2-5 cm: ~25% node +. large: 67% node +

Misdiagnosed in ~1/3 – median 6 month delay

Looks like skin SCC – distinct border, raised edge, central ulceration

Treatment:

- Stage I: WLE w/ 1 cm margin. RE-EXCISE a positive margin
- larger, poorly differentiated, or threatening the sphincter → XRT ± chemo, incl. inguinal nodes
- add chemo for T3/T4 or involved inguinal nodes
- regimens identical to anal canal SCC

ANAL CANAL SCC

ChemoRT is the initial treatment in NEARLY ALL cases – surgery is for salvage

Pre-1974: WLE or APR. Survival 30-70%, local recurrence 25-70%

Nigro (Wayne State): neoadjuvant 30 Gy + 5-FU + mitomycin C before APR → frequent pCR, which then became DEFINITIVE therapy

CHEMOTHERAPY

5-FU + mitomycin C = STANDARD

- EORTC & UKCCCR ACT I: chemoRT beats XRT alone – 18% higher local control at 5 yrs, median survival 5.4 → 7.6 yrs
- omitting mitomycin: 4-yr DFS 51% vs. 73%, salvage surgery 9% → 22%
- capecitabine is an acceptable substitute for infusional 5-FU – same efficacy, less high-grade toxicity

Cisplatin – does NOT displace mitomycin:

- ACT II, mitomycin vs. cisplatin: NO difference in OS, PFS, locoregional recurrence, or colostomy-free survival
- and consolidation 5-FU/cisplatin after chemoRT added nothing
- RTOG 98-11 (induction 5-FU/cisplatin): the MITOMYCIN arm had superior 5-yr DFS and OS
- induction chemo w/o XRT: no advantage – consider only T4 or prior pelvic XRT
- concurrent cisplatin + mitomycin, and cetuximab: unacceptable toxicity, trials aborted

RADIATION

MINIMUM 45 Gy to the primary

- initial 30.6 Gy to pelvis, anus, perineum, and INGUINAL nodes
- node + or T2-T4 → boost 9-14 Gy

IMRT PREFERRED over 3D conformal (NCCN)

Treatment BREAKS ↓ locoregional control – balance dose against toxicity

- acute: perineal skin, vagina, bladder – pain, bleeding, urinary frequency
- chronic: vaginal stenosis, fecal incontinence, impotence, pelvic & hip fracture
- barrier ointments, vaginal dilators. Incontinence and impotence may never fully resolve

INGUINAL NODES

Node + at presentation ~13%; 20-25% in older pts w/ larger tumors

- → routine inguinal radiation as part of INITIAL therapy

Post-XRT inguinal recurrence: < 2% if N0/N1 at presentation, 11-15% if N3/N4

Do NOT excise suspicious nodes or do a groin dissection BEFORE chemoRT

- wound morbidity delays the therapy that actually works

FNA not needed routinely – sampling error, PET-CT is accurate, and treatment doesn't change

- FNA if the primary is inaccessible, a second malignancy is suspected, or infection clouds the picture

SURGERY

Local Excision – rare, and only for:

- superficially invasive SCC – negative margins, < 3 mm basement membrane invasion, spread < 7 mm (T1NX)
- < 1 cm, well differentiated, completely excised w/ negative margins – no adjuvant needed
- close (< 2 mm) or microscopically + margins → add XRT; 5-yr outcomes then equal
- often UNINTENTIONAL – the small lesion is completely excised at the time of biopsy
- NCDB T1N0: 5-yr OS 85.3% local excision vs. 86.8% chemoRT

Salvage APR:

- 10-30% persist or recur after chemoRT, mostly locoregional. Risk parallels stage

- restage first – exam + biopsy, CT/PET, pelvic MRI in locally advanced
- CONTRAINDICATED: poor performance status w/ advanced age, incurable distant dz, pelvic sidewall / levator / nerve root invasion
- 5-yr survival 50-60% – better than salvage chemo or chemoRT (~30%)
- poor prognosis: + margins, + nodes, distant dz
- en bloc resection of vagina, prostate, distal sacrum is feasible w/ a planned MDT
- irradiated perineum – high infection & dehiscence rate. VRAM for large defects
- flaps do not prevent infection, but shrink the wound and speed healing

Isolated Inguinal Recurrence:

- confirm w/ FNA, restage (CT C/A/P and/or PET-CT)
- superficial inguinal node dissection then systemic therapy – 5-yr DFS 55%

RESPONSE & SURVEILLANCE

Tumor regression continues for up to 6 MONTHS after the end of radiotherapy

First exam 8-12 wks after the last dose – DRE, inspection, anoscopy. Photograph the site

Do NOT biopsy a residual but SHRINKING lesion before 6 months

- ACT II: 72% of those not in complete response at 11 wks WERE complete by 26 wks

Biopsy at once if the mass is ENLARGING, or for a persistent ulcer or mass after 6 mo

DRE + anoscopy + inguinal palpation q 3-6 mo x 4-5 yrs

CT C/A/P annually x 3 yrs if large or clinically node + at presentation

ANAL CANCER & HIV

Higher incidence – a known high-risk group

5-yr OS and complete response rates are the SAME as HIV–

Treat per the standard guidelines – do NOT modify therapy on HIV status alone (NCCN)

Tolerance and toxicity equal, but skin inflammation and pain may be worse

Use IMRT, and examine the anus more often post-treatment

ANAL ADENOCARCINOMA

Arises from an anal gland, or in a chronic fistula tract – think CROHN'S

- hard to distinguish from a distal rectal CA invading the canal

Presents as a canal MASS w/ minimal mucosal component. Mucoïd discharge common

↑ risk of inguinal node mets compared to rectal adenoCA

Staged and treated LIKE RECTAL CANCER – neoadjuvant XRT or chemoRT then APR

- better DFS and local control than upfront surgery
- Worst survival of the anorectal cancers – mean 33 mo, vs. 68 mo rectal adenoCA, vs. 118 mo SCC

OTHER ANAL NEOPLASMS

VERRUCOUS CARCINOMA

Buschke-Löwenstein tumor / giant condyloma. HPV 6 & 11

Large exophytic cauliflower lesion, infiltrates locally – fistulas, necrosis, tissue loss

Local recurrence > 50% after excision. Malignant transformation 40-60%, avg 5 yrs out

Rx: WIDE SURGICAL EXCISION

- chemoRT, laser, PDT, antiretrovirals, intralesional injection – all less successful
- APR if mesorectal nodes suspected or the canal is locally destroyed

MELANOMA

1-2% of all melanomas, ~2 per million/yr; 0.05% of colorectal malignancies

6th decade, female predominance. Bleeding blamed on hemorrhoids → late dx

30% are AMELANOTIC. May be found in a hemorrhoidectomy specimen – the one curable scenario

Workup: pelvic MRI, PET-CT, colonoscopy, full derm and ophtho exam

- SLN mapping controversial, no proven benefit. Mesorectal nodal spread not uncommon

No TNM. Falch: I local, no muscular layer; II into muscular layer; III nodes; IV disseminated

APR gives NO survival benefit over WLE – 5-yr OS 21% in both, regardless of R0

- APR for local control in larger lesions, salvage, or palliation
- death is from DISTANT dz, not local failure

XRT not routine – melanoma is not radiosensitive

Test BRAF and c-KIT – BRAF inhibitors; imatinib for c-KIT (+)

PERIANAL PAGET'S (INTRAEPITHELIAL ADENOCARCINOMA)

Arises from apocrine glands. 6-20% of extramammary

Paget's is perianal

Women, rising from the 5th decade. Scaly, weeping, ulcerative; pruritus, bleeding, pain

Misdiagnosed as a dermatosis – bx only after topical steroids fail

3 types: no associated malignancy / adjacent apocrine tumor / associated GI or GU malignancy

- SEARCH for the other cancer – colorectal adenoCA in 18.5%

Histology: abundant cytoplasm, prominent nucleoli, pleomorphic nuclei, compressed keratinocytes

- stains: cytokeratins, mucins, GCDFP, CEA

MAPPING BIOPSIES of the perianal region – avoids overtreatment and gets clear margins

Rx: WLE to clear margins ± graft or flap ± temporary diversion

- Mohs possible, but exposure and operative time limit it
- APR if margins can't be cleared w/o losing the sphincter, or for an associated anal/rectal CA
- imiquimod, XRT, PDT reported when surgery is refused or would be disfiguring

Recurrence after complete resection: 50% of invasive, 25% of noninvasive

BASAL CELL CARCINOMA

0.1% of all BCC, 0.2% of perianal tumors. NO HPV association

Synchronous lesions elsewhere are common – examine the entire skin surface

Rx: local surgical excision w/ negative margins

GIST

Anorectal = 5% of GISTs; the anal canal is only 2% of those. Men, 6th-7th decade

Well-circumscribed hypoechoic mass in the INTERSPHINCTERIC space on endoanal US

Spread is hematogenous – nodal mets rare

MRI for locoregional staging, CT or PET-CT for distant

Biopsy if large and ulcerated – a neoadjuvant TKI may help

Rx: c-KIT (CD117) + → imatinib to downsize, then resect

- local excision if < 2 cm AND < 5 mitoses / 50 hpf AND the sphincter can be spared
- radical excision + chemo if aggressive – lower local recurrence, but no survival difference
- recurrence can appear > 10 yrs out – survey long term

21 MALIGNANT DISEASE: PRESACRAL TUMORS

ANATOMY

Presacral (retrorectal) space – a potential space

- anterior: mesorectum. posterior: anterior table of sacrum
- inferior: levators. lateral: lumbosacral plexus, ureters, iliac vessels

Contents: connective tissue, nerves, fat, vessels +

TOTIPOTENTIAL cells of all 3 germ layers

- that is why the differential is so wide

Sacral Nerve Roots (Todd) – what you can take:

- bilateral S2-S5 out → COMPLETE loss of bowel and bladder function
- bilateral S3-S5 out → 40% normal bowel, 25% normal bladder
- bilateral S4-S5 out → 100% normal bowel, 69% normal bladder
- UNILATERAL S1-S5 out → 87% normal bowel, 89% normal bladder
- S1 root or sciatic nerve taken → FOOT DROP
- both S3 roots sacrificed → incontinent, plan a permanent colostomy

Pelvic Stability:

- stability lost if more than HALF the S1 body is resected
- preop XRT ↑ risk of stress fracture destabilizing the pelvic ring
- if at risk → sacropelvic reconstruction: metallic fixation, bone graft, 3D-printed titanium

EPIDEMIOLOGY & CLASSIFICATION

Incidence as low as 1 in 40,000-60,000 hospital admissions

2/3 are CONGENITAL, and 2/3 of those are developmental cysts

- next most common group = neurogenic

45-50% are malignant or harbor malignant change

Incidental discovery is RISING – cross-sectional imaging

Uhlig & Johnson (Mayo modification): congenital / neurogenic / osseous / miscellaneous, each split benign vs. malignant

THE LESIONS

Epidermoid & Dermoid Cysts:

- from a defect in closure of the ectodermal layer. Female, thin outer layer, benign
- keratinized stratified squamous epithelium
- DERMOID has skin appendages (sweat glands, hair follicles, sebaceous); epidermoid does NOT
- may communicate w/ the skin → the postanal DIMPLE
- misdiagnosed as perirectal abscess; recurrent infection → SCC

Tailgut Cysts (cystic hamartoma, mucus-secreting):

- remnants of the regressing postanal primitive hindgut. Female

- COLUMNAR epithelium, multiloculated, well defined, homogenous
- do NOT communicate w/ the rectal lumen
- glandular or transitional epithelium is what separates them from epidermoid/dermoid
- malignant transformation reported

Enterogenous (Rectal Duplication) Cysts:

- from the developing hindgut – ENDODERM, so squamous, cuboidal, columnar, or transitional lining
- multilobular: one dominant cyst w/ small satellites
- 3 criteria: attached to the alimentary tract, lined by GI-type mucous membrane, has a SMOOTH MUSCLE coat
- the muscular wall w/ myenteric plexus is what distinguishes them
- generally benign

Teratomas:

- all 3 germ layers; solid and cystic components. Pediatric, female
- mature vs. immature. Recognizable elements (hair, bone, teeth) → more likely benign
- CYSTIC components benign, SOLID components degenerate
- a/w vertebral, urinary tract, and anorectal anomalies
- malignancy tracks AGE: < 2 mo, 7% girls / 10% boys; > 2 mo, up to 48% / 67%
- incomplete or intralesional resection ↑ malignant degeneration
- transform to SCC (ectoderm), rhabdomyosarcoma (mesenchyme), or anaplastic

Sacrococcygeal Chordoma:

- MOST COMMON malignancy of the presacral space. Men, rarely < 30 yo
- from vestigial NOTOCHORD – spheno-occipital base of skull and sacrococcygeal region
- central extracellular mucin; areas of hemorrhage; invades and destroys bone
- pain in low back / pelvis / buttock, worse SITTING, better standing
- local recurrence 43%, distant 22%. 5-yr survival 67%, 10-yr 40%
- WIDE NEGATIVE MARGIN is the only predictor that matters

Anterior Sacral Meningocele:

- thecal sac protrudes through a sacral defect, contains CSF; may accompany a cyst or lipoma
- "SCIMITAR SIGN" – unilateral crescent-shaped defect / hemisacral agenesis
- headache w/ postural change and Valsalva (straining, coughing), back & pelvic pain, dyspareunia
- a/w spina bifida, tethered cord, vaginal duplication, urinary and anorectal malformation
- NEVER biopsy – meningitis and death
- Rx: obliterate the communication w/ the subarachnoid space, detether the cord, resect the tumor

Neurogenic Tumors:

- 10-15% of presacral masses. Younger pts, median age 38. > 90% BENIGN
- schwannoma (neurilemoma) and ependymoma most common
- neuropathy, low back and pelvic pain
- HIGH local recurrence, benign and malignant alike; survival poor if malignant
- nerve-sparing, function-preserving resection is feasible

Osseous Tumors:

- < 10% of presacral tumors. Male. HALF are malignant at diagnosis
- from bone, cartilage, fibrous tissue, marrow. Grow large, destroy locally
- LUNG is the most common site of mets
- giant cell tumor: benign but can metastasize to lung and turn fulminant

Currarino Syndrome:

- TRIAD: sacral malformation (agenesis or sickle) + hindgut anomaly + presacral tumor
- autosomal dominant, HLXB9 mutation
- also tethered cord, thickened filum, syrinx, GU and anorectal anomalies
- DERMOID most common mass; teratoma in 25-40%. May have more than one lesion
- malignancy is rare
- an abnormal-looking SACRUM on imaging is the tip-off

PRESENTATION & WORKUP

Often incidental on pelvic/rectal exam or imaging

Pain: vague, long-standing, pelvis / perineum / low back – worse SITTING, better standing/walking

- pain is more often MALIGNANT and is an ominous sign
- vague enough that pts get sent to psychiatry

Constipation, urinary & fecal incontinence, sexual dysfunction = sacral nerve involvement

Chronic perineal or sacrococcygeal drainage misread as fistula or pilonidal dz

Singer: 7 pts averaged 4.1 prior operations before the diagnosis was made

- REPEATED failed operations for fistula or pilonidal dz → think presacral cyst

Exam:

- DRE: extrarectal mass displacing the rectum ANTERIORLY – nearly all cases
- assess fixation to the rectal wall, and relation to prostate, vagina, coccyx/sacrum
- look for the POSTANAL DIMPLE
- proctosigmoidoscopy: normal mucosa w/ extrinsic mass effect is typical
 - inflamed or abnormal mucosa = infection or erosion into the rectum
- neurologic exam of sacral nerve function

Imaging:

- MRI = GOLD STANDARD – cord involvement, foraminal encroachment, dural compression, bone, sidewall, vessels
 - accuracy up to 100%; sens 50-88%, spec 92-97% for benign vs. malignant
 - MALIGNANT: low T1, high T2, gadolinium enhancement, irregular/infiltrative margins, heterogeneous or solid
 - gadolinium MRI detects meningocele – no myelogram needed
- CT for bone and to define the anatomic extent; CT/MR angio for vascular involvement
- ERUS at the time of flex sig – rectal wall and sphincter invasion
- plain films: osseous destruction, calcification, scimitar sign
- chronic draining sinus → MRI on a FISTULA protocol

PREOPERATIVE BIOPSY

Biopsy only if the result will CHANGE management

- YES for solid and heterogeneously cystic lesions
- NOT for purely cystic – infection, and recurrence after resection
- NEVER a meningocele – meningitis and death
- e.g. neurofibroma vs. neurofibrosarcoma – completely different approach and margins

Route: TRANSPERINEAL or PARASACRAL only, and w/in the planned field of resection

- transperitoneal, TRANSRECTAL, and TRANSVAGINAL are FORBIDDEN
- if the tract crossed rectum or vagina → that organ must come out en bloc
- spot-tattoo the skin entry site
- classic teaching = excise the tract en bloc; Messick left 20/24 tracts in situ w/ no tract recurrence

Neoadjuvant therapy needs the tissue diagnosis

- desmoid: XRT shrinks it and makes the resection easier
- Ewing's, osteogenic sarcoma, neurofibrosarcoma: preop chemo/XRT improves outcome
- aggressive angiomyxoma: hormonal therapy shrinks it
- chordoma: immunotherapy promising
- preop XRT field is SMALLER than postop, w/ less morbidity, and treats micromets

MANAGEMENT

Historically resect everything – now selective

Indications: known malignancy, malignant potential, symptoms, or documented growth

- growth also makes future resection riskier

OBSERVE: small (< 5 cm), asymptomatic, no suspicious radiologic features

- MRI q 5 yrs x 10 yrs for cystic lesions; same for benign solid (schwannoma)

- lengthen the interval if stable; ANY symptom change → re-image

Principles: FUNCTION-SPARING for benign, EN BLOC oncologic for malignant

- for malignant tumors, nothing attached to the specimen is separated from it – ureter, bone, vessel, nerve all come en bloc

Multidisciplinary team: colorectal, ortho-onc, spine, neurosurgery, urology, vascular, plastics, med-onc, rad-onc, MSK radiology, anesthesia

Preop:

- optimize anemia, protein-calorie malnutrition, debility. Social work for rehab planning
- selective COIL EMBOLIZATION of feeders for large vascular tumors (hemangiopericytoma)
- alert the BLOOD BANK. Bilateral ureteral stents for large tumors

SURGICAL APPROACH

Level of the tumor relative to S3-S4 decides the approach

- entirely BELOW S3-S4 → posterior alone
- entirely ABOVE S3-S4 → abdominal (anterior) alone
- crossing S3-S4 → COMBINED

Posterior (Kraske):

- prone jackknife, buttocks taped apart
- incision to distal sacrum/coccyx, through the anococcygeal ligament – AVOID the sphincter
- take the decussating levator fibers off the coccyx tip to enter the presacral space
- a pseudocapsule usually guides the plane off the rectum
- double-glove, index finger in the rectum to push the lesion out toward you
- lower sacrum and/or coccyx may come en bloc
- if rectal wall is involved – excise it, close in layers, then air-leak test
- COCCYGECTOMY not routine – Messick 14% vs. 20% recurrence, no difference
 - DO take it for sacrococcygeal teratoma, which is uniformly adherent

Anterior / Combined:

- supine, synchronous (modified lithotomy), or modified “sloppy” lateral
- mobilize sigmoid, enter presacral space, preserve ureters and superior hypogastric nerves
- dissect mesorectum off presacral fascia to the top of the tumor
- bulky tumor precluding a safe plane → take the rectum en bloc
- both S2 or S3 roots involved → rectum en bloc + PERMANENT COLOSTOMY (they will be incontinent)
- ligate the MIDDLE SACRAL artery ± internal iliac branches to cut blood loss
 - PRESERVE the anterior division of the internal iliac and superior gluteal – perineal/gluteal necrosis

- for high sacrectomy: close abdomen, mature the colostomy, FLIP PRONE
- silastic mesh or plastic sheeting anterior to the sacrum protects vessels/ureters during osteotomy
 - also protects the flap staged in the pelvis for later perineal reconstruction
- ortho: gluteus dissection, transect sacrotuberous & sacrospinous ligaments, divide piriformis to expose sciatic n.
- osteotomy, preserving uninvolved roots. At S2-S3 or higher, CLOSE THE THECAL SAC w/ absorbable suture
- flaps: VRAM, TRAM, omental pedicle, gracilis, gluteus (local or V-Y)

Minimally Invasive:

- lap and robotic are safe and feasible in SELECTED pts
- NOT for very large tumors or malignancy involving sidewall, sacrum, or multiple viscera
- Mullaney systematic review, 82 pts: 95% benign, conversion 5.5%, 30-d morbidity 15.7%, rectal injury 1.2%
 - vs. open historical controls: same OR time, LOS 4 vs. 9 days, comparable complications
 - no recurrences at median 28 mo

OUTCOMES

Benign recurrence 0-35% – HIGHEST for neurogenic tumors

Malignant recurrence 0-48%

R0 with WIDE margins is uniformly associated w/ lower local recurrence

Fuchs, 52 sacrococcygeal chordomas: EVERY pt w/ a wide margin survived. Lung mets in 31%

Kerekes, 1235 sacral chordomas: wide margin ↓ local recurrence; wide margin, female sex, and age ≥ 65 ↓ distant recurrence

Pan, SEER spinal chordoma: age ≥ 60, distant mets, and non-surgical therapy all ↓ survival. Site (vertebral vs. sacral) did NOT matter

22 MALIGNANT DISEASE: SPORADIC AND INHERITED

COLORECTAL CANCER

SPORADIC COLORECTAL CANCER

EPIDEMIOLOGY

~80% of all CRC is sporadic

3rd most common CA in the US and worldwide; 47% of cases in women

- 58% are > 65, but 39% of women and 45% of men are dx'd < 65
- mean age at dx: colon 68 M / 72 F; rectum 63 both
- right colon 41%, rectum 28% – right-sided in older pts, distal in younger
- highest incidence Alaskan Natives (91/100,000) & African Americans (49), lowest Asian Americans (32)

RISK FACTORS

Vieira meta-analysis, 111 studies:

- ↑ 12% per 100 g/d red & processed meat
- ↑ 7% per 10 g/d alcohol
- ↓ 17% per 90 g/d whole grains, ↓ 13% per 400 g/d dairy

Activity at guideline levels ↓ RR ~19%. Obesity & sedentary lifestyle ↑ risk

Gut microbiome of CRC pts resembles that of a high red meat diet

Family Hx: RR 2.25 w/ one 1st degree relative, 4.25 w/ > 1 IBD > 10 yrs, ureterosigmoidostomy, acromegaly, prior XRT, heavy smoking

MOLECULAR PATHWAYS

Fearon & Vogelstein 1990 – stepwise adenoma → carcinoma

Driver mutations: APC (72%), TP53, KRAS, PIK3CA

- APC – EARLY driver, tumor suppressor 5q21; loss → β-catenin to nucleus → LEF/TCF
- TP53 – LATE driver, 17p. Loss → ↑ proliferation, ↓ repair, ↓ apoptosis
- RAS mutated in ~50% (KRAS, NRAS) → NO benefit from anti-EGFR
- BRAF ~10% → worse survival in stage IV; 95% resistant to BRAF inhibition
- EMT (ZEB1/ZEB2, TGF-β, SNAIL, vimentin) is the metastasis pathway

CIN – chromosomal instability: ~85% of CRC. Loss 8p, 17p, 18q; gains 8q, 13, 20q

Hypermuted: ~15%, 1000s of mutations, few copy number changes – MMR defect

- non-hypermuted tumors carry only 100-200 mutations

CIMP – CpG island methylator phenotype: promoter hypermethylation silences genes

- MLH1 promoter methylation = mechanism of MOST sporadic MSI-H
- arises in sessile serrated lesions; BRAF mutation common

CONSENSUS MOLECULAR SUBTYPES

- CMS1 MSI immune – 14%, hypermutated, right-sided

- CMS2 canonical – 37%, WNT & MYC, CNV high, left-sided – BEST outcome
- CMS3 metabolic – 13%, KRAS, CNV & CIMP low
- CMS4 mesenchymal – 23%, EMT, TGF-β, CNV high – WORST outcome

RIGHT VS. LEFT

- right: hypermutated, older pts, CMS1 & 3 – better early prognosis, WORSE once metastatic (BRAF)
- left: younger pts, CMS2 & 4
- KRAS wild-type: cetuximab benefits LEFT-sided only, worse survival if right-sided

YOUNG-ONSET CRC (< 50)

Rising incidence, cause unknown. Predominantly LEFT sided, esp. rectal

- symptomatic at presentation; the younger, the more advanced the stage
- heritable CRC found in 16-20% – ALL should undergo genetic testing
- 75% of those have NO 1st degree relative w/ CRC
- sporadic YO-CRC: CNV high and microsatellite STABLE
- more TP53 & CTNNB1, fewer APC, KRAS, BRAF alterations
- more often need urgent intervention for obstruction; Rx otherwise unchanged

LYNCH SYNDROME (HNPCC)

Most common inherited CRC syndrome – ~3% of all CRC. Autosomal dominant, offspring 50%

HNPCC = family history criteria met. LS = germline MMR mutation confirmed

“Non-polyposis” is a misnomer – same adenoma incidence as sporadic, but progression is FASTER, some in as little as 1 yr

GENETICS

MLH1, MSH2, MSH6, PMS2; also 3' EPCAM deletion → MSH2 promoter methylation

Germline defect + somatic 2nd hit → complete MMR loss

MSI-H = > 30% instability at common loci. ~15% of ALL CRC is MSI-H

- ~70% of MMR deficiency is SPORADIC MLH1 hypermethylation, not Lynch
- BRAF V600E in 69% of hypermethylated tumors
- MLH1 loss + BRAF mutation → unlikely Lynch. BRAF normal → germline testing
- MLH1 & PMS2 are a heterodimer – loss of BOTH = methylation or MLH1 germline
- isolated loss of MSH2, MSH6, or PMS2 → germline mutation

IHC and MSI have equivalent sens/spec; IHC is cheaper and has largely supplanted MSI

- neither diagnoses LS – both test the TUMOR phenotype. Germline testing confirms

UNIVERSAL MMR testing of every resected CRC (NCCN & AGA)

- biopsy rectal CA BEFORE neoadjuvant therapy – XRT/chemo confound the analysis
- PREMM5 & MMRpro predict risk in unaffected relatives

PATHOLOGY

dMMR presents earlier – 20% of stage II, 11% of stage III, only 3.5% of stage IV

Right-sided, poorly differentiated, mucinous or signet ring, tumor-infiltrating lymphocytes

SURVEILLANCE

COY q1-2 yrs. MLH1 or MSH2: start 20-25. MSH6: 30. PMS2: no later than 35

- meta-analysis: ↓ CRC incidence (OR 0.23), ↓ CRC mortality (OR 0.06); + 7 yrs life expectancy
- MLH1/MSH2 5-yr CRC risk: 1 in 71 M (1 in 102 F) in the 20s → 1 in 7 M (1 in 12 F) by 50

Women: annual bimanual + endometrial sampling + TVUS from age 30-35

- endometrial is the 2nd most common CA in LS; MSH6 lifetime risk > 40%
- TAH/BSO after childbearing – 100% risk reduction. Do it WITH the colectomy

EGD at 30-35, repeat q2-3 yrs. Annual urinalysis for urothelial CA

All other extracolonic lifetime risks < 3% regardless of gene

SURGICAL TREATMENT

TOTAL ABDOMINAL COLECTOMY + IRA – US MSTF and ASCRS guidelines

- metachronous CRC > 60% at 30 yrs after segmental resection
- Kalady et al: advanced adenoma or CRC in 47% after segmental vs. 19% after total
- meta-analysis: metachronous 23.5% segmental vs. 6.8% total (OR 3.7) – NO survival difference
- TAC ↑ stool frequency and worsens social function – counsel pre-op
- subtotal + ileosigmoid anastomosis is the compromise – surveillance by flex sig

Rectal CA in LS: proctectomy w/ restorative anastomosis is reasonable

- 2nd colorectal malignancy 15-27% in the first decade even w/ surveillance

No benefit to prophylactic colectomy in incurable stage IV
Annual COY of the remaining bowel after any resection

MEDICAL TREATMENT

Stage III dMMR: FOLFOX, same as sporadic

Single-agent 5-FU less effective in LS – NO benefit in stage II dMMR even w/ high-risk features

Better stage-matched survival and fewer recurrences than sporadic

Checkpoint inhibitors (pembrolizumab, nivolumab, ipilimumab) FDA-approved for MSI-H metastatic CRC

- most dMMR tumors respond, most pMMR do NOT

CAPP2 RCT: ASA 600 mg/d → 50% ↓ in CA incidence.

AGA recommends, ACG conditional

VARIANTS

Turcot: CRC + CNS tumors. Type 1 (MMR) – glioblastoma.

Type 2 (APC) – medulloblastoma

Muir-Torre: sebaceous adenomas & carcinomas + visceral tumors. ~1/3 are MUTYH, not MMR

Familial CRC X: meets Bethesda but MSS. Older, distal, no extracolonic risk, CIN-high

POLE / POLD1 (PPAP): AD, limited polyposis, ↑ CRC.

POLD1 also endometrial & breast. No screening guideline

POLYPOSIS SYNDROMES

FAMILIAL ADENOMATOUS POLYPOSIS

~1% of CRC. Prevalence 2-3 per 100,000, M = F

> 100 synchronous adenomas. Near 100% CRC by age 40

- 25% are de novo – these typically present WITH invasive CA

AD w/ near 100% penetrance. APC, 5q21, > 800 mutations described

- mutation cluster region = 5' end of exon 15
- > 1000 polyps: codons 1250-1464. CHRPE: 311-1444. Desmoid: after 1444

Extracolonic:

- duodenal adenomas in 90%, but lifetime duodenal CA only 5-10% – presents 5th decade
- papillary thyroid CA up to 12%, female predominance
- hepatoblastoma, bilio-pancreatic malignancy
- CHRPE in ~80% – benign, no visual effect, but prompts genetic evaluation
- desmoids in ~15%; half intra-abdominal, typically w/in 5 yrs of an inciting event
- risk: FHx of desmoid, female, prior abdominal surgery, 3' APC (codons 1399, 1444)
- behavior unpredictable – grow, stall, or regress. OBSERVE if asymptomatic
- sulindac ± tamoxifen; adriamycin/dacarbazine in extremis
- 5-yr survival 53% in advanced desmoids requiring TPN & narcotics

Screening:

- polyps at mean age 16 – start endoscopy at PUBERTY, q1-2 yrs
- flex sig until polyps appear, then full COY. Colectomy after puberty
- EGD at 25-30, interval by Spigelman stage
- AFP + liver US in children to age 7 if family Hx of hepatoblastoma
- no consensus screening for desmoids

Surgery:

- most → TPC + IPAA. ↓ fecundity in women, risk to erectile/ejaculatory fxn, ↓ QOL
- TAC + IRA if < 20 rectal polyps or AFAP – preserves pelvic nerves & reproductive anatomy
- counsel that proctectomy is often eventually needed

- “severe” genotype: 61-74% require proctectomy w/in 20 yrs
- registry data: IPAA has better long-term survival, 2/2 metachronous rectal CA after IRA
- CLEAR the rectum of polyps and review the histology BEFORE offering a rectal-sparing op
- mucosectomy: meta-analysis of 4100 pts – more nocturnal seepage, trend toward less dysplasia
- TPC + end ileostomy eliminates CRC risk entirely
- lifelong f/u: annual proctoscopy after IRA; annual pouch scope + DRE after IPAA

Attenuated FAP: 12-100 polyps, CA at 50-70. Proximal predominance w/ rectal sparing

- APC mutation at the far 5’ or far 3’ end
- COY from late teens to early 20s, q1-2 yrs. Upper GI risk SAME as classic FAP
- may be managed by polypectomy alone; if colectomy needed, IRA usually adequate

Gardner: antiquated term. FAP + osteomas of jaw/skull, supernumerary teeth, epidermoid cysts

MUTYH-ASSOCIATED POLYPOSIS

AUTOSOMAL RECESSIVE. Base excision repair defect
→ G-to-T transversions in APC and KRAS

Attenuated phenotype, classically 20-99 polyps, mostly tubular adenomas

- CRC in the 40s-50s. Proximal, mucinous, TILs. Survival better than sporadic
- biallelic: 50x risk, 80% CRC incidence by age 70. Monoallelic: 3x
- carrier rate 1 in 45 → offspring risk only 1-2%, but SIBLINGS 25%
- COY q1-2 yrs + periodic EGD – duodenal adenomas in ~1/3, later & less than FAP
- invasive CA → TAC + IRA w/ annual rectal surveillance. Rectal CA → consider TPC
- suspect it in polyposis with NO APC mutation, or apparent recessive inheritance

SERRATED POLYPOSIS SYNDROME

Most common polyposis syndrome – up to 1:111 in screening cohorts

No germline mutation defined; RNF43 in some families. BRAF mutations common

WHO 2019 – cumulative LIFETIME polyp count, either:

- ≥ 5 serrated polyps proximal to the rectum, 2 of them > 10 mm, or
- > 20 serrated polyps of any size, ≥ 5 of them proximal to the rectum

Lifetime advanced neoplasia risk up to 50%

- clear all polyps, surveillance q1-3 yrs by burden
- CA → segmental or total colectomy after discussion of bowel function
- STRONGLY a/w smoking – push cessation
- overlaps MAP – test for MUTYH if adenomas coexist

HAMARTOMATOUS SYNDROMES

All autosomal dominant. The hamartomas themselves are non-neoplastic

Juvenile Polyposis: SMAD4 or BMPR1A (TGF-β pathway). Colon, stomach, SB, duodenum

- polyps in the 1st decade, mean age at dx 18.5, present w/ melena or hematochezia
- lifetime CRC ~40%, may approach 68% by 60. Gastric CA approaches 30%
- COY at age 12 or earlier if symptomatic. Clear all polyps. Regular EGD
- HGD, invasive CA, or unmanageable burden → TAC + IRA or TPC
 - ~half of IRA pts require completion proctectomy
- SMAD4 → hereditary hemorrhagic telangiectasia. Get a vascular assessment

Peutz-Jeghers: STK11 (LKB1), mutated in 94%; 25% de novo

- polyps mostly SMALL BOWEL – fewer but larger, obstruct or bleed
- mucocutaneous pigmentation at the vermillion border of the lips
- lifetime CA: breast up to 50%, colorectal 39%, pancreas 36%, gastric 29%, ovary 21%, lung 15%, small bowel 13%, cervix/uterus 10%, testis 9%
- upper & lower endoscopy from age 8, q3 yrs. Capsule or CT enterography for the SB
- SBO from intermittent intussusception in ~50%
- at laparotomy: on-table enteroscopy through the open ends of the resected segment – clear the whole bowel
- progression to malignancy → TAC + IRA

Cowden (PTEN hamartoma tumor syndrome): PTEN.

Bannayan-Riley-Ruvalcaba is a variant

- colonic polyps in 95% – hamartomas plus adenomas, ganglioneuromas, lipomas, leiomyomas
- lifetime CRC 9-16%, often before age 50
- COY at age 15, then q2 yrs

SCREENING THE AVERAGE-RISK PATIENT

START AT AGE 45 – ACS lowered it from 50; NCCN still says 50

COY q10 yrs if no adenomas – interval set by adenoma → carcinoma dwell time

Do not screen routinely past age 75

When to start otherwise:

- 1st degree relative w/ CRC or advanced adenoma: age 40, or 10 yrs before the relative's dx, then q5 yrs
- 2nd degree relative only, or a relative w/ polyps after 60 → treat as AVERAGE risk
- personal Hx of adenoma or CRC: COY at 1 yr, then q3-5 yrs
- IBD: q1-2 yrs w/ biopsies – from 8 yrs after dx if pancolitis, 12-15 yrs if left-sided
- Lynch, FAP, AFAP, MAP, JPS, PJS, Cowden, SPS: as above

23 MALIGNANT DISEASE: MANAGEMENT OF MALIGNANT

POLYPS

DEFINITIONS

CRC = invasion of neoplastic cells BEYOND the muscularis mucosa

Benign: dysplasia confined to the muscularis mucosa – negligible nodal risk

- pTis. The colonic mucosa has no lymphatics, so intramucosal CA does NOT metastasize
- abandon “carcinoma in situ,” “intramucosal adenocarcinoma,” “intraepithelial carcinoma”
- cured by complete endoscopic resection, nothing further

Malignant polyp: looks benign grossly, but invades THROUGH the muscularis mucosa into the submucosa and not into the muscularis propria = pT1

- ~12% of all polyps, and rising w/ screening

Pseudoinvasion: dysplastic epithelium misplaced into the submucosa, mimics invasion

- retains lamina propria, lacks other malignant features, hemosiderin present (ischemic origin)

PRECURSOR LESIONS

ADENOMAS

85-90% of sporadic CRC arises from adenomas

- tubular / tubulovillous (≥ 25% villous) / villous (≥ 75% villous) – WHO
- dysplasia graded by the revised Vienna classification
- LGD: change confined to the epithelial glands
- HGD: glandular crowding, cribriform architecture, severe cytologic atypia
- Advanced adenoma: ≥ 1 cm, predominantly villous, and/or HGD
- HGD is still BENIGN – no submucosal extension, no metastatic potential

Risk of invasive CA by size: < 1 cm 1.3%, 1-2 cm 9.6%, > 2 cm 46%, > 3 cm 76%

- no invasive CA in polyps < 6 mm
- 2-3 yrs for a < 5 mm adenoma to reach 1 cm; 2-5 yrs for a 1 cm adenoma to become CA

SERRATED POLYPS

Hyperplastic: most common, < 5 mm, rectosigmoid, round or oval, same color as mucosa – regarded as NON-neoplastic

Sessile serrated lesion (SSL): crypt distortion – horizontal crypts, dilated crypts, serrations extending to the crypt base

- right colon, larger than HPs, mucous cap, poorly defined lateral margins

Traditional serrated adenoma (TSA): rare villous polyp – cytoplasmic eosinophilia, elongated nuclei, ectopic crypts. “Pine cone” appearance, distal colon

SSL and TSA are true precursors – up to 25% of sporadic CRC

CARCINOGENIC PATHWAYS

Adenoma-carcinoma (CIN): APC loss → Wnt/β-catenin overactivation → KRAS → TP53 loss drives HGD → carcinoma

Serrated: BRAF or KRAS mutation → MAPK activation + CpG island methylation (CIMP) → tumor suppressors silenced

LESION ASSESSMENT

Document size, location, morphology, and pit/vascular pattern on EVERY polyp

Depth of invasion is the SINGLE most important feature for resectability and nodal risk

PARIS CLASSIFICATION

Threshold is 2.5 mm above the surrounding mucosa

- 0-I polypoid: Ip pedunculated, Isp subpedunculated, Is sessile
- 0-II nonpolypoid: IIa elevated, IIb flat, IIc depressed
- 0-III excavated
- mixed lesions named together – IIa + IIc, Is + IIa, Is + IIc

Risk of invasion is proportional to SIZE and degree of DEPRESSION

- Paris 0-I ≤ 5 mm: essentially 0% invasive CA
- Paris 0-III ≥ 15 mm: 90%
- Moss et al: depression (0-IIa + IIc) is an independent risk factor for submucosal invasion
- depression, ulceration, or gross wall deformity → deep invasion. Do NOT attempt endoscopic cure

LATERALLY SPREADING TUMORS

Nonpolypoid, > 10 mm, growing laterally rather than vertically. ~9% of polyps at COY

- LST-G granular: homogeneous < 2% invasion; MIXED nodules up to 30% if > 30 mm
- LST-NG non-granular: pseudo-depressed 12.5% if < 20 mm, 83% if > 30 mm
- Burgess et al: LST-NG or LST-G mixed in the RECTOSIGMOID carries the highest malignancy risk

PIT & VASCULAR PATTERN

Kudo pit pattern – chromoendoscopy w/ indigo carmine, or digital CE:

- I round: normal. II stellate or papillary: hyperplastic
- IIIs small tubular, IIIL large tubular, IV dendritic/gyrus-like: adenoma, resect endoscopically
- Vi irregular size & arrangement: superficial SMI
- Vn amorphous, nonstructured: DEEP SMI → surgery

NICE – narrow band imaging, works without magnification:

- 1: same or lighter color, no or isolated lacy vessels, uniform spots – hyperplastic/serrated
- 2: browner, brown vessels surrounding oval or branched white structures – adenoma
- 3: dark brown, disrupted or missing vessels, amorphous or absent surface – DEEP SMI
- 96.4% specificity for deep invasion in lesions > 10 mm

DEPTH OF INVASION

HAGGITT LEVELS (PEDUNCULATED)

- 0: confined to mucosa, muscularis mucosa intact – benign
- 1: through the muscularis mucosa, limited to the HEAD
- 2: into the NECK (head-stalk junction)
- 3: into any part of the STALK
- 4: into the submucosa of the bowel wall BELOW the stalk
- every sessile lesion w/ invasive CA is, by definition, level 4

KIKUCHI / KUDO SM LEVELS (SESSILE)

- sm1: upper 1/3 of the submucosa
- sm2: middle 1/3
- sm3: lower 1/3 – independent risk factor for nodal disease
- Haggitt 1-3 are all sm1; Haggitt 4 may be any of the three
- LIMITATION: defining the thirds requires the muscularis propria in the specimen, which a polypectomy rarely provides → measure absolute depth in μm from the muscularis mucosa instead

NODAL RISK BY DEPTH (KITAJIMA)

- pedunculated: 0% for Haggitt 1, and for Haggitt 2-3 when SM depth < 3000 μm
- non-pedunculated: 0% if SMI < 1000 μm , 3.9% if < 2000 μm , 17.1% if $\geq 2000 \mu\text{m}$

ADVERSE HISTOLOGIC FEATURES

Meta-analysis, 23 studies / 4510 pts – OR for lymph node metastasis:

- tumor budding (single cells or clusters at the invasive front): OR 7.74
- unfavorable grade – poorly differentiated or mucinous: OR 5.60
- lymphovascular invasion: OR 4.81
- SMI > 1000 μm : OR 3.87

Features are ADDITIVE

Margin < 1 mm, or indeterminate, also predicts residual cancer

- 1-2 mm is intermediate risk; > 2 mm is a clear margin

Reports routinely OMIT budding and LVI – have a GI pathologist review the slides at tumor board

ENDOSCOPIC CURE

WHAT MAKES A RESECTION CURATIVE

EN-BLOC removal is MANDATORY for any malignant polyp

- piecemeal = NON-CURATIVE. Fragmentation destroys orientation and margin interpretation
- NCCN: piecemeal EMR of an otherwise favorable malignant polyp STILL requires surgery
- $\leq 2 \text{ cm}$ can usually be taken en-bloc by EMR; > 2 cm needs ESD
- pin the specimen to cork – unpinned in formalin the edges curl, and lateral vs. deep margin and depth become unmeasurable

Curative criteria – ALL FOUR must be met:

- (1) all resection margins negative
- (2) SMI < 1000 μm
- (3) well or moderately differentiated
- (4) NO lymphovascular invasion and NO tumor budding

WHEN TO OPERATE INSTEAD

Endoscopic features of deep SMI – Paris 0-IIc or 0-III, ulceration, Kudo Vn, NICE 3

- biopsy the abnormal portion, TATTOO, and refer for oncologic resection
- tattoo in MULTIPLE QUADRANTS just distal to the lesion, photograph, document in the report
- EXCEPTION: pedunculated (Paris Ip) w/ deep features confined to the head, Haggitt 0-2 – en-bloc resection at the level of the stalk carries a favorable prognosis

Any adverse histologic feature, positive margin, or piecemeal removal → oncologic resection

Draw a CEA BEFORE resecting a suspected malignant polyp

Baseline chest / abd / pelvis CT once malignancy is confirmed

- WAIT 3-4 weeks after the polypectomy before ERUS or cross-sectional imaging – reactive adenopathy gets read as N+ and drives overtreatment
- if no tattoo was placed at the index scope, re-scope within 1-2 weeks and tattoo

ACPGBI RISK SCORE

Weighted, additive:

- margin < 1 mm: 4. Margin 1-2 mm: 1
- Haggitt level 4: 4. Kikuchi sm3: 4. Kikuchi sm2: 2
- poor differentiation: 3. LVI: 2. Mucinous: 1. Tumor budding: 1

Total score → residual CA risk → action:

- 0 very low, < 3%: routine f/u
- 1 low, < 5%: assess other factors, close f/u
- 2 medium, 5-10%: discuss risk/benefit of surgery
- 3 high, 8-15%: discuss, ERR TOWARD SURGERY
- ≥ 4 very high, > 20%: RESECT unless the pt is unfit

UK network, 173 pts: 37 (21.4%) went to primary surgery and 43% of those had residual disease; the 136 surveilled had 4.4% recurrence

RECTAL MALIGNANT POLYPS

Transanal endoscopic surgery (TES) is PREFERRED over flexible EMR/ESD in the rectum

- en-bloc, in either the submucosal OR full-thickness plane
- fragmentation < 10%, recurrence 5-11%
- full thickness = a “total biopsy” – gives true sm level, budding, and all high-risk features, so the pt can be properly stratified for salvage resection
- handles circumferential lesions and lesions extending into the anal canal
- LIMIT: rigid instruments – the proximal rectum is hard to reach

- downside of full thickness: added morbidity, functional derangement (esp. distal lesions), and extramural scarring that makes later proctectomy harder
- Western ESD lags Asia – R0 71.3% vs. 85.6%, bleeding 4.2% vs. 2.4%, perforation 8.6% vs. 4.5%
- N. American rectal ESD: en-bloc 82.5%, R0 74.9%, curative in 81.8% of favorable malignant polyps
- ERUS is reliable ONLY before polypectomy – post-polypectomy inflammation makes it useless

SURVEILLANCE

Recurrence and metastasis after endoscopic removal occur within 3-5 yrs

ESGE: COY at 6 months after ANY piecemeal resection of a polyp > 10 mm

JGES: COY within 3 yrs after en-bloc resection – local recurrence is rare

No consensus on CEA or CT interval – periodic, by expert opinion

Benign adenoma intervals, for reference:

- 1-2 tubular adenomas < 1 cm: 5-10 yrs
- advanced adenoma, or 3-10 adenomas: 3 yrs
- > 10 adenomas, or incomplete resection: < 3 yrs
- large sessile or piecemeal: 3-6 months, then 6-12 months, then annually to yr 5

24 MALIGNANT DISEASE: COLORECTAL CANCER —

PREOPERATIVE EVALUATION AND STAGING

DIAGNOSIS

Workup follows a positive screening test, unexplained iron-deficiency anemia, or symptoms. Screening begins at 45 (ACS) or 50 (USPSTF, NCCN) and stops between 75 and 86 by life expectancy

Any positive stool test → COLONOSCOPY. Stool tests triage, they do not replace endoscopy

- FOBT guaiac: 3 cards / 3 days, annual, ↓ CRC mortality 15-33%, but low specificity and needs dietary & med restriction. FIT: human globin, no restrictions, annual, USPSTF preferred

- stool DNA-FIT (Cologuard): FIT + KRAS, NDRG4, BMP3, β-actin, q3 yrs. 13% false pos, 8% false neg

CT colonography: CO2 per rectal tube, supine & lateral. Still needs a FULL cathartic prep, not covered by Medicare. Comparable to COY for polyps > 1 cm, poor below that – small lesions look like stool

- PROCEDURE OF CHOICE for incomplete colonoscopy or an obstructing lesion, though what it finds proximal to an obstruction cannot be biopsied anyway

Colonoscopy: most sensitive & specific – diagnostic, therapeutic, and preventive. Perforation 0.1%, incomplete in 5-10%. Synchronous CA in 3-5% of rectal CA pts, synchronous polyps up to 30%

BIOPSY & LOCALIZATION

Intraperitoneal colon CA headed for colectomy – biopsy may not change management

- but it detects Lynch pre-op, guides chemo if incurable, and insurers often deny staging without tissue

RECTAL tumors: ALWAYS biopsy, and send IHC for MMR

- neoadjuvant therapy alters the tumor and makes molecular analysis impossible afterward
- radiation oncology will not start without histologic confirmation

Tattoo every colonic lesion – endoscopic distances are notoriously inaccurate (cecum excepted)

- India ink, submucosal, DISTAL to the tumor, at least 2 quadrants. NEVER inject through the tumor – reports of implantation
- rectal tattoo error 2-21%, and submucosal tattoos are hard to see through a thick mesorectum
- tattoo causes reactive adenopathy and altered wall signal → OVERSTAGING on MRI. If not inked at dx, get the MRI and CT FIRST, then tattoo

DELINEATING COLON FROM RECTUM

NCCN: the rectum begins at a virtual line from the sacral promontory to the symphysis pubis on MRI, and ends at the upper border of the anorectal ring

Rigid proctoscopy thirds: low 0-6 cm, mid 7-11 cm, high 12-15 cm from the verge

Delphi consensus now favors the SIGMOID TAKEOFF on cross-sectional imaging – the end of the mesorectum, seen as an acute angulation. Reproducible, and tumor-board friendly

FOXTR0T: upfront chemo for locally advanced COLON CA, also an option for bulky upper rectal tumors

CLINICAL EVALUATION

Duration, pain, bleeding (mixed in vs. bright red vs. maroon), tenesmus, continence, caliber change, weight loss, anemia

Family Hx to trigger genetic counseling. Comorbidity, frailty screen, nutrition, prior abdominal surgery

DRE – the single most important test for a distal rectal tumor. Document:

- % of luminal circumference and the quadrant(s) involved
- distance from the distal edge of the tumor to the top of the anorectal ring. Firm vs. soft, mobile vs. tethered vs. FIXED
- involvement of sphincters, posterior vaginal wall, levators, prostate
- sphincter bulk and tone – needed if restorative proctectomy is contemplated

Proctoscopy:

- the OPERATING SURGEON does it – localization at the index colonoscopy is often wrong, especially “sigmoid” or “rectosigmoid” lesions that prove to be mid or distal rectum
- also the tool for assessing response to neoadjuvant therapy

Blood work:

- CBC, chemistry, CEA
- CEA < 5 ng/mL carries a better prognosis STAGE FOR STAGE than ≥ 5
- normalization after neoadjuvant a/w complete pathologic response
- failure to normalize after resection → residual or metastatic dz
- LFTs are neither sensitive nor specific for liver mets – NCCN no longer recommends them

AJCC 8TH EDITION

T:

- Tis: invasion of the lamina propria. T1: submucosa. T2: muscularis propria
- T3: subserosa or non-peritonealized pericolic / perirectal tissue
- T4a: PERFORATES the visceral peritoneum. T4b: directly invades other organs or structures
- perforation w/ tumor cells continuous to the serosa through inflammation = T4a
- in the low rectum, with no peritoneal covering, adherence to an adjacent organ = T4b

N:

- N1a 1 node. N1b 2-3 nodes. N2a 4-6 nodes. N2b ≥ 7 nodes
- a node counts as positive if the deposit is ≥ 0.2 mm
- isolated tumor cells (≤ 20 in the subcapsular sinus) = N0(i+), NOT stage III. Micrometastases (≥ 20 cells, or 0.2-2 mm) = N1 – N1mi is unnecessary, outcomes match > 2 mm
- N1c: NO positive nodes but tumor deposits in the subserosa or pericolic / perirectal soft tissue
 - N1c makes it STAGE III on its own; the NUMBER of deposits is not counted or added to the node count

M:

- M1a one organ (liver, lung, ovary, non-regional nodes), no peritoneal dz
- M1b more than one organ
- M1c PERITONEUM $\pm$ other organs – NEW in the 8th ed

Stage groups:

- I: T1-2 N0. IIA: T3 N0. IIB: T4a N0. IIC: T4b N0
- IIIA: T1-2 N1, or T1 N2a
- IIIB: T1-2 N2b, T2-3 N2a, T3-T4a N1
- IIIC: T4a N2a, T4b N1-2
- IVA M1a, IVB M1b, IVC M1c

Prefixes: c clinical, p pathologic, yp after neoadjuvant therapy

SYSTEMIC IMAGING

CT CHEST / ABDOMEN / PELVIS

Synchronous metastatic dz in 30% at presentation. Oral AND IV contrast

- liver mets in 20-34%. Staging sensitivity 85%, PPV 96%, false positive 4%
- MRI beats CT for liver lesions < 10 mm. Renal insufficiency: non-contrast chest CT + gadolinium MRI of abd/pelvis

CT CANNOT stage mesocolic or mesorectal nodes, and cannot give rectal T stage or fascial involvement – it cannot separate tumor from peritumoral fibrosis

- its value is DISTANT disease

PET-CT

NOT for initial staging. Use it for equivocal CT findings, to exclude extrahepatic dz before radical liver surgery, to confirm high metastatic risk (EMVI on MRI, high CEA), or when IV contrast is contraindicated

- FDG also accumulates in inflammation and adenomas – false positives, incl. post-XRT necrosis. False negatives in mucinous tumors and in anything < 10 mm

RECTAL CANCER LOCAL STAGING

ENDORECTAL ULTRASOUND

Water-filled balloon over a 7 or 10 MHz transducer – 10 preferred

5 layers: 1 white balloon-mucosa interface – 2 black mucosa & muscularis mucosa – 3 white submucosa –

4 black muscularis / fascia propria – 5 white interface with perirectal fat

uT0 mucosa only – uT1 submucosa – uT2 into but not through the muscularis propria – uT3 through into perirectal fat – uT4 adjacent organ

T stage accuracy 63-96%. Older meta-analysis of 42 studies / 5000 pts: sens 81-96%, spec 91-98%, but more recent data are worse at 55-82%

BEST USE: separating T1 from T2. Accuracy 73% for T1, sens 71%, spec 100%

- DO IT when local excision is on the table – LE suits low-risk T1, NOT T2
- can also stratify sm1 / sm2 / sm3

Nodes: accuracy 75%. The 5 mm size criterion predicts poorly against histology

- nonvisible $\rightarrow$ likely negative. Hyperechoic $\rightarrow$ inflammatory
- hypoechoic > 5 mm, or mixed echogenicity > 5 mm $\rightarrow$ treat as metastatic
- nodes < 5 mm are often invisible yet harbor CA 20-50% of the time

LIMITS: operator dependent; cannot tell inflammation or desmoplasia from tumor, so T2 vs. early T3 is unreliable

- CANNOT see the mesorectal fascia except at the vagina or seminal vesicles $\rightarrow$ USELESS for CRM
- do it BEFORE neoadjuvant therapy and BEFORE polypectomy

PELVIC MRI

STANDARD OF CARE for locally advanced rectal CA. Thin-slice high-resolution T2

- 3 planes: oblique axial PERPENDICULAR to the tumor, sagittal along the long axis, oblique coronal parallel to the anal canal
- do NOT use an endorectal coil or endorectal contrast – it stretches the rectum and ruins mural assessment
- gadolinium changed treatment in 24% by downstaging T, and improves EMVI detection

CRM – the closest distance from tumor to the MESORECTAL FASCIA

- POSITIVE = tumor within 1 mm
- sens 77%, spec 94%. The ONLY pre-op variable that predicted local recurrence, DFS, and OS
- negative CRM: 67% 5-yr survival vs. 47% if positive
- retraction away from the CRM after neoadjuvant is a GOOD prognostic sign

Accuracy rises with T stage – T3/T4 near 100%, but T2 vs. early T3 is still overstaged

ESMO T3 subclassification, not in AJCC: T3a < 1 mm, T3b 1-5 mm, T3c 6-15 mm, T3d > 15 mm beyond the muscularis propria

Nodes: MORPHOLOGY beats size – irregular border + mixed signal intensity, sens 85%, spec 97%; a meta-analysis of 21 studies is more sober at sens 77%, spec 71%

Low rectal CA (0-6 cm) = ~1/3 of all rectal CA. Staged by relation to the intersphincteric plane and levators

- invasion of the intersphincteric plane or levators: 18-FOLD ↑ CRM involvement → ELAPE or exenteration

Pelvic sidewall nodes: 15% in distal tumors vs. 8% higher up – a cause of recurrence despite a good TME

MRI risk groups:

- GOOD: T1-T3a/b, N0, no EMVI, CRM clear – local recurrence < 10%
- BAD: T3c/d-T4 or N1/2, CRM clear – 10-20%
- UGLY: CRM threatened (< 1 mm) or involved, EMVI present, or low CA into the intersphincteric plane or levators – > 20%

RESTAGING MRI

Controversial – performance is WORSE than at primary staging: yT sens 50.4% / spec 91.2%, yN 76.5% / 59.8%, yMRF 76.3% / 85.9%

mrTRG 1 complete – 2 dense fibrosis, no obvious tumor – 3 > 50% fibrosis/mucin – 4 mostly tumor – 5 no change

PROGNOSTIC FACTORS

PATHOLOGY

LVI – consider adjuvant chemo in stage I/II; strengthens the case for radical resection of a malignant polyp

PNI – independent prognosticator over LVI and nodes after neoadjuvant; perineural tumor may be radioresistant

Tumor budding – cells or clusters in the stroma or at the tumor edge, i.e. EMT. Treat like LVI/PNI

Poorly / undifferentiated grade is worse, stage-independent. Mucinous, signet ring (> 50% intracytoplasmic mucin), and adenosquamous are chemoresistant and do worse

dMMR / MSI-H – IHC on every CRC. dMMR w/ NORMAL BRAF → germline testing. Better prognosis than MSS despite poor differentiation; 5-FU less effective; PD-1 inhibitors approved for metastatic MSI-H

Minimum 12 nodes for accurate staging – fewer are retrieved after neoadjuvant, which may itself mark response

- lymph node RATIO: lower is better, independent predictor of OS and DFS
- IDEA: adequate harvest w/ only 1-2 positive nodes → 3 months of FOLFOX = 6 months

Extranodal tumor deposits – the STRONGEST independent predictor of poor outcome; same T stage, 5-yr survival 37% without vs. 2% with

Mesorectal grade – independent predictor of local and overall recurrence. Intraoperative rectal perforation → record as pT4

EMVI

Tumor cells in the microvasculature BEYOND the muscularis propria. Incidence 9-61%

Brown MRI grading: spec 88-96% but sens only 29-62% – small vessels cannot be resolved

Predicts local recurrence, synchronous mets, and a 4-FOLD risk of metachronous mets within 1 yr – even a T1 or T2 w/ MRI-detected EMVI should be considered for chemotherapy

CRM POSITIVITY

Predictors: T3/T4, tumor < 4 cm from the verge, ANTERIOR quadrant, EMVI

- ALL FOUR → incomplete resection risk up to 60% → total neoadjuvant therapy

NCCN: neoadjuvant for all clinical stage II (cT3/4 N0) and III – heterogeneous, some overtreated. UK and ESMO now select by MRI rather than TNM alone

Colon CA has a radial margin too – mesenteric attachment plus the non-serosalized / retroperitoneal cut edge. < 1 mm independently predicts survival and recurrence even after CME

AGE

< 45 is worse overall – adverse features, advanced stage – but stage for stage the young may do BETTER, being treated more aggressively

Older pts have worse baseline continence and tolerate a diverting ileostomy poorly (dehydration)

- frail w/ CRM NOT threatened → upfront non-restorative proctectomy, Hartmann, or APR; if CRM threatened → short course XRT (5 Gy x 5, total 25 Gy) then resection

PREOPERATIVE PREPARATION

Document baseline fecal, urinary, and sexual function before any restorative proctectomy

Enterostomal therapist for stoma marking and teaching.

Child-bearing age: sperm banking or egg harvest BEFORE radiation or operation

Risk-stratify and optimize – ACS NSQIP risk calculator, “Strong for Surgery” checklist

ASCRS pre-op checklist – compliance a/w better specimens and lower 30-day morbidity:

- pathology reviewed and invasive CA confirmed; complete colonic evaluation if unobstructed
- tumor location – distance from verge, length, quadrant, relation to levators & anorectal ring. Family Hx, continence, and sexual function documented
- clinical staging of the primary (MRI ± ERUS) and for distant dz (chest / abd / pelvis). CEA drawn. Neoadjuvant considered and documented for > T2 or node positive dz
- if neoadjuvant given, restage and re-localize just before the operation. Tumor board documented, stoma site marked

NAPRC: treatment within 60 days of dx, negative proximal / distal / circumferential margins, ≥ 12 nodes

25 MALIGNANT DISEASE: COLON CANCER SURGICAL

TREATMENT — PRINCIPLES OF COLECTOMY

PREOPERATIVE LOCALIZATION

Mislocalization by colonoscopy alone: 11-21%, changes the planned operation in 11%

- worse once cecal & rectal tumors are excluded – no landmarks in distal ascending, transverse, left colon

NEVER trust “cm from the anal verge” – repeat flex sig on any left-sided neoplasm before booking

- misclassifying a rectal CA as sigmoid = wrong treatment entirely

Tattoo:

- India ink, SUBMUCOSAL. Full-thickness → ink sprayed through the abdomen, useless
- raise a 0.5-1.0 mL saline bleb, then inject 0.75-1.0 mL ink into the bleb
- 3 or more quadrants, opposite wall or just DISTAL to the tumor. Document it in the report
- ↓ operative time, ensures the right segment comes out

Clip + imaging:

- supine plain film right after the scope while the colon is still gassed, or staging CT
- useful when ink is unavailable

Be prepared for intra-op colonoscopy – CO2 insufflation to limit bowel dilatation

ONCOLOGIC PRINCIPLES

EXTENT OF RESECTION

≥ 5 cm proximal AND distal margin – minimizes anastomotic recurrence

- with high ligation it is nearly impossible to get < 5 cm, the ends go ischemic first

Feeding vessel taken AT ITS ORIGIN – maximizes lymphadenectomy

Surgeon inks the non-peritonealized margin, as is routine for a proctectomy specimen

HIGH VS. LOW TIE (IMA)

High ligation gives a higher NODE YIELD

- but NO functional or oncologic difference, high vs. low
- St. Mark's, 1,370 pts: no difference if the left colic preserved – high ligation actually fared worse
- French Association for Surgical Research: no difference

No universal nomenclature – IMA is only a few cm if it ends where it branches into superior hemorrhoidal + left colic

- others call it IMA until it crosses the common iliac. That designation ignores branch-point naming

LYMPHADENECTOMY

Adequate = vessels taken at origin + ≥ 12 nodes harvested and examined

Node count and involved:harvested RATIO = markers of adequacy of resection, a/w outcome

Extended lymphadenectomy for para-aortic nodes: benefit in highly selected pts only, NOT standard practice

COMPLETE MESOCOLIC EXCISION – TECHNIQUE IN CH 37

Tumor + lymphovascular supply + CENTRAL vascular ligation + mesocolon in an INTACT visceral peritoneal envelope

Rationale borrowed from TME. NO RCT vs. standard colectomy exists

Bertelsen et al, right colon: 5-yr recurrence 9.7% vs. 17.9%; median nodes 38 vs. 21

- intact peritoneal lining a/w 15% improvement in survival
- central ligation captures apical nodes and “skip lesions,” ~5% of cases
- standardizes the operation – the real benefit may just be refocusing on the basic principles
- Drawbacks: technical difficulty, longer OR
- SMV injury in 1.6% of right hemicolectomies – the most feared complication
- laparoscopic CME feasible & safe. No functional difference vs. conventional

NO-TOUCH

Pedicle ligated BEFORE mobilization, on the theory of shed tumor cells

Garcia-Olmo: no circulating tumor cells detected with tumor manipulation

RCT vs. conventional colectomy: NO difference in DFS, OS or recurrence-free survival in stage II/III
→ historical only

EN BLOC RESECTION OF ADHERENT ORGANS

Adhesions to adjacent structures are MALIGNANT in ~40%

If uncertain – abutment vs. true invasion – take it EN BLOC. Never divide the adhesion

- without en bloc: ↑ recurrence, ↓ survival

Most commonly involved: abdominal wall, bladder, duodenum, omentum, ovary, peritoneum, retroperitoneum, small bowel, stomach, ureter, uterus

Abdominal wall → en bloc resection + reconstruction

Tail of pancreas / spleen → distal pancreatectomy + splenectomy. Case reports of en bloc pancreaticoduodenectomy

Neoadjuvant for locally advanced colon CA:

- FOXTROT: downstaging, less apical node involvement, fewer + margins, ↓ incomplete resection rate
- regression in 59%, some pCR. Well tolerated, no ↑ peri-op morbidity
- NCCN: consider for clinical T4b
- chemo/XRT an option for sigmoid tumors invading bladder or pelvic organs if small bowel dose can be limited

RESECTION BY TUMOR LOCATION

CECUM & ASCENDING COLON

Right hemicolectomy with ileocolic anastomosis

- boundaries: ~10 cm proximal to the ICV → proximal transverse colon

- ileocolic ligated at origin adjacent to the duodenum – dissect the duodenum free and PROTECT it first
- right colic (if present) and right branch of middle colic at their origins
- medial → lateral or lateral → medial: same operation, order reversed
- anastomosis intra- or extracorporeal, hand-sewn or stapled, any configuration – NO technique superior
- mesenteric defect closure controversial – defect is large, obstruction unlikely
- mobilization along ANATOMIC PLANES improves prognosis

HEPATIC FLEXURE

Proximal flexure lesion → right hemicolectomy may be adequate

Otherwise EXTENDED right: terminal ileum → distal transverse colon

- take the main middle colic trunk provided retrograde IMA flow will perfuse the splenic flexure
- open the lesser sac along its ENTIRE length for exposure to the blood supply

Splenectomy:

- required in < 1% of splenic flexure mobilizations
- inadvertent = capsular tear from poor exposure + aggressive retraction
- ↑ morbidity, ↑ mortality, ↑ post-op sepsis. Varty: no effect on long-term survival

TRANSVERSE COLON

Supply is middle colic plus right and left colic – pick the operation that removes the draining mesentery

Proximal transverse → extended right

Mid to distal transverse → extended right (most surgeons), extended left, or subtotal

Extended LEFT = main middle colic trunk + left colic

Segmental transverse colectomy:

- only for a mid-transverse tumor in a REDUNDANT colon
- end-to-end colocolic – side-to-side sits under tension as the mesenteries retract
- concern for adequacy of the lymphadenectomy
- otherwise reserve for palliation or the frail

SPLenic FLEXURE & PROXIMAL DESCENDING

Left colectomy: high ligation of the LEFT COLIC + left branch of the middle colic

- transverse-to-sigmoid anastomosis
- PRESERVE the root of the IMA and superior hemorrhoidal to perfuse the retained sigmoid
- if a colorectal anastomosis is made instead, the IMA root can be divided
- fully mobilize the splenic flexure, take the left side of the omentum

DISTAL DESCENDING

Formal left colectomy including the sigmoid, with colorectal anastomosis

- IMA at its origin – identify and preserve the LEFT URETER
- IMV near the ligament of Treitz at the inferior border of the pancreas – this is what buys REACH
- mobilize the transverse colon, release the omentum off the stomach
- retroileal anastomosis if reach is still short

SIGMOID COLON

Proximal sigmoid → left colectomy, for node harvest

Mid & distal sigmoid → anterior resection / sigmoid colectomy

- NO proven oncologic benefit to a formal left colectomy for distal sigmoid tumors
- incise peritoneum from IMA origin to just distal to the sacral promontory
- stay just deep to the arc of the superior hemorrhoidal vessels → preserves hypogastric nerves, left ureter, gonadals
- ureteric injury usually occurs at the level of the iliac artery
- low lithotomy, endoscopic inspection + CO2 leak test every time

SPECIAL CIRCUMSTANCES

OBSTRUCTION & PERFORATION

15% of colon CA presents with acute obstruction or perforation

Water-soluble contrast enema unless recently scoped

- confirms Dx, evaluates for synchronous distal lesions, preps the distal colon for intra-op scope

Right-sided obstruction:

- right colectomy – the dilated segment comes out with the specimen
- competent ICV → ileum of reasonable caliber → primary anastomosis if the pt is otherwise well
- dilated, edematous ileum → end ileostomy + mucus fistula, venting the corner of the transverse staple line
- alternatives: closed distal segment left in (ONLY if no synchronous distal lesions), formal mucus fistula, or ileocolic anastomosis + diverting ileostomy

Distal obstruction:

- proximal colon ischemic → SUBTOTAL colectomy
- dilated but viable → endoluminal stent as a BRIDGE to formal resection
- otherwise Hartmann with end colostomy
- primary anastomosis only RARELY – leak risk. If done, on-table lavage to clear the diverted segment

Perforation:

- same principles, plus a high recurrence rate and a real need for adjuvant chemo
- AVOID a high-risk anastomosis whose leak would delay or obviate chemo

LYNCH SYNDROME

~3% of all colorectal CA. Metachronous risk from the germline defect

ASCRS: TOTAL or SUBTOTAL abdominal colectomy,
NOT segmental, for a curable colon CA in Lynch

26 MALIGNANT DISEASE: RECTAL CANCER —

NEOADJUVANT THERAPY

STAGING AS IT DRIVES NEOADJUVANT RX

MRI = modality of choice for local staging – has supplanted TRUS (operator dependent, only moderate accuracy)

- gives T & N stage, relation to sphincter complex and peritoneal reflection, EMVI, adjacent organ invasion
- MOST IMPORTANTLY the relation of tumor to the mesorectal fascia = radiologic CRM

CRM threatened / involved: tumor ≤ 1 mm from the mesorectal fascia, breaching it, or invading the intersphincteric plane

- + CRM $\rightarrow$ $\uparrow$ local recurrence, $\uparrow$ mets, $\uparrow$ death. True CRM is only knowable histologically

No MRI available $\rightarrow$ exam + TRUS + pelvic CT is adequate

NCCN rectum: line from sacral promontory to top of pubic symphysis, down to the palpable top of the anorectal ring

Who gets radiation:

- NCCN (US): ALL clinical stage II & III
- ESMO: proctectomy alone allowed for early cT3 (< 5 mm beyond muscularis propria)
- ESMO even permits surgery alone for cN1 upper & mid rectum if the CRM is free on imaging

Restaging after neoadjuvant:

- no agreed recommendation. Consider it if the CRM was threatened – retraction off the CRM = better prognosis
- neither XRT nor chemo kills in a wave front – resect ALL tissue originally involved, regardless of appearance
- the one real indication: when nonoperative management is being considered

THE FOUNDATION TRIALS

Swedish Rectal Cancer Trial: short-course + immediate surgery vs. surgery alone – LR 11% vs. 27%, with an OS and DFS benefit

- criticized: a 27% failure rate in the surgery arm = bad surgery, so XRT only rescued poor technique

Dutch TME Trial: short-course + TME vs. TME alone.

Surgeons proctored, pathology standardized

- 2-yr LR 2.4% vs. 8.2%; 10-yr 5% vs. 11%
- NO difference in OS. More perineal wound complications with XRT

$\rightarrow$ even with optimal surgery, neoadjuvant XRT improves local control

German CAO/ARO/AIO-94: pre-op vs. post-op chemoradiation, 50.4 Gy / 28 fx (post-op arm got a 5.4 Gy boost)

- LR 6% vs. 13%. NO OS or DFS difference
- completed chemo/XRT 92% vs. 54%; completed full chemo 89% vs. 50%

- acute AND chronic toxicity worse post-op

$\rightarrow$ PRE-OP is the standard

Uppsala: pre-op 25.5 Gy vs. post-op 60 Gy – LR 12% vs. 21%, no survival difference

- half the post-op arm had XRT delayed by prolonged recovery from surgery

EORTC: pre-op 34.5 Gy dropped LR 30% $\rightarrow$ 15%

MRC, fixed & tethered tumors: 40 Gy / 20 fx, surgery ≥ 4 wks later – better local control and DFS, no $\uparrow$ complications

Bottom line of the era: XRT improves LOCAL CONTROL only. Pre-op beats post-op for both control and toxicity

SHORT-COURSE VS. LONG-COURSE

Short course: 25 Gy / 5 fx. Cheaper, faster, less early toxicity, less time away from home

Long course: 50.4 Gy / 28 fx with concurrent 5-FU – INFUSIONAL beats bolus

Polish trial (cT3/cT4): NO difference in sphincter preservation, OS or DFS. 4-yr LR 11% vs. 16% (NS). Late toxicity equal

Trans-Tasman (cT3, infusional 5-FU): no difference in LR or survival, no difference in severe late toxicity

Downstaging is a function of the INTERVAL, not the schedule

- short course only looked non-downstaging because surgery was at 1-2 wks

Neoadjuvant Rx should NOT change the extent of proctectomy or “convert” an APR

Stockholm III: short-course/immediate vs.

short-course/delayed 4-8 wks vs. long-course/delayed

- significantly MORE surgical complications with short course + immediate surgery

$\rightarrow$ DELAY surgery after short course – and the delay makes room for systemic chemo

TOTAL NEOADJUVANT THERAPY (TNT)

RATIONALE

Local control is solved – LR now 5-10%. Distant failure is not: 30-40% in T3, T4 or N+

In the old sequence systemic chemo does not start until ≥ 21 weeks after treatment begins

- every month adjuvant chemo is delayed $\rightarrow$ 14% relative $\downarrow$ in OS
- compliance collapses post-op: complications, poor performance status, patient refusal

Move systemic chemo to the FRONT – earlier treatment of micrometastases, better tolerance, better delivery

PCR AS AN ENDPOINT

pCR after long-course chemo/XRT: 15-20%. Downstaging 40-50%

pCR is TIME dependent, not only regimen dependent – dead cells take time to resorb and the pathologist cannot tell live from dead

- longer interval $\rightarrow$ higher pCR, up to a limit

- NEVER compare two regimens by pCR unless the surgical interval was the same

CHEMOTHERAPY ALONE – SKIPPING RADIATION

XRT toxicity is the motive: sexual & bladder dysfunction, sphincter dysfunction, pelvic fractures, marrow suppression

MSKCC pilot, 32 pts cT3 at 5-12 cm: 6 cycles FOLFOX ± bevacizumab, XRT held for nonresponders

- R0 100% (only 2 needed XRT), pCR 25%, 4-yr LR 0%, DFS 84%. 72% were cN+

FOWARC (China, 495 pts): chemo/XRT vs. FOLFOX + XRT vs. FOLFOX alone

- pCR 14% / 27.5% / 6.6%
- 3-yr DFS 72.9 / 77.2 / 73.5% – NO difference. LR 8 / 7 / 8.3%. OS ~90% in all arms
- anastomotic leak LOWEST in the chemo-only arm: 8% vs. 20% vs. 18%

PROSPECT: FOLFOX with SELECTIVE radiation vs. standard chemo/XRT. 1194 pts, accrual closed 2019

OXALIPLATIN CONCURRENT WITH XRT

STAR-01 (747 pts): pCR 16% in both arms. Grade 3/4 toxicity 24% vs. 8%

NSABP R-04 (1608 pts, 4 arms): pCR no different for 5-FU vs. capecitabine (17.8 vs. 20.7%) or ± oxaliplatin (19.5 vs. 17.8%)

- no difference in sphincter-saving surgery, 3-yr LR, 5-yr DFS or OS. More grade 3/4 diarrhea with oxaliplatin

CAO/ARO/AIO-04 (1265 pts): small pCR gain 17% vs. 13%, 3-yr DFS 75.9% vs. 71.2%

- but the oxaliplatin arm received 8 cycles vs. 4 – confounded

→ concurrent oxaliplatin buys toxicity, not oncologic benefit

CONSOLIDATION VS. INDUCTION

Consolidation: systemic chemo AFTER chemo/XRT, during the resting interval

Induction: systemic chemo BEFORE chemo/XRT

Timing Consortium, 4 sequential groups, 259 pts: 0 / 2 / 4 / 6 cycles FOLFOX after chemo/XRT

- pCR 18% / 25% / 30% / 38% (p = 0.0036), no ↑ toxicity
- 5-yr DFS 50% with no chemo vs. 81% with ANY chemo. No difference among 2, 4, 6 cycles

Spanish GCR-3: induction vs. adjuvant CAPOX – same pCR, DFS, LR, but protocol completion 91% vs. 54%

CAO/ARO/AIO-12 (306 pts): consolidation pCR 25% vs. induction 17% – ONLY the consolidation arm met the hypothesis

- induction gives better delivery of the CHEMO (97% full XRT dose in the upfront-XRT arm vs. 91%)
- consolidation gives the higher pCR and a longer window to judge response

MSKCC cohort (651 pts): TNT → more total chemo delivered, fewer dose reductions, more likely to get > 6 cycles

SHORT-COURSE RADIATION + SYSTEMIC CHEMO

RAPIDO: high risk only (cT4a/b, cN2, EMVI, involved MRF, lateral nodes) – 5 Gy x 5, 2 wk rest, 6 cycles CAPOX, surgery at 4 wks, vs. long-course chemo/XRT

- pCR 28% vs. 14%; 3-yr disease-related treatment failure 23.7% vs. 30.4%. No OS difference

PRODIGE-23: induction FOLFIRINOX x 6 → chemo/XRT → surgery → adjuvant, vs. standard chemo/XRT

- pCR 28% vs. 12%, 3-yr DFS 76% vs. 69%

Polish II (cT4 or fixed cT3): short course + 3 cycles FOLFOX vs. long-course chemo/XRT

- R0 77% vs. 71%, pCR 16% vs. 12% – both NS. 3-yr OS 73% vs. 65%

- at 7 yrs NO difference in OS, DFS or LR

- post-op complications identical → the extra interval does NOT make the surgery harder

Washington U pilot: short course + 4 cycles FOLFOX, surgery at 4 wks – downstaging 70%, pCR 29%

- vs. matched long-course: 3-yr distant metastasis-free 88% vs. 70%, DFS 85% vs. 68%

WATCH & WAIT – AS A CONSEQUENCE OF TNT (DETAIL IN CH 28)

Habr-Gama 2004: first series managed nonoperatively after a complete clinical response, pCR rate 26%

- refined regimen 45 Gy + 9 Gy boost, 3 cycles 5-FU/LV during XRT and 3 after → 68% cCR at 8 weeks
- 197 selected cCR pts managed nonoperatively: 5-yr DFS 60%

Higher pCR from TNT ↑ the cCR rate → organ

preservation is now a stated goal, not an accident

This is the one setting where local RESTAGING actually changes management

BOTTOM LINE

Pre-op beats post-op. Short course = long course for local control and survival

NO neoadjuvant regimen has improved OS – every one of them improves LOCAL CONTROL

Concurrent oxaliplatin: NOT worth the toxicity. Sequential (TNT) systemic chemo: yes

NCCN now permits essentially any sequence of the three modalities, traditional or TNT

Who can skip XRT: early cT3 (< 5 mm beyond the muscularis) with a clear MRF by ESMO criteria

- and, pending PROSPECT, chemo alone with radiation held in reserve for nonresponders

Adjuvant therapy after resection belongs to ch 30

27 MALIGNANT DISEASE: RECTAL CANCER — LOCAL

EXCISION

PATIENT SELECTION

The trade: ↓ morbidity and better function bought with ↑ local recurrence. COUNSEL the pt explicitly
Stage cannot be known until AFTER the excision – nearly all “by stage” LE data is retrospectively selected, biased in favor of LE

~15% of rectal CA presents as stage I

Workup:

- DRE: character, fixation, relation to the anorectal ring. Assess sphincter bulk & tone – full-thickness LE can take part of the IAS
- CEA, CT chest / abdomen / pelvis
- rectal-protocol MRI or TRUS. MRI separates cT1-2 from cT3-4 and reads the mesorectal fascia
- MRI is LESS accurate for nodes – heterogeneous signal and irregular margins suggest involvement

Classic criteria:

- T1, well to moderately differentiated
- NO lymphovascular or perineural invasion
- < 3 cm and < 1/3 of the circumference
- < 8 cm from the anal verge for standard transanal – higher needs TEM or TAMIS

Anterior lesions: LE scars the plane and adheres to adjacent organs, compromising a later proctectomy
LE pts require MORE frequent surveillance – matters for pts with limited resources or travel

PREDICTING NODAL DISEASE

Occult nodal disease is the main driver of the higher local recurrence after LE

Depth – Kikuchi SM level:

- SM1, superficial 1/3: 3%
- SM3, deep 1/3: up to 23% – behaves like a T2
- LE acceptable for SM1 and SM2 only
- SM level is readable ONLY after full- or partial-thickness excision or ESD – a post-hoc tool for deciding on completion surgery

By T stage:

- T1: 6-11% overall; 6% if NO adverse histology (Swedish registry)
- T2: 30-40% occult nodal involvement

Lymphovascular invasion – the most consistent predictor:

- Chang, 943 pts: OR 11.5
- pT1: LVI– PD– 7.5% → LVI+ 60% → LVI+ PD+ 100%
- pT2: LVI– PD– 15.3% → LVI+ 67% → LVI+ PD+ 100%
- Hogan: LVI also a/w SYSTEMIC recurrence in rectal CA (HR 2.57) and ↓ OS

Poor differentiation:

- Bosch meta-analysis, 17 studies: RR 4.9 for nodal mets

Tumor Budding – Hase 1993, ITBCC 2016:

- a single tumor cell or a cluster of ≤ 4 cells ahead of the invasive front

- INDEPENDENT predictor of nodal mets in pT1 and of survival in stage II
- counted on H&E, ONE hotspot of 0.785 mm² at the invasive front, three-tier system
- NOT the same as tumor grade. Now required in synoptic reporting

TECHNIQUE

FULL-THICKNESS en bloc excision for known or suspected cancer – perirectal fat at the base

- partial thickness only for benign-appearing lesions – preserves the wall and every future option

Mark 5-10 mm circumferentially with cautery before cutting
Bowel prep for visualization (an enema may suffice), IV antibiotics within 1 hr

Position the tumor DEPENDENT – anterior tumor → prone
Close TRANSVERSELY to avoid stricture

- extraperitoneal defect may be left open to granulate
- peritoneal entry MUST be closed – laparoscopic assist and pneumatic leak test

TEM – Buess, early 1980s:

- 4 cm table-mounted operating proctoscope, continuous CO2 pneumorectum, 3 instrument ports
- reaches proximal lesions that standard transanal excision cannot
- limits: capital cost, steep learning curve, restricted instrument mobility, insufflation problems
- position the tumor “toward the sky” so it falls away from the wall

TAMIS – Atallah 2010:

- flexible disposable single port, standard laparoscopic instruments, far cheaper
- multiple quadrants in one setup, no positioning constraint
- has largely displaced TEM for mid and proximal lesions
- hybrid with a transanal approach if the lesion sits at the top of the sphincter

COMPLICATIONS

Overall complication rate LOWER than radical surgery, regardless of platform

Urinary retention – MOST COMMON, up to 5%. Self-limited, from urethral pressure, anal stretch, edema, pain

Bleeding up to 5%, several days post-op – suture line dehiscence, scab sloughing, anticoagulation

- cautery, suture, epinephrine injection. Major intra-op bleeding is rare

Pelvic abscess – open vs. closed extraperitoneal defect

- RCT showed no difference in early or late complications (only 44 pts, underpowered)
- 991-pt matched analysis: no difference in 30-day morbidity

→ management of the defect is at the surgeon's discretion

ONCOLOGIC RESULTS

T1

LE alone is ACCEPTABLE for favorable T1 – SEER and 2 meta-analyses show equivalent cancer-specific and 5-yr OS vs. radical

- caveats: only ONE (underpowered) RCT exists, and one meta-analysis found inferior oncologic results overall

Positive margin → ↑ recurrence and ↓ 5-yr OS

UK 21-center TEM collaborative, 424 pts: + margins in 11% of pT1, 23% of pT2, 42% of pT3

- LR after TEM for T1: 10% / 13% / 19% at 2, 3, 5 yrs
- LR predicted by depth of invasion, maximum diameter, and LVI
- favorable (SM1, LVI-): 3-8%. Unfavorable (SM2-3, LVI+): 18-42%

Junginger, median f/u 8.6 yrs: low- vs. high-risk 5- and 10-yr LR 7%/12% vs. 32%/35%

- cancer-specific survival 98%/91% vs. 84%/74%

T2

RADICAL RESECTION REMAINS THE STANDARD
unless comorbidity is prohibitive or the pt refuses a stoma

LE alone: LR 13-30%, some series to 66%

- vs. proctectomy: 5-yr LR 47% vs. 6%, OS 65% vs. 81%
- > 20% of T2 pts in SEER are being treated by LE, with suboptimal survival

LE + ADJUVANT CHEMORADIATION

CALGB 8984: T1 by LE alone vs. T2 by LE + adjuvant chemo/XRT – T2 still had worse 10-yr OS and DFS, LR 18% vs. 8%

Meta-analysis, 14 studies, T2 after LE: LR 15% with adjuvant chemo/XRT vs. 10% with completion proctectomy

Cutting systematic review: LR 6% T1, 14% T2, 34% T3
→ adjuvant Rx after LE is INFERIOR to radical surgery, but better than LE alone

NEOADJUVANT CHEMORADIATION THEN LE

Premise: up to 30% pCR, so occult mesorectal nodes may be sterilized

ACOSOG Z6041, phase II, 84 pts with T2: downstaging in 64%

Lezoche RCT, LE vs. lap proctectomy: all R0, LR 8% vs. 6%, cancer-related survival 89% vs. 94% (NS)

GRECCAR 2, cT2-3, 145 pts: 3-yr LR 6% vs. 3%, OS 89% vs. 95% – both NS

- only 8% of those taken on to completion proctectomy had nodal disease – most were operated for nothing

CARTS, cT1-3 after chemo/XRT: 5-yr LR 8%, DFS 82%, OS 83%

Major downside: WOUND HEALING – the rectal wall closure breaks down in the radiated field

If there is a complete clinical response, LE only helps when viable tumor is in the WALL but not the nodes (see ch 28)

SALVAGE

Use of LE has more than DOUBLED over two decades
Local recurrence is the dominant failure pattern: 20-30%
Bikhchandani, 27 pts multimodal salvage: R0 93%, 5-yr recurrence-free 47%, 5-yr OS 50%

- most re-recurrences were DISTANT

You et al, extended resection for local failure: R0 only 80%, 3-yr re-recurrence-free 43%, 5-yr OS 63%

- compare 92-97% 5-yr DFS after up-front radical surgery for early rectal CA

Friel: salvage results inferior to primary radical treatment

- salvage works in only some pts. Do NOT select for LE on the assumption that salvage will bail you out

QUALITY OF LIFE

Better bowel function and better QOL than radical resection

Pucciarelli: minimal impact on anorectal function or QOL at 1 yr after LE + neoadjuvant chemo/XRT

CARTS: HRQOL back to baseline, with better emotional well-being than radical surgery

BOTTOM LINE

T1, favorable histology, negative margin → LE ALONE

T1 with LVI, poor differentiation, budding, or SM3 → completion proctectomy, or chemo/XRT if unfit

T2 → radical resection. LE only for palliation, prohibitive risk, or a trial

T3 → LE for palliation only, usually for bleeding

Any node positivity → RADICAL

28 MALIGNANT DISEASE: RECTAL CANCER —

NONOPERATIVE MANAGEMENT

DEFINITIONS

pCR: ypT0N0 after proctectomy, or ypT0 after full-thickness LE w/ no radiologic nodal disease

cCR: no residual tumor on clinical, endoscopic AND radiologic exam

Watch & Wait (WW): cCR + no immediate surgery + strict surveillance

- cCR is the PREREQUISITE for entering WW – no cCR, no WW

Local regrowth: tumor reappearing after an apparent cCR

- NOT the same as local recurrence (tumor in the pelvis after curative-intent surgery)
- regrowth is mostly in the bowel wall → DRE & flex sig detect it

Induction chemo: given BEFORE CRT. Consolidation chemo: during/after CRT, before surgery

TNT: CRT + full course of systemic chemo (8 cycles FOLFOX or 5 cycles CapeOX)

RATIONALE

Concept borrowed from anal CA – pCR seen in APR specimens after nCRT

Avoids: permanent stoma, temporary stoma & its reversal, leak, peri-op mortality

Avoids LARS, and the sexual, urinary, financial & social burden of proctectomy

Sphincter preservation is NOT the same as function preservation

Accidental vs. Intentional WW:

- Accidental: pt refused surgery or unfit – still the only version accepted at many centers
- Intentional: distal CA likely to need coloanal or APR, treated w/ optimal nCRT, restaged, entered deliberately
- WW discussed BEFORE nCRT is started

PATIENT SELECTION

BASELINE STAGE

More advanced tumor → less likely to achieve cCR

High-risk features that MANDATE nCRT: mrCRM ≤ 1 mm (mrT3c,d or T4), EMVI+, N1c/N2, lateral pelvic nodes

- none of these is a contraindication to WW, but cCR is less likely
- and the response is harder to read w/ extensive disease – fibrosis vs. tumor

Intermediate (mrT3a,b, N0/1, no high-risk features): benefit of nCRT debated

- MERCURY: low local recurrence w/ proctectomy alone
- still offer nCRT if distal, for the chance at organ preservation

Early (mrT1/T2N0): not standard nCRT candidates

- pCR rate is HIGHER than in advanced disease

- reasonable to offer nCRT for the sole purpose of avoiding a stoma

TUMOR LOCATION

DISTAL rectum benefits most – the pt who would otherwise need coloanal or APR

- also the tumors accessible to DRE, so they can be followed

mrT2 beyond the reach of DRE is surrounded by mesorectal fat, low CRM risk → up-front proctectomy

- exception: obese pt, long anal canal, tumor just above the anorectal ring

MRI defines relation to sphincter, levators, prostate/vagina, peritoneal reflection

ENDOSCOPIC FEATURES

Size predicts response – smaller tumors respond more completely

Ulceration and tethering are NOT exclusion criteria

Large circumferential tumors that heal into a concentric stenotic scar → poor WW candidates

- lumen can't be assessed, so surveillance is not possible

Document baseline endoscopic images – you are comparing against them for years

Confirm invasive adenocarcinoma first. If it is an ESD/LE lesion, do not irradiate it

ASSESSMENT OF RESPONSE

First assessment 6–8 weeks after completion of nCRT

- classic 6-week interval to surgery gives pCR ~18%
- response is TIME-DEPENDENT and nonlinear – longer interval, higher pCR
- consolidation chemo during the waiting period ↑ cCR and ↑ organ preservation

Three groups, three paths:

Incomplete (iCR): deep ulcer, elevated borders, distorted wall; hard mass or stenosis; mrTRG3-5, diffusion restriction

- → SURGERY

Near-complete (nCR): no mass, only superficial shallow ulcer; minimal/questionable irregularity; mrTRG2

- → reassess in 8–12 weeks. Must show CONTINUED response

Complete (cCR): whitening ± telangiectasias; smooth on DRE; low signal T2, no restriction, mrTRG1

- → WW, reassess in 12 weeks

Most reach strict cCR criteria by 28–34 weeks after RT; a few take up to 52 weeks

Any stall in response, or any sign of regrowth → operate

CRITERIA FOR A CCR – THREE PILLARS

DRE:

- regular smooth mucosal surface, only minor induration
- ulceration, nodule, stenosis or mass → NOT a cCR

Endoscopy:

- whitening of the mucosa, submucosal telangiectasias, no ulcer/mass/stenosis

- flexible > rigid: retroflexion, documentation, comfort.
- NBI helps

MRI:

- low-signal intensity at the original tumor bed on T2W = mrTRG1
- NO diffusion restriction on DWI in the corresponding area

Each modality alone is accurate but imperfect – combine them

Maas et al: all three concordant for absence of tumor → 98% accuracy

Biopsy:

- residual cells are randomly distributed through the wall after nCRT
- a negative biopsy is NOT required for WW, and does NOT exclude residual tumor
- AVOID biopsy in the incomplete responder – a false negative arms the pt to refuse an operation he needs

IMAGING

ERUS largely abandoned – discomfort, and cannot read the mesorectum

MRI is the modality of choice. mrTRG1 (max regression) → mrTRG5 (none)

PET/CT: early data disappointing; the Δ in SUV and metabolic tumor volume is the best predictor

SURVEILLANCE ON WW

Only for the pt who will actually comply – this is the premise of the whole strategy

DRE + endoscopy q3 months for 2 years

MRI q6 months for 2 years

Then q6 months for years 3–5, then YEARLY indefinitely

- regrowths reported as late as 7 years

Conditional survival: cCR sustained past 2 years → regrowth risk $\leq$ 10%

CT chest/abd/pelvis as for any CRC – mandatory in anyone who regrows

Colonoscopy per personal-history-of-CRC guidelines

LOCAL REGROWTH & SALVAGE

Actuarial regrowth at 2 yrs ~25%; approaching 30% at 3 yrs

- pooled 2-yr rate 15.7% (95% CI 11.8–20.1); series range 3–30%
- exponential curve – MOST occur in the FIRST YEAR

Vast majority have an endoluminal component → caught by DRE/scope

Small minority are mesorectal or lateral sidewall only – MRI is the only way to see these

The only proven risk factor is baseline cT stage:

- each increment adds ~10%: cT2 20%, cT3 30%, cT4 40%
- the effect is confined to the first 2 years

Salvage:

- ~90% of regrowths are resectable R0 (range 80–100%, avg 95.4%)
- salvage is usually PROCTECTOMY
- salvage proctectomy = same R0 rate, morbidity and oncologic outcome as proctectomy done at 8–12 wks for an iCR
- transanal LE as salvage ONLY for baseline cT2N0 – NOT for baseline stage II/III

Distant disease:

- cCR on WW: lower distant met rate than incomplete responders; no difference vs. pCR
- distant mets 0–16.7% (avg 6.8%)
- REGROWTH predicts distant disease – likely more aggressive biology
- unclear that up-front proctectomy would have prevented it

ONCOLOGIC & FUNCTIONAL OUTCOMES

DFS favors proctectomy (pooled HR 0.47, 95% CI 0.28–0.78)

- driven entirely by regrowth, which is salvageable 80–100% of the time

NO difference in overall survival

OPRA: stage II/III, TNT w/ induction vs. consolidation chemo, then WW or TME by response

- organ preservation higher w/ consolidation; DFS equal to historical TNT + TME

Habr-Gama (São Paulo): the original series – ~27% cCR after long-course CRT, entered WW

- 5-yr OS ~100%, DFS ~92%, endoluminal recurrences rare and salvageable

A randomized trial of WW vs. proctectomy in cCR will never happen – no pt with a cCR will consent to randomization

Function:

- WW beats proctectomy on QOL, defecation (Vaizey, LARS), urinary (IPSS) and sexual scores
- BUT a THIRD of WW pts still carry significant LARS symptoms – that is the radiation, not the operation
- WW also beats nCRT + local excision on continence scores and manometry

Organ preservation vs. local excision:

- full-thickness LE of the scar to “prove” a cCR is not required and costs function
- if a cCR is present, WW is the better organ-preserving option
- nCRT + LE is the compromise for the early distal tumor that does not achieve cCR

PREDICTING RESPONSE

MSI phenotype ($\leq$ 5% of rectal CA): more sensitive to nCRT in one large series – NOT confirmed in NCDB
K-ras, or K-ras/p53 mutations: more RESISTANT
Gene expression profiling has failed – intra- and intertumoral heterogeneity

DNA repair gene score: low score → poor response in 70–90%

Patient-derived organoids (“tumoroids”) – ex vivo response tracks clinical response

THE CONSENT CONVERSATION

Say the number: 1 in 4 will regrow, most in the first year

~90% of those are salvaged R0, almost always by the proctectomy you were trying to avoid

DFS is worse, OS is the same

Surveillance is q3 months for 2 years and never truly stops
– if he won’t come, don’t offer WW

Radiation alone still leaves a third of them with meaningful bowel dysfunction

If he has an early tumor, be explicit that the nCRT is being given ONLY to preserve the rectum

29 MALIGNANT DISEASE: PROCTECTOMY FOR RECTAL

CANCER

PRINCIPLES

Heald: TME – sharp dissection in the avascular areolar plane b/n the presacral fascia and the fascia propria of the rectum

- “the holy plane.” Done right it is SHARP and BLOODLESS
- violate the fascia propria → retained mesorectum → recurrence
- violate the presacral fascia → nerve injury, or presacral bleeding

Quirke: ink the radial margin, measure the CRM, grade the mesorectum

TME is a TECHNIQUE, not an operation – the operation is proctectomy

- radial margin positivity 25% → 7%; local recurrence now 4–8% worldwide
- CRM < 1 mm → local recurrence > 50%. CRM > 1 mm → 17%

The effects of nCRT and of good surgery are ADDITIVE, not compensatory

Priorities, in order:

1. adequate mesorectal excision by sharp dissection
2. nerve-sparing
3. continent reconstruction – but NEVER at the cost of a margin or of function

ANATOMY

IMA off the aorta → left colic + superior rectal 2–3 cm from its origin

- SRA to upper 1/3, middle rectal to mid (identifiable in only 10–20%), inferior rectal (off internal iliac) to lower 1/3
- upper rectum drains portal via IMV; the rest systemic via the iliacs

Presacral fascia covers the iliacs, hypogastric nerves and presacral veins

- presacral venous pressure can EXCEED IVC pressure

Denonvilliers' fascia – anterior, at the middle third. Stay POSTERIOR to it to spare the periprostatic/perivaginal nerves

- there is little or NO mesorectum anteriorly at the distal third

Waldeyer's fascia – posterior at ~S4, fusion of presacral fascia & fascia propria. MUST be divided to reach the levators

Autonomic nerves – where they are lost:

- superior hypogastric plexus: anterior aorta, from the IMA to the bifurcation → retrograde ejaculation
- splits at the promontory into R & L hypogastric nerves – sweep them POSTERIORLY at the start
- joined by the pelvic splanchnics (nervi erigentes) → inferior hypogastric (pelvic) plexus

- pelvic plexus is tethered to the mesorectum INFEROLATERALLY – separate it deliberately
 - sympathetic injury: retrograde ejaculation, bladder dysfunction. Parasympathetic: loss of erection
- Left ureter: medial to the gonadal vessels at the left common iliac bifurcation

PREOPERATIVE

Colonoscopy on all – synchronous polyps 13–62%, synchronous CA 2–8%

MBP + ORAL antibiotics: half the SSI, ↓ leak, ↓ ileus, ↓ readmission, no ↑ C. difficile

- enemas do NOT substitute – they won't clear the colon b/n a loop stoma and the anastomosis

Mark stoma sites; ETRN/CWOCN teaching preoperatively

Localize by proctoscopy in the OR – palpation is unreliable, especially after nCRT

THE OPERATION

MOBILIZATION

Lithotomy if a handsewn coloanal is planned; split-leg for a stapled anastomosis

Full splenic flexure takedown. High IMV ligation at the ligament of Treitz → up to 15 cm of extra reach

IMA at the aorta vs. superior rectal at its origin (~2 cm out, sparing left colic):

- NO oncologic difference – the choice is conduit reach vs. perfusion
- IMA flush: max mobility, conduit fed ONLY by the left branch of the middle colic
- SRA at origin: dual supply, less reach. Usually impossible for a distal tumor

Conduit must reach the base of the penis (M) or the mons pubis (F)

RECTAL DISSECTION

Score the peritoneum 1–2 cm above the promontory, just posterior to the arc of the SRA

Cautery or scissors in small brushing strokes. St. Mark's retractor, anterior traction toward the pubis

Divide Waldeyer's, then the lateral stalks, to the pelvic floor

Anteriorly stay behind Denonvilliers' unless the tumor is anterior

Tumor-specific mesorectal excision – PROXIMAL cancers:

- no oncologic benefit to mesorectum > 5 cm distal to the tumor
- preserves distal rectum → better function, lower leak than a coloanal
- divide the mesorectum PERPENDICULAR to the rectal axis. Do NOT cone in

Margins:

- proximal 5 cm. Distal 2 cm; 1 cm accepted for distal tumors, ≥ 5 mm after nCRT
- the RADIAL margin is the one that drives local recurrence

SPECIMEN GRADING (QUIRKE)

Ink the non-peritonealized radial margin – the SURGEON does this, before it leaves the room

Complete: intact glistening fascia propria, bulky smooth mesorectum, no defects

Nearly complete: moderate bulk, irregular, no defect down to the muscularis propria

Incomplete: little bulk, defects down to the muscularis propria

Grade + CRM both predict local recurrence. This is the surgeon's report card

RECONSTRUCTION

Straight stapled colorectal/coloanal – most common. Bring the spike out as close to the transection staple line as possible

Handsewn coloanal – for an ultralow anastomosis, or to salvage a stapler misfire

- orienting sutures on the conduit so it isn't handed down twisted
- single-layer absorbable, four quadrant sutures first

Colonic J-pouch:

- fold 7 cm, 75–80 mm linear stapler → 7–8 cm common channel
- must BOTH reach the anus and fit the pelvis – not for visceral obesity or a narrow male pelvis

End-to-side ("Baker"):

- 3 cm side limb. Function equal to a J-pouch at 1 yr, and it fits where a pouch will not

Transverse coloplasty:

- 8–10 cm longitudinal incision ending 5 cm from the end, closed transversely
- ABANDONED – higher leak, no functional advantage

Leak test every one: flexible scope + CO₂, pelvis filled with saline. Mucosa must be pink; clip bleeders

Diversion:

- loop ileostomy 25–30 cm proximal to the ileocecal valve for all low colorectal & coloanal anastomoses
- loop transverse colostomy if the colon is redundant – less dehydration than an ileostomy

MINIMALLY INVASIVE

Lap: medial-to-lateral, high IMA & IMV, 45 or 30 mm stapler – the pelvis rarely takes a 60 mm

- minimize fires → fewer crossing staple lines → lower leak

Robotic: single dock, ports 8 cm apart along a line from the R ASIS through the umbilicus to the L subcostal margin

ACOSOG Z6051 & ALaCaRT: both FAILED noninferiority on the composite pathologic endpoint (CRM, distal margin, mesorectal grade)

- but long-term survival is equivalent, as is the NCDB data

taTME:

- two teams; purse-string, then full-thickness circumferential transection starting in the intersphincteric plane, down-to-up
- meta-analysis: no difference in pathology or short-term outcomes vs. lap LAR
- international registry: MORE intra-op adverse events – bleeding, URETHRAL transection
- steep learning curve, unproven benefit

ABDOMINOPERINEAL RESECTION

Indication is tumor invading the EAS or the pelvic floor
INCONTINENCE ALONE IS NOT AN INDICATION – that pt gets a Hartmann or an intersphincteric proctectomy

Splenic flexure does not need to be mobilized

Abdomen to the pelvic floor, create the colostomy, close, then turn PRONE for the perineum

Bump under the pelvis, buttocks taped apart, anus sewn shut

Landmarks marked first: tip of the coccyx, ischial tuberosities, midpoint of the perineum

Enter the pelvis POSTERIORLY through the anococcygeal ligament onto the anterior coccyx – the safest entry
Then posterolaterally through the levators, tuberosity to tuberosity

Evert the specimen posteriorly. ANTERIOR dissection
LAST – urethra, prostate, vagina, specimen perforation

ELAPE / cylindrical APR:

- stop the abdominal dissection HIGH, finish from below with a wide levator excision
- avoids the "waisting" of the specimen at the pelvic floor
- national database, 954 pts: NO benefit over standard APR
- no reason to take pelvic floor contralateral to a non-circumferential tumor
- leaves a defect that usually needs a flap

Perineal reconstruction:

- wound complications up to 66% with primary closure – weight-bearing and irradiated
- gracilis, VRAM, anterolateral thigh, gluteal fasciocutaneous
- VRAM gives the most bulk but frequently costs an abdominal wall hernia
- omental pedicle flap fills the pelvis and keeps small bowel out of a boost field. Fiducials if a boost is planned

INTERSPHINCTERIC RESECTION

Takes part or all of the IAS to restore continuity for a very distal cancer

No agreed definition of how much sphincter is removed – the literature is therefore uninterpretable

Series mostly Japan & South Korea, results mixed

- ypT3 disease and PARTIAL EAS resection → suboptimal outcomes. Do not stretch the indication

LARS

< 4 cm of native rectum left → reservoir function is gone

Delphi consensus definition: 8 symptoms + 8

consequences, need ≥ 1 of EACH

- symptoms: unpredictable function, emptying difficulty, altered consistency, urgency, incontinence, frequency, repeated painful BMs, soiling

LARS score – 5 items (flatus & liquid incontinence,

clustering, frequency, urgency), 0–42

- 0–20 none, 21–29 MINOR, 30–42 MAJOR

Improves but PLATEAUS at ~2 yrs. It does not resolve

Rx ladder: 25–35 g fiber + 6–8 glasses non-caffeinated

fluid → biofeedback/pelvic floor PT → SNS (74%

success) → transanal irrigation, 5HT3 antagonists,

stoma

EXTENDED & MULTIVISCERAL RESECTION

~10% of primary rectal CA invades an adjacent organ =

T4b

Anterior: vagina, uterus, prostate, seminal vesicles, bladder. Lateral: ureters, iliacs. Posterior: coccyx, sacrum

Pelvic SIDEWALL involvement is usually unresectable – consider IORT

Women: en bloc uterus, adnexa, vagina. Men:

exenteration w/ cystoprostatectomy is cleaner than shaving the prostate

- colonic urinary conduit avoids a small bowel anastomosis

Beyond-TME planes (PRCbTME): lateral to the ureters, exposing iliacs, lateral nerves and lateral nodes

Neoadjuvant therapy first. R0 is everything

5-yr OS 53% for multivisceral resection of a PRIMARY, vs.

20% for recurrent disease

Survival after standard resection: Stage I 85-100%, Stage II 60-80%, Stage III 30-50%

OUTCOMES & SYSTEMS

Stage for stage, rectal CA survival now equals or exceeds colon CA (SEER, 372,130 pts)

US CRM positivity 17.2% – DOUBLE the European rate

Guideline-concordant chemoradiation: 78% at high-volume centers vs. 69% community

NAPRC: mandatory MRI staging of every new rectal cancer, mandatory multidisciplinary board

30 MALIGNANT DISEASE: COLORECTAL CANCER —

POSTOPERATIVE ADJUVANT THERAPY AND SURVEILLANCE

COLON CANCER – STAGE III

Stage at presentation remains the most significant prognostic factor

Surgery alone 5-yr OS 40–60%; with modern regimens 70–80%

ALL resected stage III should be considered for adjuvant chemo

Moertel (1990): 5-FU/levamisole x 12 mo – 41% ↓ recurrence, 33% ↓ mortality

IMPACT: 3-yr OS 62% → 71% w/ 5-FU/leucovorin, < 3% grade 4 toxicity

- LV beat levamisole, and 6 months = 12 months → 6 mo of 5-FU/LV became the standard

MOSAIC: FOLFOX4 vs. LV5FU2. Stage III 5-yr DFS 67% → 73%, 6-yr OS 69% → 73%

- oxaliplatin = inter- and intra-strand DNA crosslinks

CAPOX: 3-yr DFS 67% → 71% vs. 5-FU/LV.

Capecitabine alone ≥ infusional 5-FU/LV, fewer AEs

- CAPOX and FOLFOX are equivalent – different durations

DURATION – IDEA

~13,000 stage III pts, 3 vs. 6 months of FOLFOX or CAPOX, noninferiority design

Overall, 3 months FAILED noninferiority on 3-yr DFS

Low risk (T1–3, N1):

- 3 mo CAPOX IS noninferior to 6 mo
- 3 mo FOLFOX could NOT be proven noninferior
- NCCN: CAPOX x 3 mo, or FOLFOX x 3–6 mo

High risk (T4 and/or N2):

- 3 mo CAPOX not proven noninferior; 3 mo FOLFOX is INFERIOR
- NCCN: CAPOX x 3–6 mo (category 1 for 6), or FOLFOX x 6 mo

The entire point of the question: oxaliplatin neuropathy is cumulative and can be PERMANENT

COLON CANCER – STAGE II

Benefit is modest and inconsistent. Oxaliplatin adds NOTHING in stage II

IMPACT B2: 5-yr OS 82% vs. 80%, ns

Intergroup: DFS 76% vs. 72% ($p = 0.049$), OS 81% vs. 76%, ns

QUASAR: 22% ↓ recurrence, 18% ↓ death – but absolute 5-yr OS benefit only 3–4%

MOSAIC & NSABP C-07 stage II subgroups: no DFS or OS benefit from adding oxaliplatin

High-risk features (ASCO / ESMO) – prompt consideration of adjuvant chemo:

- T4
- poor differentiation
- lymphovascular invasion

- perineural invasion
- close or positive margin
- bowel obstruction
- < 12 lymph nodes harvested

MSI / MMR – the caveat:

- MSI-H / dMMR: better prognosis, fewer nodal and distant mets
- MSI-H is RESISTANT to 5-FU – do NOT give a single-agent fluoropyrimidine
- if you are going to treat, oxaliplatin is required
- BRAF V600E: worse OS, additive to MMR status
 - BRAF wt + dMMR 5-yr OS 89.7%; BRAF mut + pMMR 69.1%
- reflex MSI/MMR w/ BRAF testing on every CRC specimen

Oncotype DX (12-gene): stratifies recurrence RISK but does not predict RESPONSE → limited utility

RADIATION FOR COLON CANCER

Essentially no role – small bowel is in the field, and CRM positivity is uncommon in the colon

One RCT (chemoRT for T4/stage III): no OS or DFS difference, closed early, 54% significant toxicity

T4 w/ close or positive margin: NCDB + SEER, 28,000 pts – still no OS or DSS benefit

- if you suspect a close radial margin: CLIP the area, and consider an omental flap to keep small bowel out of the field

RECTAL CANCER – ADJUVANT

AFTER NEOADJUVANT RADIATION

NCCN, stage II/III mid & low rectal CA: neoadjuvant RT (short or long course) + systemic chemo, 6 months of peri-operative therapy in total

Base the adjuvant decision on CLINICAL stage, NOT the final ypStage – nCRT downstages

- not supported by definitive data, but that is the guideline

Who benefits is unsettled:

- 2 RCTs: modest DFS advantage for FOLFOX over 5-FU/LV
- meta-analysis ~1200 pts: NO difference in OS, DFS or distant recurrence

Only ~50% complete the full prescribed adjuvant course, and each 4-week delay costs ~14% survival

- this is the argument that moved chemo forward into TNT (ch 26)

WITHOUT NEOADJUVANT RADIATION

MRI specificity 75% for T, 71% for N → 20–25% are mis-staged and go straight to surgery

NCCN, no neoadjuvant therapy given:

- low-risk stage II (pT3N0, negative CRM): chemoRT then 5-FU/LV or capecitabine PREFERRED; observation acceptable

- high-risk stage II (pT4N0 or CRM+) or ANY stage III: chemoRT then FOLFOX or CAPOX

- 6 months total

POSTOPERATIVE radiation is the morbid option – AVOID it if you can

- small bowel adherent in the pelvis → radiation enteritis
- needs a HIGHER dose than preop, and postop tissue is hypoxic → less tumoricidal
- irradiates the conduit and sphincter: stricture, poor function, perforation, fistula
- reasonable alternative for node-positive disease w/ a NEGATIVE CRM: chemotherapy ALONE

AFTER LOCAL EXCISION

High-risk features on the LE specimen:

- poor differentiation, LVI, R1 resection, fragmentation of the specimen, margin positive or < 1 mm
- any of these → PROCTECTOMY if he will tolerate it

LE alone: local recurrence 7–23% vs. 0–9% for proctectomy; 5-yr OS 70–92% vs. 80–96%

ACOSOG Z6041 – cT2N0, chemoRT then LE: 3-yr DFS 87%, below stage-matched proctectomy

- an oncologic COMPROMISE, for the pt who cannot or will not have a proctectomy

TARGETED AGENTS

NO role in the adjuvant setting after curative resection

AVANT (bevacizumab): no DFS difference, more grade 3–5 AEs

PETACC-8 & N0147 (cetuximab, KRAS wt): no DFS difference, more toxicity, fewer completed 12 cycles

SURVEILLANCE

Only for the pt willing and able to be treated for what you find

- otherwise it is economic and emotional burden, ordered to satisfy the physician's curiosity

80% of recurrences appear within 2–3 yrs → front-load intensity, taper, stop at 5 yrs

THE SCHEDULE

H&P q3–6 months x 2 yrs, then q6 months to 5 yrs

- abdomen, wound, DRE, and the perineal wound after APR

CEA q3–6 months x 5 yrs

CT chest/abd/pelvis q6–12 months x 5 yrs (ASCRS: annually x 5)

Colonoscopy at 1 yr; if negative repeat at 3 yrs, then q5 yrs

- adenoma found → back to yearly
- reasonable to defer the first scope to 2–3 yrs IF a good-quality preop scope and no Lynch

Rectal after local excision: ERUS or MRI q3–6 months x 2 yrs, then q6 months to 5 yrs

Do NOT use LFTs, hemoccult or serum hemoglobin for surveillance

CEA

Detects 30–60% of recurrences. Independent predictor of recurrence

- > 15% will have a FALSE elevation with no recurrence
- 1/3 of primary CRCs do not produce CEA at all
- identifies disease w/ normal conventional imaging in up to 23%
- don't check while on 5-FU – falsely elevated

Rising CEA – the algorithm:

- confirm the elevation
- physical exam, colonoscopy, CT chest/abd/pelvis
- all negative → PET/CT
- still negative → repeat CEA + imaging q3 months until disease appears or the CEA falls

COLONOSCOPY

The purpose is METACHRONOUS neoplasia, not luminal recurrence

Anastomotic recurrence is < 4% of all recurrences

- if you can see it in the lumen it grew in from outside – usually unresectable

Metachronous CA within 2 yrs 0.7%; second primary at 5–10 yrs 2–3%

First surveillance scope: neoplastic polyps in 18%, but only 3% are > 1 cm

Stop scoping the pt with limited life expectancy

DOES INTENSITY MATTER?

FACS (2014, 1202 pts): minimal vs. CEA vs. CT vs. both. More curative salvage ops (7–8% vs. 2%) but NO survival difference (82% vs. 84%). Adding CT to CEA added nothing

GILDA (2016, 1228 pts): intensive found recurrence 6 months EARLIER – no DFS, no OS, no QOL difference

COLOFOL (2018, 2509 pts): CT + CEA q6 mo vs. at 12 & 36 mo. Recurrence 22% vs. 19%, disease-specific mortality 11% vs. 11%, OS mortality 13% vs. 14%

NCDB/Alliance (8529 pts): testing intensity NOT associated w/ recurrence, resection of recurrence, or OS

2019 meta-analysis, 13,216 pts: no OS, cancer-specific or relapse-free survival benefit to intensive follow-up

Bottom line: intensive surveillance finds recurrence

- EARLIER, but the pt does not live longer
- the older positive meta-analyses were lead-time bias and stage migration

SPECIAL SITUATIONS & REALITY

Stage I colon: excluded from the trials. Colonoscopy ONLY – no routine CEA, no routine imaging

Stage I rectal after TRANSANAL excision: local recurrence 4–33% – LOCAL surveillance takes precedence

- ERUS in expert hands outperforms DRE, endoscopy, CT and CEA – catches ~30% the others miss
- ERUS-guided biopsy confirms extraluminal recurrence
- extend beyond 5 yrs – local recurrence seen past 7 yrs

Compliance is dismal: 14% complete recommended CT,
33% colonoscopy, 24% CEA; only 50% get all three

- only 30% of ASCRS members follow a formal national guideline
- surgeons cost more than GPs, with no difference in recurrence, survival or QOL

Cost ~\$5400/pt over 5 yrs – but > \$100,000 per curable recurrence actually detected

31 MALIGNANT DISEASE: COLORECTAL CANCER —

MANAGEMENT OF DISTANT METASTASES

OVERVIEW

Most CRC deaths are 2/2 metastatic dz – 22% metastatic at presentation

5-yr OS Stage IV: 10-14%

Sites: liver 75%, lung 22% – then brain, bone, ovary, peritoneum, adrenal

- > 70% of pts w/ lung mets have synchronous liver mets
 - solitary met 3-yr OS: liver 26.8%, lung 34.0% (SEER)
 - multi-organ oligometas do WORSE than single-organ
- NCCN & ASCRS endorse NO single strategy – individualize by MDT

THE PRIMARY TUMOR

Bleeding:

- usually low volume → manage the anemia non-operatively
- XRT palliates surface bleeding well, esp. rectum
- resection PREFERRED for proximal tumors or true hemorrhage
- endovascular embolization or covered stent for palliation

Obstruction:

- 8-30% present partially or completely obstructed
- proximal stool column w/ no distal stool on CT = impending obstruction
- ASCRS: stent before colectomy or diversion if obstructing colon CA w/ incurable mets
 - ↓ short-term morbidity, ↓ interval to chemo, NO effect on OS
 - median stent patency 106 days (68-288) – erosion, abscess, fistula
 - distal rectal tumors NOT stentable – stent sits on the anorectal ring
- resection if easily resectable and expected to live ≥ 6 months
- LOW threshold for diversion – a leak delays or omits adjuvant chemo

Diversion – pick the stoma for the NEXT operation:

- always vent the distal limb – loop or divided end-loop – or closed-loop perforation
- mid/proximal sigmoid PREFERRED, or transverse just right of the middle colic
- distal transverse/descending colostomy injures the marginal artery → devascularizes the distal colon once the IMA is taken high at LAR
- loop ileostomy least disruptive BUT closed loop if competent IC valve + complete distal obstruction; dehydration w/ chemo enteritis

Perforation:

- source control FIRST, oncologic resection only if feasible
- AVOID en bloc resection of vital structures – prognosis too poor

UNRESECTABLE SYNCHRONOUS METS

Control of the DISTANT dz drives survival – prioritize it
Chemo first if the primary is minimally symptomatic

- < 20% ever need palliative intervention on the primary while on chemo
 - prophylactic resection of an asymptomatic primary: 84% (2000) → 52% (2011)
 - retrospective: resection + chemo adds 6-14 mo DFS/OS – heavy selection bias
 - JCOG1007, CAIRO4, GRECCAR 8 pending
- Surgery ONLY for impending obstruction, significant bleeding, perforation
- Reimage q2 months if conversion to resectability is a possibility

SYSTEMIC THERAPY

Regimens: FOLFOX, FOLFIRI, CAPEOX, FOLFOXIRI ± bevacizumab

- FOLFOX or CAPOX PREFERRED over capecitabine or 5-FU/LV alone if mets resectable

Biomarkers – MUST have before choosing a biologic:

- anti-EGFR (cetuximab, panitumumab) ONLY if RAS/BRAF wild-type AND LEFT-SIDED
- any KRAS/NRAS mutation → no benefit. Right-sided primary → no benefit
- BRAF V600E: poor prognosis – encorafenib + cetuximab
- MSI-high / dMMR: checkpoint blockade (pembrolizumab, nivolumab ± ipilimumab)

Timing rules:

- 4 weeks off FOLFOX/CAPOX before surgery
- 6 weeks off bevacizumab before elective surgery, restart 6-8 weeks post-op
- if clearly resectable, LIMIT neoadjuvant to 2-3 months, give the other 3 post-op

Chemo alone: 5-yr OS 20%, median OS 23 mo

RESECTABLE SYNCHRONOUS METS

~20% of pts w/ CRC mets are resectable or potentially resectable

Three NCCN sequences – all acceptable, none proven superior:

- (1) synchronous or staged colectomy + metastasectomy → 6 mo adjuvant
 - the healthy pt w/ a small solitary peripheral liver or lung met
- (2) neoadjuvant chemo 2-3 mo → colectomy + metastasectomy → 3-4 mo adjuvant
 - > 4 liver mets w/ an asymptomatic primary
- (3) colectomy → chemo 2-3 mo → staged metastasectomy → 3-4 mo adjuvant
 - impending obstruction w/ a large borderline-resectable met

Resection first – arguments:

- no shedding from the primary during systemic Rx, no emergent surgery on progression
- avoids chemo liver injury – steatohepatitis, sinusoidal obstruction syndrome, noncirrhotic portal HTN

Chemo first – arguments:

- treats micromets, cytoreduces for R0, converts unresectable dz
- declares tumor biology – AVOIDS futile surgery in progressors
- avoids a surgical complication delaying chemo

Rectal primary w/ synchronous mets:

- SHORT COURSE XRT attractive – gets to multi-drug chemo far sooner
- XRT first allows accurate assessment of primary response
- watch-and-wait NOT validated in Stage IV – every W&W series excluded them

METACHRONOUS METS

20% develop mets after curative treatment – > 85% within 3 years

- liver 60%, lung 39%, extra-regional nodes 22%, peritoneum 19%

Predictors: male (HR 1.2), T4 (HR 1.6), N1 (HR 2.8), N2 (HR 4.5), high grade (HR 1.4), + margin (HR 2.1)

Prognosis BETTER than synchronous dz

LIVER METASTASES

RESECTION

Highest cure rate of any modality

5-yr OS 24-58% (avg 40%), 30-day mortality < 3%, morbidity < 15%

- vs. 10-11% 5-yr OS w/ systemic chemo alone

20% resectable at presentation, another 10% convert after induction chemo

Modern definition of resectable:

- fit for surgery + R0 achievable + enough viable parenchyma to sustain life
- the OLD criteria (< 4 lesions, small, no extrahepatic dz) are gone
- bilobar/multiple mets and extrahepatic dz (esp. lung) no longer exclude
- close or difficult lesions → ablation, or en bloc vascular resection w/ reconstruction

Future Liver Remnant (FLR):

- FLR > 30% of total hepatic volume is the MINIMUM if chemo given > 12 weeks
- 3D CT reconstruction is the standard measurement

Portal Vein Embolization / Ligation:

- expands functional volume 39% over 4-8 weeks
- STANDARD OF CARE for resectable liver mets w/ FLR < 30%
- 25-38% progress and never reach the staged hepatectomy

Two-Stage Hepatectomy:

- stage 1 clears the FLR of dz + portal vein ligation – can include the primary
- stage 2 formal anatomic resection of the diseased lobe once hypertrophied

ALPPS:

- portal vein occlusion + in-situ partition at the future transection line
- much faster regrowth – volumetrics at 1-2 wks, stage 2 as early as 7-10 days
- *LIGRO RCT* vs. *two-stage*: resection rate 92 vs. 80%, median OS 46 vs. 26 mo ($p = 0.028$), no difference in complications or 90-day mortality
- still NOT standard – “the hepatobiliary controversy of the decade”

OSLO-COMET RCT, lap vs. open: LOS 2 vs. 4 d, complications 19 vs. 31%, better QOL, OS identical (80 vs. 81 mo). Use lap where anatomy allows

ABLATION & LOCOREGIONAL

Resection PREFERRED over every locoregional therapy – better DFS and OS

~80% of pts have unresectable dz

Radiofrequency Ablation (RFA):

- 375-500 kHz alternating current, tissue to 100 °C → coagulation necrosis
- BEST for peripheral lesions < 3 cm
- heat sink – ineffective near large vessels; desiccation limits the zone
- OS 28-53 mo, 5-yr OS 43%. Morbidity < 2%, mortality < 1%

Microwave Ablation (MWA):

- 900-2450 MHz, molecular friction. NO heat sink effect
- handles lesions > 3 cm and central/perivascular lesions – now favored over RFA
- 5-yr OS 55% vs. 43% RFA vs. 20% chemo alone

TACE:

- mitomycin/doxorubicin/cisplatin + lipiodol, then an embolic agent
- SALVAGE only – unresectable, not ablatable, failed systemic chemo
- median OS 8-9 mo. Post-embolization syndrome 60-80%
- DEBIRI (irinotecan-eluting beads): OS 16 mo, toxicity 10%
- *Martin RCT*: FOLFOX-DEBIRI downsized to resection 35% vs. 6% w/ FOLFOX alone, no PFS difference

TARE / SIRT (yttrium-90):

- response 35-40%, median OS 8-14 mo
- *FOXFIRE + SIRFLOX + FOXFIRE-Global* ($n = 1103$): MORE toxicity, NO OS benefit added to FOLFOX

SBRT:

- 25 Gy x 3 consecutive daily fractions = 75 Gy, 4D CT planning
- median OS 24-27.6 mo, 5-yr 18%. 20% hepatic toxicity
- no clear advantage over modern chemo (OS 23.3 mo)

Hepatic Artery Infusion (floxuridine pump):

- mets derive their supply from the hepatic artery – near-complete first-pass extraction
- adjunct to systemic chemo at high-volume centers, for conversion or adjuvant
- biliary sclerosis is the dose-limiting toxicity

LUNG METASTASES

2nd most common site – 22% of CRC mets

Isolated lung mets prognostically BETTER than liver – 5-yr OS up to 68%

NO prospective trial of pulmonary metastasectomy has ever been done (PulMiCC pending)

STS Consensus + NCCN:

- goal = complete resection w/ preservation of functional lung
- wedge metastasectomy over lobectomy. AVOID pneumonectomy
- NO number cutoff – any count acceptable if R0 achievable
- re-resection appropriate in selected pts
- minimally invasive PREFERRED – localization technique doesn't matter
- consider mediastinal LN sampling – involved in up to 44%, worse survival

Morbidity 11%, mortality 1.1%, LOS 4-7 days

Ablation and SBRT are reasonable alternatives – NCCN less firm about resection here than for liver

OTHER SITES

OVARIAN

5-10% of women w/ CRC, skewed younger. Median survival 19-27 mo

Disproportionately CHEMO-RESISTANT, grow large before becoming symptomatic

Spread is HEMATOGENOUS – a/w vascular invasion, smooth capsule, no surface deposits

IHC – met CRC vs. ovarian primary:

- CRC: CK20+, CEA+, CDX2+, CK7–, CA125–
- Ovarian: CK7+, CA125+, CK20–, CEA–, CDX2–

True Krukenberg = > 10% signet-ring cells – usually a GASTRIC primary

Oophorectomy if grossly abnormal or direct extension into the capsule

- prophylactic oophorectomy of normal ovaries NO LONGER recommended (NCCN)
- reasonable to reoperate for metachronous ovarian mets

BRAIN

1-4%, nearly always w/ widespread dz. Do NOT image the brain w/o neuro signs

Surgery for an isolated met – but 50-60% local recurrence within 1 yr

SRS has replaced WBRT adjuvantly, and for unresectable solitary or few lesions < 3 cm

WBRT only if multiple and not an SRS/surgery candidate – NO OS benefit, fatigue/alopecia/neurocognitive decline

PERITONEAL

19% of metachronous mets. Quantify burden by Sugarbaker PCI (13 regions)

REFER limited peritoneal burden to a peritoneal surface malignancy center – CRS/HIPEC in Ch 35

ADRENAL

Uncommon, late in the course. Surgery rarely an option

SURVEILLANCE – STAGE IV TREATED w/ CURATIVE INTENT

Same as Stage II/III but MORE frequent imaging

- H&P q3-6 mo x 2 yrs, then q6 mo to 5 yrs
- CEA q3-6 mo x 2 yrs, then q6 mo to 5 yrs
- CT C/A/P q3-6 mo x 2 yrs, then q6-12 mo to 5 yrs
- colonoscopy at 1 year

32 MALIGNANT DISEASE: LOCALLY RECURRENT

RECTAL CANCER

OVERVIEW

LRRC is an EXTRA-TME pathology – a fundamentally different operation than the T4 advanced primary
Local recurrence still ~10% in unselected series despite TME + neoadjuvant

Exenteration is the ONLY chance of cure – 5-yr OS 40-50% IF margins are clear

- comparable to resected hepatic or pulmonary mets
Untreated → malignant fistula, obstruction, neuropathic pain from nerve/muscle/bone infiltration

Palliative exenteration has NEVER been shown to return QOL to baseline – offer VERY selectively

Expect taTME, TAMIS, TEM and watch-and-wait to ↑ the need for radical salvage

R0 rates at specialist centers have climbed from < 50% (mid-2000s) to ~70%

COMPARTMENTS & PATTERNS OF RECURRENCE

Recurrence sits OUTSIDE the mesorectal plane – pelvic floor muscle, presacral fascia, lateral compartment (iliac vessels, piriformis), sacrospinous & sacrotuberous ligaments

Five compartments: central/axial, anterior, posterior, R lateral, L lateral

- each subdivided supra- vs. infralevator by the pelvic floor
- older fixity classifications (F0/FR/FNR) do NOT work well for LRRC – think in compartments

Landmarks:

- ischial spine = center of the LATERAL compartment
- urethral orifice at the pelvic floor = center of the ANTERIOR compartment
- posterior recurrence arises from S3 down + sacrospinous lig. out to the ischial spine, abutting piriformis laterally and obturator internus anteriorly

Central / axial – historically 50%:

- peri-anastomotic or off the rectal scar after APR. TRUE isolated anastomotic recurrence is uncommon
- involves vagina/uterus or prostate/seminal vesicles ± bladder → total “soft tissue” exenteration

Anterior: pubic rami/symphysis resection + perineal urethrectomy for a clear margin

Posterior: presacral fascia, sacrum, sacral roots → en bloc sacrectomy

Lateral: iliac vessels, lumbosacral trunk, sciatic nerve, sidewall muscle & ligament

Contemporary series of > 200 exenterations: total cystectomy 49%, sacrectomy 58%, lateral compartment 73%, major lumbosacral or complete sciatic nerve excision 38%

WORKUP

Detection is HARDER than expected – often no luminal component, symptoms nonspecific

Biopsy the radiographic abnormality – but often unsafe (path crosses vessel or bowel)

- radiologic change + FDG avidity is sufficient evidence to proceed

MRI + PET-CT = the mainstay:

- MRI PPV 100%, NPV 89% for LRRC. It defines the operative plan
- MRI accuracy DROPS for the small sidewall structures
- predicted HIGH SACRAL or LATERAL involvement on MRI predicts an involved margin
- plan the BIGGER resection – never “shave” an involved structure
- PET finds mets CT misses, and separates post-XRT fibrosis from tumor

Scope for a synchronous lesion. CT C/A/P for extrapelvic dz

Rising CEA w/ no radiologic dz → seek histologic proof.

Do NOT explore the pelvis to diagnose

MDT:

- colorectal, urology, orthopedics, vascular, plastics, med & rad onc, radiology, pain, palliative care, stoma nurse, psycho-oncology
- determines resectability ~70:30 in referred pts, independent of when the surgeon sees them
- 1 in 5 pts deemed suitable will still choose palliation
- Sweden: < 1/3 of LRRC pts nationally were discussed at MDT before exenteration

RESECTABILITY

Indication = a PREDICTED R0 w/ acceptable morbidity in a consented, motivated patient

NO LONGER a contraindication:

- lateral compartment / internal iliac involvement – R0 69% now vs. 21% in 2008
- common & external iliac artery and vein – R0 38-58%
- sciatic nerve & lumbosacral trunk – R0 equal to central tumors, function better than expected
- HIGH sacrectomy above S2/S3 – R0, cancer-specific and overall survival all preserved
- but ↑ blood loss, ↑ complications, more sacral neurologic deficit. Consent for it

Still a contraindication:

- UNRESECTABLE distant metastatic disease
- an R0 that cannot be predicted; the unfit or unmotivated patient

Metastatic dz – present in up to 40% at diagnosis of LRRC:

- resectable isolated mets → STAGE them, do NOT do synchronous metastasectomy
- liver/lung FIRST, then the exenteration – pelvic recovery is far longer
- delay to metastasectomy ↑ cancer mortality

NEOADJUVANT & INTRAOPERATIVE RADIATION

Long-course chemoXRT for ALL who were not irradiated for their primary

Re-irradiation ~2000 cGy w/ 5-FU – contentious; morbidity vs. local control unsettled

- reimaging at 4 weeks, operate if no progression

IOERT / intraoperative brachytherapy – for close margins

or R1/R2:

- 1000 cGy: minimal residual dz
- 1500 cGy: gross residual < 2 cm
- 2000 cGy: unresectable or gross dz > 2 cm

NO data on adjuvant chemo after LRRC resection – reasonable to give, systemic failure dominates

OPERATIVE PRINCIPLES

Three phases: DISSECTION (adhesiolysis, ureterolysis, proximal & distal control) → RESECTION → RECONSTRUCTION

Operate in VIRGIN planes – circumnavigate the involved compartment, never re-enter the old TME plane

Gain proximal & distal control of organs, vessels and nerves BEFORE the final resection

If the tumor abuts an organ, vessel, nerve or bone – RESECT IT. A “close shave” = R1

“6 C philosophy”: Coherent plan, Clear exposure, Control (proximal & distal), Circumnavigate the tumor, Constantly reassess, and stay Calm

Preparation:

- 2 consultant colorectal surgeons. Urologist in 50% (conduit), another 5-10% for ureteric reimplant
- orthopedics, plastics, vascular arranged in ADVANCE
- modified Lloyd-Davies on a nonslip gel mattress, distal end rolled back 10 cm to lift the pelvis off the table if abdomino-lithotomy sacrectomy is planned
- suture the anus closed, occlude existing stomas
- mark the VRAM harvest AND the post-VRAM stoma sites
- prep the legs to the knees if a vein graft may be needed
- meticulous adhesiolysis – an enterotomy becomes an enteric fistula
- infant feeding catheters up the cut ureters – monitors urine, prevents back pressure & ATN

Central recurrence:

- posterior first, above the neorectum off presacral fascia, then anterolateral in the EXTRA-TME plane
- divide vas deferens / round ligament; ureter is immediately deep to peritoneum – retract medially
- ligate the ANTERIOR division branches of the internal iliac (uterine, sup. & inf. vesical) → GU organs swing off the sidewall and expose the obturator fossa
- take endopelvic fascia and levator ABDOMINALLY under direct vision – easier than from the perineum

- perineal urethrectomy at the base of the penis – wider membranous urethral margin, and avoids the dorsal venous complex in the irradiated cave of Retzius
- female: INTERLABIAL radical vaginectomy w/ cystectomy – wider margin, less venous bleeding, preserves labia majora for primary closure w/o a flap
- perineal surgeon works to the BONY landmarks (coccyx, ischial tuberosities, inferior pubic rami) by MRI plan, NOT by the abdominal surgeon's direction

Lateral neurovascular resection:

- start LATERAL to the common/external iliac on the psoas – a virgin plane, as for a lateral node dissection
- ligate the internal iliac close to its origin → exposes piriformis, lumbosacral trunk, sacral roots
- ligate the lateral branches (gluteal, ascending lumbar) – they retract into the sidewall and bleed
- ischial spine taken w/ osteotome or Gigli saw; sciatic nerve exits posterolateral to it
- femoral nerve found above the inguinal ligament lateral to the artery, or between the two heads of psoas at L4-L5
- 96% w/ COMPLETE sciatic nerve resection still mobilize – ankle orthotic + “rolling gait.” QOL back to baseline by 12 months
- common/external iliac VEIN: LIGATE, do NOT graft – venous patency is poor (thrombosis, PE), collaterals limit leg swelling
- arterial grafts DO stay patent – autologous vein PREFERRED over synthetic (graft infection)

Posterior / sacrectomy:

- circumnavigate first – leave the tumor attached ONLY at the sacrum
- divide the sacrospinous ligament off the spine to free the sacrum
- BELOW S3 → abdomino-lithotomy sacrectomy. No prone phase, keeps vascular control and better sciatic access
- AT or ABOVE S2/S3 → prone jackknife w/ spinal surgeon: fluoroscopy, laminectomy, transverse osteotomy, divide sacrospinous & sacrotuberous ligaments
- vessel-loop L5 and S1 abdominally beforehand – they are very hard to identify prone
- anterior cortical sacrectomy: partial-thickness osteotomies around a high central lesion between the L5/S1/S2 foramina – PRESERVES the sacral roots

RECONSTRUCTION

Ureteric reimplant ± Boari flap after partial cystectomy

Four urinary options after total cystectomy:

- isolated ileal conduit
- isolated colonic conduit – NO enteric anastomosis, so no enteric leak
- incontinuity colostomy + conduit (“wet colostomy”)

- separated colonic colostomy + conduit w/ a communal exit

VRAM flap for the perineum, and for the posterior vaginal wall if the bladder is preserved

Mesh for soft tissue and bony defects

Vascular reconstruction IMMEDIATELY after transection – limb ischemia and compartment syndrome w/ legs up

Post-op:

- ICU. TPN from POD 0 – malnutrition and prolonged ileus are the rule
- limb checks q4h x 3 days after arterial reconstruction, compression stockings
- drain fluid creatinine on days 1 and 5 to catch a urine leak
- ureteric catheters out days 10-14

OUTCOMES

PelvEx Collaborative (n = 1184): LOS 15 days, major complications 32%, reintervention 10%

R0 55% (PelvEx), 58% (systematic review)

5-yr OS 40-50% IF R0 – THE MARGIN IS THE DRIVER

Koh et al: a microscopically clear margin predicts lower local recurrence and higher OS – but a margin > 0.1 mm confers NO additional benefit. Challenges the 1 mm R0 definition borrowed from primary rectal CA

55% will recur after radical salvage surgery

- 14-21% locally re-recurrent – further radical surgery can still be feasible
- the MAJORITY recur systemically – this remains the dominant failure mode

33 MALIGNANT DISEASE: APPENDICEAL NEOPLASMS

OVERVIEW

Rare – most present as appendicitis and are found
INCIDENTALLY on the path report

SEER 1973-2001: signet ring adenoCA 0.15 up to
mucinous tumors 1.3 per 1,000,000/yr

29% of pts > 65 w/ an appendiceal CA or NET were first
misdiagnosed as appendicitis

aNEN = 43-57% of primary malignant appendiceal tumors

Differential: mucinous neoplasm, adenoCA subtypes,
melanoma, leiomyoma, lipoma, fibroma, neuroma,
lymphangioma, met dz (esp. ovarian), cecal CA at the
appendiceal base

- non-neoplastic: Crohn's, neutropenic enterocolitis, TB, actinomycosis, mesenteric cystic dz, NSAID injury, and appendicitis itself

AVOID preoperative biopsy – often nondiagnostic, risks
perforation, mucin extravasation, ↑ recurrence

- APPENDECTOMY is the diagnostic test, and often the definitive one

TERMINOLOGY – THE PSOGI RECLASSIFICATION

ADENOMA is NO LONGER used for mucinous lesions – it
now designates only the rare appendiceal lesion w/
colorectal-type histology

*Simple mucocele: a distended mucin-filled appendix – NO
hyperproliferation, NO neoplasia*

LAMN / HAMN: dysplastic, but NO infiltrative growth.

Separated by degree of epithelial dysplasia

- HAMN staged as invasive adenoCA for its higher recurrence risk – evidence for that is thin
- HAMN is NOT associated w/ distant metastatic potential

*Mucinous adenocarcinoma: infiltration and invasion are
present*

Goblet cell tumors – formerly “goblet cell carcinoid” /
“adenocarcinoid,” classified as NET

- NOW goblet cell ADENOCARCINOMA, an
EPITHELIAL tumor

MUCINOUS NEOPLASMS – LAMN & HAMN

Usually incidental. Colonoscopy: “volcano sign” – raised
circumferential lesion w/ central umbilication

CT: thickened or irregular appendiceal wall w/ calcification.
Peritoneal mucin or masses = advanced dz

US: layered “onion skin sign” – called pathognomonic,
rarely used in the US

Larger = more likely malignant, but NO definitive size
cutoff – imaging cannot reliably call it

Scope preop: cecal extension (appendectomy/cecectomy
vs. right colectomy) + synchronous neoplasia

Frozen section is UNRELIABLE for mucinous tumors –
wait for permanents to dictate management

Treatment:

- LAMN or HAMN confined to the appendix,
unperforated, MARGINS CLEAR → appendectomy
ALONE
- take a cuff of cecum in continuity if the lesion sits near
the base
- mesentery, mesocolon or bowel involvement → radical
resection
- questionable whether completion right hemi is needed
even for a positive appendectomy margin
- right hemicolectomy does NOT reduce the risk of PMP

Mucin on the serosal surface (T4a) – the risk factor for peritoneal recurrence:

- what matters is TUMOR CELLS outside the appendix,
not the mucin itself
- ACCELLULAR mucin: peritoneal recurrence 4-7%
- CELLULAR mucin: peritoneal recurrence 33-75%
- surveillance CT q3-6 mo x 2 yrs, then yearly to 5 yrs
- diagnostic laparoscopy at 12 months is an alternative
to imaging alone

Prognosis:

- LAMN confined to the appendix: excellent
- spread beyond the appendix: 5-yr survival still 79-86%
- acellular mucin on the surface: recurrence ~5-20%

ADENOCARCINOMA

Subtypes: mucinous, non-mucinous (intestinal), signet ring

- signet ring WORST, mucinous & intestinal
intermediate, goblet cell most favorable
- recent SEER: OS and cancer-specific survival
COMPARABLE for mucinous vs. non-mucinous

Mucinous type invades locally and perforates → mucin into
the peritoneum → PMP

Prognosis driven by nodal mets, distant mets, and
differentiation

- tumor perforation is NOT an obvious adverse
prognosticator

Staged as for colorectal CA. CT C/A/P, colonoscopy, CEA

- CA-125 and CA 19-9 only if the primary site is unclear

Surgery:

- confined to the SUBMUCOSA – well-differentiated T1
→ appendectomy alone
- everything else → RIGHT HEMICOLECTOMY
- controversial: some series show comparable survival
after appendectomy alone, and nodal mets are
relatively rare in the mucinous type

Perforated appendiceal tumor w/ free mucin – 4 steps:

- (1) excise the perforated lesion – appendectomy or
right hemicolectomy
- (2) peritoneal washings for cytology
- (3) biopsy any suspicious peritoneal lesion
- (4) DELAY formal cytoreduction – refer to a specialized
surgeon/center
- prophylactic oophorectomy is NOT helpful

Adjuvant chemo – modeled on colon CA, FOLFOX or CAPOX:

- ALL Stage III
 - selected Stage II: T4, poor differentiation, perforation, or < 12 nodes examined
 - Stage IV: add irinotecan and bevacizumab
- Surveillance mirrors resected colon adenoCA. CRS ± HIPEC for carcinomatosis – see Ch 35

NEUROENDOCRINE TUMORS (ANEN)

3rd most common GI NET site – after small bowel and rectum

Found in 0.3-2.3% of appendectomies. In children, the 2nd most common bowel tumor

Female predominance. Unusually EARLY onset – peak 15-19 yrs (F), 20-29 yrs (M)

- median age 42 if non-metastatic, 31 if > 2 cm, 29 if metastatic

Presents as appendicitis in > 54%, obstructive features in 25%

- most sit at the TIP, so true luminal obstruction is uncommon

Carcinoid syndrome is EXTREMELY RARE – hormones degraded by the liver, needs liver mets

Workup:

- CT/MR miss them until large. Luminal contrast is unhelpful for the appendix
- somatostatin receptor imaging and In-111 octreotide UNRELIABLE for aNEN < 1 cm
- Ga-DOTATOC PET has better spatial resolution, unproven for preop localization
- Chromogranin A NOT a screening test – not elevated until > 2 cm
 - but TREND it after treatment – catches recurrence before imaging does
- 5-HIAA only if carcinoid syndrome is suspected

Risk features – these drive the operation:

- SIZE ≥ 2 cm – the single most valuable predictor of nodal mets
- > 95% are < 2 cm; 60-85% are < 1 cm; only 2-17% are > 2 cm
- < 1 cm: risk of nodal mets is VERY LOW
- LOCATION AT THE BASE (7-10%) – best predictor of recurrence, from an incomplete margin
 - 33% of base tumors have mets
- mesoappendiceal invasion – strong predictor of worse prognosis, independent of size
- Ki-67 > 2%, or high mitotic index
- angio- or neuro-invasion – 60% w/ vascular invasion have nodal mets
- involved surgical margin

Operation:

- < 2 cm, at the tip, NO risk feature → APPENDECTOMY ALONE. No staging, no surveillance
- ≥ 2 cm → RIGHT HEMICOLECTOMY

- 1-2 cm PLUS any one risk feature → right hemicolectomy
- up to 3 cm w/ serosal or mesoappendiceal invasion → right hemicolectomy
- incompletely resected base tumor → right hemicolectomy
- resectable distant mets → still consider right hemi + metastasectomy
- inspect and palpate the rest of the small bowel – synchronous NETs

Advanced or unresectable: somatostatin analogues improve PFS

Surveillance: long-term f/u for nodal, resected locoregional or distant mets. Yearly CgA ± MRI

- outcomes differ LITTLE between appendectomy and right hemi – these tumors are indolent

OTHER NEOPLASMS

Goblet Cell Adenocarcinoma (GCA):

- features of BOTH neuroendocrine and adenoCA – its own natural history, prognosis, treatment
- composed of goblet, endocrine and Paneth cells
- can coexist w/ high-grade adenoCA – the HIGH-GRADE component dictates Rx and prognosis

Clear cell carcinoid:

- exceedingly rare, case reports only. Lipid-laden, resembles GCA
- may in fact be an appendiceal MET from a non-appendiceal clear cell CA

Pseudomyxoma Peritonei (PMP):

- perforated mucinous neoplasm seeding the peritoneum with mucin
- REFER any pt w/ peritoneal mucin deposits to a peritoneal surface malignancy center
- CRS ± HIPEC, and “preemptive” CRS/HIPEC for advanced-stage neoplasms – Ch 35

34 MALIGNANT DISEASE: GIST, NEUROENDOCRINE

TUMORS, AND LYMPHOMA

GASTROINTESTINAL STROMAL TUMORS

ORIGIN & HISTOLOGY

Arise from the interstitial cells of Cajal – pacemakers of the gut, within the muscularis propria

20% of all soft tissue sarcomas, ~10 per million/yr. ALL carry malignant potential, 10-30% progress

Spindle 70%, epithelioid 20%, mixed 10% – misread as leiomyoma / leiomyosarcoma before IHC

Immunohistochemistry:

- CD117 (KIT) 95%, DOG-1 (anoctamin-1) 98%, CD34 60-70%
- desmin, S100, smooth muscle actin NEGATIVE – those mark leiomyoma

Mutations – analysis of KIT & PDGFRA is MANDATORY:

- KIT (c-kit, exons 11 & 9): 70-80%
- exon 11 → longest imatinib response & best survival; exon 9 → short response, worse
- PDGFRA (exons 18, 12, 14): 10-20% – response equal to KIT
- neither → stain for SDH. Absent stain = SDH mutation → genetic counseling

DISTRIBUTION & SYNDROMES

97% sporadic, median age 65, M:F 1:1

Stomach 56% > small bowel 32% > colon & rectum 5-10%, mostly RECTAL

- also mesentery, omentum, retroperitoneum

NF-1: multifocal small bowel GISTs, mutated NF1

Carney–Stratakis: germline SDH mutation – gastric GIST + paraganglioma, young pts

Carney triad: gastric GISTs in young females + paraganglioma + pulmonary chondroma

PRESENTATION & DIAGNOSIS

Slow growing, usually incidental on scope, imaging, or at surgery

Erode into the lumen → bleeding; through the serosa → peritonitis

20% overt mets at dx – LIVER first, then peritoneum.

Lung, bone, node RARE

Submucosal → mucosal biopsy low yield unless ulcerated.

EUS + FNA ↑ yield

AVOID biopsy of a resectable primary – highly vascular, risks rupture & seeding

- biopsy only if metastatic, unresectable, or lymphoma suspected

CT enterography & MRI – MRI PREFERRED for rectal & duodenal, and for serial response

PET not for dx – use it to judge treatment response

RISK STRATIFICATION (MODIFIED NIH / JOENSUU)

HIGH RISK if any of: > 10 cm any mitotic rate, > 5 cm w/ > 5 mitoses / 50 HPF, or RUPTURE

Non-gastric site is higher risk than gastric at the same size & mitotic index

After resection of a NON-ruptured GIST, mitotic rate is the strongest prognostic factor

SURGERY

Complete excision with a 1-2 cm margin = GOLD

STANDARD for localized dz

Do NOT rupture the tumor or violate the pseudocapsule → seeding

No spread to draining nodes → lymphadenectomy NOT necessary

Small bowel & colon: segmental resection. Rectosigmoid / upper rectum: anterior resection

Mid–low rectum: by size, distance to verge, sphincter involvement

- full thickness local excision via TEM, TEO, or TAMIS
- Changchien: LE mean 4.5 cm vs radical 7.2 cm, yet LR 77% vs 31%
- LAR or APR for bulky low tumors that fail to respond to neoadjuvant imatinib

IMATINIB (GLEEVEC)

TKI binding the ATP site on CD117 & PDGFRA. FIRST LINE since 2000

Neoadjuvant:

- shrink the tumor to limit extent of resection and morbidity
- Cavar (rectal GIST): ↑ organ preservation & negative margins vs surgery alone

Adjuvant:

- ACOSOG Z9001: 1 yr at 400 mg/day vs placebo → ↑ RFS (HR 0.6)
- EORTC 62024: 2 yrs → ↑ RFS
- Scandinavian SSG XVIII: 3 yrs beats 1 yr for both RFS and OS
- PERSIST-5: 5 yrs → 5-yr RFS 90%, OS 95%. Most recurrences AFTER stopping
- high risk → treat at least 3 years
- f/u H&P + cross-sectional imaging q 6-12 mo x 10 yrs
- q 3-4 mo x 2 yrs after stopping the drug – recurrence peaks there

Recurrent / Metastatic:

- imatinib remains primary Rx. Sunitinib & regorafenib if resistant or intolerant
- Du RCT: resecting residual dz while on imatinib ↑ OS
- debulking helps only if R0/R1 – leaving gross tumor does NOT

NEUROENDOCRINE TUMORS

TERMINOLOGY & GRADE

NET is the PREFERRED term (NANETS, ENETS).

“Carcinoid” is legacy – Oberndorfer 1907

Store chromogranin A & synaptophysin in secretory granules

WDNET (includes carcinoid): smooth, round, polypoid, normal overlying mucosa

PDNET: large, infiltrative, necrosis & hemorrhage – small cell or large cell carcinoma

Grade – mitoses / 10 HPF or Ki-67:

- G1 low < 2, G2 intermediate 2-20, G3 high > 20
- G3 = neuroendocrine carcinoma, aggressive. WDNET spans G1 and G2

INCIDENCE & DISTRIBUTION

~1% of all new cancers in the US. 52-58% of NETs are GI, 21-32% bronchopulmonary

MC sites: rectum, lung, bronchus, appendix, small bowel

Median age at dx: midgut 64, colon 65, rectum 56

PRESENTATION

Midgut – vague & insidious: bloating, cramping, episodic diarrhea, labeled IBS

- advanced → obstruction from tumor OR mesenteric desmoplasia, or ischemia
- think occult NET in chronic bowel complaints or postprandial pain

Rectal – incidental at screening endoscopy, hematochezia mistaken for hemorrhoids

- indolent, rarely if ever produce carcinoid syndrome

Colonic – worse prognosis, present locally advanced and often with distant mets

CARCINOID SYNDROME

Liver inactivates the peptides – needs LIVER METS or a primary outside the portal system

< 10% of colorectal NETs. Up to 90% of pts with metastatic NETs

Hindgut NETs rarely secrete serotonin – rarely cause the syndrome even when metastatic

Flushing 85% (kallikrein), watery diarrhea & cramping 80% (serotonin), palpitations, wheezing

- triggers: stress, food, caffeine, alcohol

Cardiac: serotonin → fibroblasia, ↑ vascular tone, bronchoconstriction, plt aggregation

- RIGHT sided – tricuspid & pulmonic stenosis, RVH & fibrosis
- left side SPARED – the lung inactivates serotonin

WORKUP

CgA elevated in 60-100%, sens & spec 70-100%. Tracks tumor volume

- falsely ↑ by PPI, renal or liver failure, chronic gastritis

24-h urine 5-HIAA: specificity 88%. < 1% of hindgut NETs secrete – NOT routine

- hold 24 h: banana, kiwi, pineapple, plums, pecans, walnuts, tomatoes, alcohol
- and expectorants, methocarbamol, phenothiazines, phenacetin

Full colonoscopy in all – synchronous lesions

CT chest/abd/pelvis, ARTERIAL phase at 40 sec + delayed washout – NETs are hypervascular

SRS (octreotide scan) 80-90% sensitive. Positive → will respond to octreotide

- part of the initial MIDGUT workup. NOT for the initial hindgut workup

68Ga-DOTA PET/CT: sens 93%, spec 96%. FDG-PET only for PDNET or equivocal SRS

Rectal 1-2 cm → ERUS, EUS, or rectal MRI for size, depth, and nodes

AJCC STAGING – COLON & RECTAL NETS

T1: lamina propria or submucosa. T1a < 1 cm, T1b 1-2 cm

T2: muscularis propria, or > 2 cm with LP / submucosal invasion

T3: through MP into subserosa or non-peritonealized pericolic / perirectal tissue

T4: peritoneum or other organs

Stage I T1 – IIA T2 – IIB T3 – IIIA T4 – IIIB N1 – IV M1

TREATMENT

Appendiceal NETs – see Ch 33

Midgut (jejunum & ileum):

- greater malignant potential – even sub-cm lesions present advanced
- < 1 cm: 20-30% already node positive
- oncologic resection with WIDE lymphadenectomy is PREFERRED
- resect even with mets – prevents obstruction, ischemia, fibrosis
- map the mesenteric resection BEFORE dividing bowel – preserve length
- 1/3 are multicentric – run the whole small bowel

Colon:

- cecum & ascending usually bulky → formal right hemicolectomy + lymphadenectomy
- small colonic NETs: node mets only 4% – endoscopic removal reasonable

Rectal < 1 cm, T1:

- endoscopic resection, and TATTOO the site
- positive margin is the SOLE predictor of recurrence (5-20%)
- EMR & ESD beat conventional polypectomy for complete resection (OR 0.42)
- no further workup and no f/u required

Rectal 1-2 cm:

- controversial. Size > 1 cm and LVI predict node mets
- T1 or T2, node negative, no LVI or PNI → TRANSANAL EXCISION (TES)
- TES also re-excises the scar after a positive endoscopic margin

Colonic or rectal > 2 cm, into MP, N+, or adverse histology:

- RADICAL resection + lymphadenectomy, same oncologic principles as adenoCA

No role for routine adjuvant medical therapy after resection of any of these

Metastatic:

- somatostatin analogs FIRST – octreotide, lanreotide, pasireotide
- 80% of NETs carry somatostatin receptors
- symptom control AND antiproliferative – so use regardless of symptoms
- diarrhea: loperamide, diphenoxylate/atropine. Flushing: antihistamine or H2

- ECOG RCT 5-FU/doxorubicin vs 5-FU/streptozocin: OS 15 vs 24 mo – chemo is weak
- interferon α : 12% respond. XRT for bone mets only
- liver debulking $\uparrow$ survival & palliates hormonal Sx
- chemoembolization – hepatic mets are fed by the hepatic artery

SURVEILLANCE & PROGNOSIS

Hindgut < 1 cm completely removed: NO follow-up

1-2 cm rectal excised: NANETS none, NCCN ERUS or MRI at 6 & 12 mo

More advanced: annual visit + CT or MRI abd/pelvis for 7-10 yrs – recurs late

5-yr survival rectal < 1 cm no LVI ~100%, node positive 54-73%, metastatic 15-30%

Midgut is worst: node negative 80%, node positive < 40%. Metastatic midgut incurable

LYMPHOMA

INCIDENCE

Primary extranodal lymphoma: in an organ w/ no evidence of spread from distant nodes

GI tract is the MC extranodal site – but colon & rectum are the LEAST common GI site

- 10% of GI lymphomas, < 1% of all colorectal neoplasms

Cecum MC (73%), then rectum. Age 50-70, men 2:1

Risk factors:

- FHx lymphoma, prior XRT or chemo, transplant, immunosuppression, HIV, hep C
- IBD itself is NOT a risk factor – azathioprine, 6-MP, and infliximab ARE

HISTOLOGY

Nearly all NHL – primary colorectal Hodgkin only in case reports

Extranodal marginal zone B cell ~45%, diffuse large B cell (DLBCL) ~45%

- follicular, mantle cell, Burkitt's, T cell each < 5%

Subtype dictates treatment and outcome

PRESENTATION & DIAGNOSIS

Weight loss, night sweats, fever, lethargy, palpable abdominal mass (up to 80%)

Otherwise mimics CRC – bleeding, anemia, pain, diarrhea, obstruction

Dawson's criteria: dominant colorectal lesion with only local adenopathy, no palpable superficial nodes, no chest adenopathy, normal WBC & differential, uninvolved liver and spleen

- now liberalized to any dominant extranodal site after clinical staging

Scope: mucosal nodularity & induration, or a mass $\pm$ ulceration

- colonoscopic biopsies are often too SUPERFICIAL – dx may only come at resection

Need histology + flow cytometry + molecular studies.

PET/CT chest, abd, pelvis

BONE MARROW biopsy is critical to stage

vs adenoCA on imaging: extension into T1, well-defined margins, preserved fat planes, no invasion of adjacent structures, perforation WITHOUT desmoplasia

Modified Ann Arbor staging:

- I: single node region or lymphoid structure
- II: two or more node regions, same side of the diaphragm
- III: node regions on BOTH sides of the diaphragm
- IV: extranodal site beyond that designated E
- A no symptoms / B fever 38°, drenching sweats, 10% wt loss over 6 mo
- E: single extranodal site contiguous or proximal to a known nodal site

TREATMENT

Nodal lymphoma = chemo & XRT. Colorectal is

DIFFERENT – irradiated bowel does badly

Localized dz $\rightarrow$ SURGICAL RESECTION FIRST, then chemo

- rationale is twofold: cure, and prevention of obstruction, perforation, bleed
- Korean series, 345 pts w/ colorectal DLBCL, surgery + chemo vs chemo alone
- relapse 15.3% vs 36.7%; 3-yr OS 91% vs 62%

Node positive (stage IIE) in 62-86% at surgery $\rightarrow$

EXTENDED lymphadenectomy, as for adenoCA

Adjuvant CHOP x 3-6 cycles $\pm$ rituximab

Unresectable DLBCL, or localized dz unfit for surgery: full 6 cycles CHOP

External beam XRT NOT a preferred adjuvant – small & large bowel toxicity

- rectal lymphoma is the exception – can be irradiated sparing small bowel

Two scenarios that MUST be operated:

- acute perforation of a colonic lymphoma $\rightarrow$ resect
- chemo-induced tumor necrosis $\rightarrow$ subacute perforation, abscess, or fistula
- percutaneous drainage, antibiotics, optimize counts, THEN resect

Less common subtypes:

- MALT: stomach & small bowel mostly. H. pylori drives 90% of GASTRIC MALT, role in colorectal unclear. Size determines local excision vs radical resection
- Follicular: adjuvant chemo of unclear benefit. Upfront surgery for localized dz
- T cell: 18% of primary GI lymphoma – multifocal ulcers, young pts, hematochezia, fever, perforation. POOR prognosis even when localized
- Mantle cell: usually a SECONDARY GI site, not a primary

35 MALIGNANT DISEASE: CYTOREDUCTIVE SURGERY AND HIPEC

BACKGROUND

TERMS

Peritoneal malignancy: primary (mesothelioma – rare) or secondary

- secondary from colorectal, appendiceal, gastric, ovarian

Carcinomatosis: diffuse involvement of parietal AND visceral peritoneum – a terminal event, usually via inoperable obstruction

Colorectal peritoneal metastases (CPM): the PREFERRED term for resectable dz, analogous to liver or lung mets. “Carcinomatosis” is what is NOT curable by CRS

Sugarbaker, NIH, 1980s – IP vs IV 5-FU RCT with protocol second look at 6 mo

- no survival or time-to-recurrence benefit
- but peritoneal implants at second look 2/10 (IP) vs 10/11 (systemic)

PSEUDOMYXOMA PERITONEI

Diffuse mucinous ascites from peritoneal implants – “jelly belly”

Incidence 2-4 operable cases per million per year

Primary is a perforated mucinous epithelial tumor of the APPENDIX in the majority

NOT benign – “borderline malignant” at best, with inevitable persistence and progression

PSOGI / Carr classification:

- primary: LAMN, HAMN, appendiceal adenocarcinoma ± signet ring cells
- implants: mucinous carcinoma peritonei – low grade or high grade

Appendiceal mucinous neoplasms stay in the peritoneum, rarely node or distant mets

- systemic chemo is generally INEFFECTIVE → ideal for locoregional therapy

THE REDISTRIBUTION PHENOMENON

Three mechanisms drive where disease lands:

- normal peritoneal fluid circulation & absorption
- gravity
- relative sparing of motile organs, especially small bowel

Absorption sites = greater omentum and the sub-diaphragmatic lacunae, RIGHT > left

- explains the omental cake – the omentum filters and concentrates tumor cells

Gravity → pelvis and paracolic gutters

Peristalsis protects the small bowel – overcome by invasive tumor, adhesions, advanced dz

Look HERE at imaging, laparoscopy, or laparotomy: right hemidiaphragm, omentum, pelvis, ovaries

Peritoneal cavity holds ~50 mL serous fluid

COLORECTAL PERITONEAL METASTASES

Synchronous CPM in ~7%; a further 10-20% develop metachronous CPM

Risk factors for peritoneal dissemination:

- T4, tumor perforation, poor differentiation
- mucinous and signet ring histology
- possibly intraluminal stenting of an obstructing tumor

Differs from PMP – invasive, so small bowel serosa & mesentery are commonly involved

Metachronous dz seeds prior dissection planes – Toldt's fascia, the presacral/TME plane

NO imaging detects lesions < 3-5 mm – not CT, not PET-CT, not MRI

- diagnostic laparoscopy is more accurate for the accessible cavity

Systemic chemotherapy underperforms in CPM:

- CAIRO2 subgroup, CAPOX + bevacizumab ± cetuximab: median OS 15 mo, PFS 6 mo
- pooled US trials: median OS 12.7 mo, significantly worse than non-peritoneal sites

SCORING

PERITONEAL CANCER INDEX (PCI) – SUGARBAKER

9 abdominopelvic regions + 4 small bowel regions = 13 regions

Lesion size score per region 0-3: 0 none, 1 ≤ 0.5 cm, 2 ≤ 5 cm, 3 > 5 cm or confluent

- MAXIMUM SCORE 39

Independent predictor of survival even after a complete cytoreduction

- French multicenter, 525 pts, 5-yr OS: 44% for PCI < 6, 22% for PCI 7-12, 7% for PCI > 19
- morbidity & mortality also rise with PCI
- PCI > 19 has traditionally been a contraindication to surgery

Only truly accurate at LAPAROTOMY – “radiological PCI” and “laparoscopic PCI” UNDERESTIMATE

Do NOT use PCI to select PMP patients – high-PCI PMP is often completely cytoreducible and curable

COMPLETENESS OF CYTOREDUCTION (CC) SCORE

Assigned by the surgeon at the END of the case – its main drawback

- CC-0: no visible macroscopic residual disease
- CC-1: nodules < 2.5 mm
- CC-2: 2.5 – 25 mm
- CC-3: > 25 mm, or confluent unresectable nodules anywhere

PMP: CC-0 and CC-1 both count as complete – nodules < 2.5 mm are penetrable by IP chemo

CPM: the benefit is essentially confined to CC-0. CC-1 does WORSE

Complete cytoreduction is not achievable in ~20% taken to the OR for established CPM

- extensive SMALL BOWEL involvement is the MC reason to abandon

CYTOREDUCTIVE SURGERY

Complete macroscopic tumor removal, guided by the redistribution pattern

Standard: radical greater & lesser omentectomy, pelvic ± diaphragmatic peritonectomy, BSO in females

Peritonectomy procedures:

- right parietal, left parietal, pelvic
- right & left diaphragmatic
- right & left liver capsulectomy

Organs resected:

- greater & lesser omentum, spleen
- ovaries / tubes, uterus, gallbladder
- partial or total gastrectomy – 12% of PMP pts in the DiFabio series

Advanced PMP may need a combination of most of these

– paradoxically the LEAST aggressive

- histology demands the MOST extensive surgery

Such combinations are NOT justified in biologically aggressive dz like CPM

Stoma for extensive left-sided anastomoses; consider for all anastomoses if anatomically possible

No data on anastomosis before vs after chemoperfusion, or stapled vs hand-sewn

No role for debulking in CPM – exception is palliative BSO for symptomatic, chemo-resistant ovarian mets

HIPEC

Hyperthermia is itself anti-tumor and potentiates several agents

Performed AFTER a complete macroscopic cytoreduction

Open, semi-open, or closed perfusion technique

Regimens for CPM:

- mitomycin C, 60-90 min at 41-43 °C, open or closed
- oxaliplatin, 30 min at 43 °C, open
- some protocols add simultaneous IV 5-FU
- NO standard agent or duration for either appendiceal or colorectal dz

Also rewarms a hypothermic patient at the end of a long laparotomy

- BJM: markedly reduced reoperation for postoperative bleeding

PIPAC (Reymond 2011): aerosolized IP chemo at laparoscopy for diffuse low-volume dz not resectable. Disease control & palliation of ascites in ovarian, gastric, colorectal, mesothelioma. Being studied to convert unresectable to resectable

EVIDENCE

Verwaal RCT (phase III, 2003, 8-yr f/u 2008):

- 105 pts: 5-FU/LV + palliative surgery vs CRS + HIPEC with mitomycin C
- median survival 22.2 vs 12.6 mo
- CC-0 subgroup: 5-yr survival 45%

Elias 2009 (French, retrospective, 48 vs 48):

- CRS + HIPEC vs chemo alone: 5-yr OS 51% vs 13%, median 62.7 vs 23.9 mo

PRODIGE 7 – the trial that changed the field:

- CRS alone vs CRS + oxaliplatin HIPEC, all with COMPLETE cytoreduction
- median survival IDENTICAL at 42 months
- HIPEC arm had MORE complications at 90 days; treatment-related mortality 1.5% in both
- read as: HIPEC adds nothing once CRS is complete
- caveat – oxaliplatin may simply be the wrong IP agent. Mitomycin C may re-emerge
- BJM: HIPEC may still prolong “obstruction-free survival”

Ovarian cancer RCT (2018): HIPEC + CRS DID beat CRS alone

Complete cytoreduction & HIPEC: 5-yr survival 40-60% in highly selected pts

Adverse prognostic factors:

- signet ring cell histology, systemic mets, high tumor VEGF expression
- positive peritoneal cytology, IBD-related colorectal cancer
- ovarian mets are NO LONGER adverse, provided cytoreduction is complete

SYSTEMIC CHEMOTHERAPY: TIMING

~50% of pts who undergo CRS + HIPEC develop EXTRA-peritoneal recurrence

Little evidence for adjuvant chemo after CRS + HIPEC – 280-pt series showed no difference by timing

2017 systematic review: adjuvant may help; neoadjuvant unclear and may WORSEN OS in some

Passot: major or complete pathologic response to neoadjuvant → significant survival benefit

- so neoadjuvant selects good tumor biology, like it does for liver mets
- and buys a trial of time with re-imaging in borderline resectable cases

PREVENTION & EARLY DETECTION

The shift in the field: from treating established CPM to preventing it

High-risk features:

- limited synchronous peritoneal mets completely resected at the primary operation
- isolated synchronous ovarian mets
- primary tumor perforation (including iatrogenic rupture)
- pT4 primary
- mucinous primary

Three strategies:

- Proactive: resect the primary + organs at risk (omentectomy, appendectomy, BSO) + HIPEC
- Italian PROMENADE trial, Spanish HIPECT4 (T4a/b, mitomycin C)
- Adjuvant: HIPEC at or soon after the primary resection

- Dutch COLOPEC (T4 or perforated): oxaliplatin
HIPEC did NOT improve 18-mo peritoneal RFS
- Second look: planned relook 6-12 mo after resection, with CRS + HIPEC of anything found
 - Elias 2011, 41 high-risk pts w/ normal imaging & markers: 56% had dz at second look
 - 5-yr OS 90%, DFS 44%
 - French PROPHYLOCHIP: NO difference in 3-yr DFS vs surveillance

SPECIAL SITUATIONS

Synchronous liver or lung mets:

- no longer an absolute contraindication
- up to 3 resectable liver mets and PCI < 10 may benefit, some cured
- same principles for treatable (resect or ablate) lung mets

Palliative CRS:

- complete removal is a PREREQUISITE – “debulking” morbidity outweighs benefit
- systemic chemo + best supportive care is better if CC-0 is unachievable
- exception: large ovarian colorectal mets – a sanctuary site from systemic chemo

BOTTOM LINE

Refer to a peritoneal malignancy center BEFORE starting any treatment

If CPM is found at surgery: minimize dissection, do NOT disrupt planes, biopsy, and stoma rather than resect for impending obstruction

The two prognostic factors that matter: COMPLETENESS of cytoreduction and EXTENT of disease (PCI)

PMP → CRS + HIPEC, cure is realistic even at high PCI

CPM → CC-0 or do not start. HIPEC is an adjunct of uncertain value after PRODIGE 7

36 MALIGNANT DISEASE: COLORECTAL CANCER —

MINIMALLY INVASIVE SURGERY

EVOLUTION & EVIDENCE

PORT SITE RECURRENCE

1994 report of 21% port site recurrence → societies advised AGAINST lap for curable cancer

Disproven – actual rate ~1%, same as open wound implants. ASCRS registry: 0.4%

- review of the index videos → the problem was TECHNIQUE, not pneumoperitoneum

COLON CANCER TRIALS

COST (Nelson), Barcelona (Lacy), COLOR (Veldkamp), CLASICC (Guillou)

All reached the same conclusion – lap colectomy in EXPERIENCED hands is oncologically NOT inferior

- faster recovery, ~1 day shorter LOS, better short-term function, better cosmesis
- by 2-3 months NO difference in function or QOL
- longer operative time and higher cost

Learning curve is steep – 20-152 cases to basic proficiency

Complete mesocolic excision – see Ch 37

RECTAL CANCER TRIALS – THE CONTROVERSY

Proctectomy techniques are LESS mature than colectomy.

This is where MIS is contested

CLASICC:

- CRM+ 12% lap vs 6% open. CONVERSION 34%
- concluded short-term outcomes did not justify routine use
- but at median 5 yrs, NO difference in OS or DFS

COREAN:

- after neoadjuvant CRT, mid-low rectal cancer
- ↓ blood loss, ↓ post-op pain, ↓ LOS. 3-yr DFS comparable

COLOR II:

- non-inferior. CRM+ and 3-yr local recurrence equivalent to open

ACOSOG Z6051 & ALaCaRT:

- both FAILED to show non-inferiority
- composite endpoint: CRM negative + distal margin negative + complete or near-complete mesorectum
- no difference in LOS. Incision shorter with lap
- authors of both concluded lap proctectomy should NOT be done for rectal cancer
- reassuring: 3-yr DFS in Z6051 was EQUIVALENT

The unresolved question is whether a pathologic composite endpoint predicts oncologic outcome. The survival data so far say no

WHY THE PELVIS IS HARD

Poor ergonomics, 2-D view, inflexible instruments, coning, fulcrum effect

Bulky uterus & adnexa, redundant pelvic peritoneum, bulky tumor, narrow pelvis

Obese, male, irradiated, low tumor = the case that converts

MIS gain is more than incision size – ↓ systemic

inflammatory response, ↓ wound complications,

- ↓ LOS, ↓ disability. Elderly, obese and frail may gain the MOST

LAPAROSCOPIC PROCTECTOMY

INDICATIONS

Resectable, biopsy-proven adenoCA of the rectum, EXTERNAL sphincter not involved

Upper third → LAR with partial mesorectal excision, if distal margin is adequate

Mid & distal third → LAR with TME, guaranteeing a distal margin ≥ 1 cm

Very low, into the IAS → ISR with hand-sewn coloanal – but ↑ CRM+ and poor function

PREOPERATIVE

Only ABSOLUTE contraindication: cannot tolerate pneumoperitoneum & steep Trendelenburg

Relative: morbid obesity, prior pelvic surgery, severe cardiopulmonary dz, dense adhesions

Ureteral stents for bulky pelvic tumors or prior pelvic surgery – lighted stents help in robotic

- cases given the absent haptics. Not essential

Stoma site marking & education by an enterostomal therapist

ACCESS & VESSEL LIGATION

Lithotomy or supine split-leg, arms & head tucked – the table WILL be tilted. Surgeon on the right

10 mm umbilical open access for a 30° scope, 10-12 mm RLQ lateral to rectus for the stapler,

- three 5 mm (RUQ, left lumbar, LLQ) – the left ports are the assistant's

Trendelenburg + right tilt → small bowel into the RUQ

MEDIAL to LATERAL: incise retroperitoneum from the sacral promontory to the IMA root

- do NOT injure the superior hypogastric plexus, left ureter, or gonadal vessels

Dissect deep to the IMV, sweeping the descending mesocolon off the retroperitoneum

- do NOT injure the pancreatic tail

Isolate the IMV at the ligament of Treitz – protect the communicating branches of the left colic

- and the left branch of the middle colic

Re-visualize the left ureter, THEN divide the IMA and IMV

SPLenic FLEXURE & LEFT COLON

Mobilize for a tension-free lumen AND a tension-free mesentery – blood supply

Divide omental attachments, mobilize the left mesocolon, divide the bare area at the root of the

- distal transverse mesocolon. Then the white line of Toldt, then the sigmoid mesocolon

Extra caution with:

- prior aortic surgery – can interrupt or reverse marginal artery flow
- prior left nephrectomy, prior colonic surgery

Griffith's point (splenic flexure): marginal artery of

Drummond tenuous, ABSENT in up to 5%; a 1.2-2.8 cm² area may have no vasa recta

Sudeck's point (rectosigmoid): distal to the last collateral to proximal arteries

Right colon: marginal artery poorly developed in 50% – vulnerable in low-flow states

MESORECTAL MOBILIZATION – TME

Incise peritoneum at the lateral border of the mesorectum, MEDIAL to the ureters

Connect anteriorly at the peritoneal reflection in the cul-de-sac

Start POSTERIOR at the sacral promontory – assistant lifts the redundant colon out of the pelvis

Sharp cautery between the parietal and visceral layers of the endopelvic fascia

Hypogastric nerves cross anterolaterally at the aortic bifurcation, ~2 cm medial to the ureters

Stay on the fascia propria – protects hypogastrics, pelvic plexus, and cavernous plexus

Straying into the pelvic sidewall → internal iliac vessel bleeding and nerve injury

An AVASCULAR plane is the correct plane. Moderate bleeding = you are in the wrong plane

Below S3 divide the rectosacral (Waldeyer's) fascia. NO blunt dissection – presacral venous plexus

Anterior plane, anterior OR posterior to Denonvilliers', chosen by TUMOR POSITION

- anterior tumor → anterior to Denonvilliers', exposing both seminal vesicles
- in females, the rectovaginal septum

Localize the tumor with flexible endoscopy or rigid proctoscopy BEFORE transecting anything

DISTAL TRANSECTION & ANASTOMOSIS

Distal margin ≥ 1 cm

Why laparoscopic transection is hard:

- fulcrum effect of a stapler through an iliac fossa port
- limited angulation – a stapler would have to pass THROUGH the iliac bone for 90° at the levators
- multiple cartridge firings → ↑ leak risk
- intersecting lateral staple lines (dog ears) are weak spots
- oblique transection → stricture from poor perfusion at the acute angle

Options: tilt the rectum left, suprapubic stapler, or a stapler through a gel hand port

- gel port fixes the fulcrum, stabilizes pneumo, allows palpation. If you want it, place it at the START

Avoiding dog ears:

- "tennis racket": spike through the stump LATERALLY – eliminates one dog ear
- "reverse smile": spike centrally just posterior to the staple line, retract the shaft and angle
 - anteriorly – the whole linear staple line is excised, making it single-stapled

- do NOT entrap the vagina or other pelvic structures

Extract via Pfannenstiel or the LLQ port with a wound protector. Transect and place the anvil

ICG fluorescence angiography to check conduit perfusion – no definitive data yet (IntAct RCT ongoing)

Leak test with CO₂ – standard via proctoscope, or reverse via peritoneal insufflation

Divert for a distal anastomosis, prior radiation, or significant comorbidity

INTERSPHINCTERIC RESECTION

Assess sphincter function preoperatively, counsel on functional derangement. Only for tumors

- NOT amenable to standard LAR

Transanal circumferential incision of anal mucosa in the intersphincteric groove, DISTAL to dentate

En bloc rectum + IAS, transanal extraction, hand-sewn coloanal, proximal diversion

LAPAROSCOPIC APR

Still required in ~18% of rectal cancers. Also for

recurrence, salvage of anal CA or melanoma, and

- for pts with marginal continence or high risk of severe LARS

No RCT subset analysis for APR – data are limited

Ng RCT (open 48 vs lap 51): earlier bowel function (3.1 vs 4.1 d), earlier ambulation (4.4 vs 5.9 d)

- but higher cost (\$9588 vs \$7517) and longer OR (213 vs 163 min). 5-yr survival 75% vs 76%

CRM reporting after lap APR is poor – only 6 of 19 series report it, range 2-21%

- Dutch TME trial: CRM+ in 30% of the 455 APR patients

Abdominal steps identical to LAR – put a left trocar at the marked colostomy site

Perineal phase can be done laparoscopically (retrograde): anus sewn shut, start in the ischiorectal

- plane, divide levators, place a SILS or TEM port, insufflate to 15 mmHg, dissect to the cervix
- or seminal vesicles. Smoke & collapse are the problem – two CO₂ lines or continuous exchange

ROBOTIC

3-D magnification, 7 degrees of freedom, stable camera, tremor filtering, shorter learning curve

Against: no haptics, bulk, assistant sees only 2-D, longer OR, cost. 3-D laparoscopes erode the edge

ROLARR:

- primary endpoint was CONVERSION – robotic did NOT reduce it
- no difference in short- or long-term outcomes
- £980 MORE expensive, excluding acquisition and maintenance

Other RCTs: similar short-term, long-term and functional outcomes vs laparoscopy

Docking is the time sink – but meta-analyses now show equal operative time with experience

Fully robotic or HYBRID (lap flexure & vessels, robot docked for the pelvis)

- hybrid puts the robot where it earns its keep – the narrow pelvis

High IMA ligation with wide lymphadenectomy, preserving the hypogastric plexus trunks at the origin

Do NOT grasp the mesorectum with the robotic left hand – the instruments will lacerate the fascia

Tumor $\geq$ 2-3 cm above the anorectal ring → double-stapled anastomosis feasible

- < 2-3 cm → ISR with transanal extraction and hand-sewn coloanal

Robotic APR / ELAPE: avoid “coning in” at the levators – take them wide at their origin

Single-port platform is the emerging role, particularly for transanal work

TRANSANAL TME

Proposed 2010 for the obese male, narrow irradiated pelvis, bulky mesorectum, low tumor

Built on TATA, TEM, TAMIS, NOSE and NOTES

Claimed advantages:

- longer distal margin – transection under DIRECT vision
- ↓ CRM+, improved TME quality
- avoids multiple firings and dog ears, allows a single-stapled anastomosis

Concerns:

- urethral injury, CO2 embolism
- reported MULTIFOCAL local recurrence, attributed to pelvic insufflation spreading cells
- 6-center study: 2-yr LR 3% with NO multifocal pattern
- COLOR III will settle it

Setup:

- abdominal phase before, after, or synchronous.
Lithotomy, Trendelenburg, right tilt
- pudendal block, retractor to the dentate line, flexible platform beyond the levators, 2 stitches
- 0 PDS or Prolene purse string on a 26 mm needle from 5 o'clock clockwise, 1-2 mm apart,
 - 90° entry, full thickness bites. Avoid the vaginal wall anteriorly
- abdominal team clamps at the rectosigmoid to stop colonic distension

Where to place the purse string:

- tumor to anorectal junction > 4 cm → 3 cm from the anal verge
- ARJ distance 2-4 cm → 1 cm distal to the tumor
- < 2 cm → divide the rectal wall FIRST without the platform, then purse string, then place it

Rectal washout, pneumorectum, mark 1 cm below the knot, 360° full-thickness incision

POSTEROLATERALLY

Dissect circumferentially – do NOT funnel. Presacral space posteriorly is safest to start

- avoid the posterior midline initially – the anococcygeal ligament blocks you
- anteriorly stop near the neurovascular bundles at 2 and 10 o'clock
- in males the dissection MUST stay posterior to the prostate and urethra. Lateral landmark
 - is the parietal fascia – too far lateral injures nerves and vessels

Anvil with a tubular drain as a handle, pulled through the distal purse string. Single-stapled

- end-to-end or side-to-end, air leak test, proximal diversion

HAND-ASSISTED

Incision the length of the surgeon's glove size in centimeters

The argument: you make a 4-8 cm extraction incision anyway – so make it FIRST and use it throughout

Periumbilical port first to exclude occult unresectable mets before the larger incision

Lower midline hand port if conversion is likely – extends directly to laparotomy

HALAR vs straight lap vs open:

- retains most benefits of pure laparoscopy
- complication rate & LOS comparable to straight lap
- oncologic outcomes comparable to straight lap and open
- SHORTER operative time and LOWER conversion rate
- slightly longer incision, ↑ inflammatory markers – both of unclear significance

The pragmatic answer to Z6051 / ALaCaRT: mobilize the colon laparoscopically, do the mesorectal

- dissection OPEN through the base of the hand port. Keep the hand in a “C” and out of the view

37 MALIGNANT DISEASE: MINIMALLY INVASIVE

COMPLETE MESOCOLIC EXCISION

CONCEPT

CME (Hohenberger 2009) – the colonic analogue of TME.

Four elements:

- sharp en bloc dissection along the embryologic plane b/n mesocolic & parietal fascia, envelope INTACT
- all lymphovascular tissue from pericolic out to the base of the feeding aa.
- CVL – high tie at the most proximal extent of the feeding aa. & vv.
- adequate bowel length to guarantee the nodal harvest

Not a new idea – Jamieson 1909, Enker 1979, routine in Japan since the late 1990s

Original description required a Kocher to expose the vessel origins – modern practice does NOT, anterior SMV dissection only (= JSCCR D3)

“CME + CVL” is a misnomer – the extent of lymphadenectomy varies b/n authors. Here CME + CVL = CME + D3

Left ≠ right in difficulty

- left: high IMA tie at the aorta is already routine at proctectomy – D3 is a small step up
- right: SMV tributaries & trunk of Henle – the reason Western adoption has lagged

LYMPHADENECTOMY LEVELS (JSCCR)

D1: *epicolic & paracolic, along the marginal artery*

D2: *D1 + intermediate nodes along the tumor-supplying artery*

- right: ICA at its root, at the lower border of the duodenum
- left: IMA just proximal to the left colic origin
- right branch of MCA can be taken high WITHOUT exposing the SMV

D3: *D1 + D2 + central*

- right: expose the ANTERIOR surface of the SMV (SMA dissection NOT required), vessels at origin, nodes along SMV & lateral to SMA – groups 203, 213 (+ 223 for extended right)
- left: IMA divided at the aorta, aortic wall exposed – group 253 (+ 223 & SMV exposure for extended left)

D4: *along aorta & IVC, or SMA/SMV central to the MCA origin*

n(1)–n(4)(+) designate the area of the nodal mets – n(4)(+) counts as distant mets

Level by cT stage:

- pTis excised endoscopically: none, or D1 if resecting
- pT1: D2 – ~10% D1/D2 mets, 1.9% D2
- cT2: D2 minimum, D3 reasonable – 0.7-1% central mets & cT staging is inaccurate
- cT3/T4: D3 is STANDARD in Japan
- central node mets by pT (25,617 pts): T2 0.9%, T3 2.3%, T4a 5.7%, T4b 6.7%

QUALITY INDICATORS OF A CME SPECIMEN

Plane of dissection – muscularis propria (poor) / intramesocolic / mesocolic (good). Quirke grading moved to the colon

LN count, ideally mapped by JSCCR station

Morphometry: tie → tumor, tie → bowel wall, area of mesentery (mm²)

Large & small bowel length

- graded off high-res photos of the unfixed unopened specimen, front & back, mesentery laid flat next to a ruler

West & Hohenberger: CME removes significantly more tissue & more nodes than non-CME

Mesocolic plane achieved in only 32% of specimens – but carried a 27% 5-yr survival advantage in stage III over the muscularis propria plane

Mesocolic plane is easier on the LEFT than right or transverse

Bowel margin, Japanese rule:

- 10 cm from the tumor, 5 cm beyond a feeding vessel in the direction of lymph flow
- longitudinal spread > 10 cm is rare – 0% left, 1-4% right
- Western CME instead takes the NEXT feeding vessel beyond the 10 cm → longer specimens, larger mesentery

NODAL BIOLOGY

Halsted model: stepwise pericolic → intermediate → central. D3 helps

Fisher model: nonlinear, bypasses nodal basins. D3 does not help

Skip mets – central positive with pericolic & intermediate negative: 0-19.8% of stage III

Central (D3+) mets in stage III: 8.3% (JSCCR, 1355 pts) up to 19.7% (244 right colon CA)

D3+ independently a/w cancer-specific death – HR 2.29

Right vs left stage III (4034 pts): D3+ 8.5% vs 3.7%, skip mets 13.7% vs 9%

- D3+ predicted worse OS only on the LEFT → D3 may matter most in left-sided stage III
- 5-yr OS worse for right-sided overall, 77.4% vs 80.9%

Past some distance a positive node is a marker of systemic dz, not a target

ANATOMY – THE RIGHT SIDE IS THE PROBLEM

Trunk of Henle (GCT / gastropancreaticocolic trunk):

- present in 37.8-89%
- confluence of RGEV + ASPDV + colic vein(s); a single colic vein in 55.8%
- NO common trunk b/n colic & gastroepiploic veins in 20.7-21.6%
- PRESERVE the RGEV and the ASPDV. Take the SRCV / RCV

SMV tributaries:

- MCV: one in 72-74%, two 22-26%, three 1.8-3.5%.
Drains directly into SMV in 86-89%
- SRCV in 28.8-83.6%; RCV in only 19-24.3% but always drains directly into the SMV
- a single ICV in 100%, into the SMV in 92.8-100% (else GCT or jejunal trunk)
- ICV may NOT travel with the ICA – can enter the SMV up to 3 cm cranial to the ICA base

Arterial:

- ICA off the SMA in 100%, crosses the SMV either anteriorly or posteriorly
- RCA present in only 10-63% – off SMA in 33%, else off ICA or MCA; crosses SMV anteriorly in 87-97%
- MCA off the SMA in 100% – single 88%, double 11.7%
- SMA sits just left and posterior to the SMV
- IMA splits into left colic & superior rectal 2-3 cm off the aorta

Avulsion of the MCV from overzealous traction is the classic mechanism of hemorrhage

TECHNIQUE – RIGHT & TRANSVERSE

Medial to Lateral:

- avascular plane mesocolon/retroperitoneum to the 2nd portion of duodenum & head of pancreas
- ICA & ICV at the root of SMA/SMV, then cephalad anterior to SMA & SMV
- RCA (if present) at its base; GCT dissected preserving ASPDV & RGEV; SRCV/RCV divided
- MCA & MCV at their base last
- cecal/ascending: spare the main & left branch of MCA, take the right branch only
- hepatic flexure/transverse: MCA at its base
- distal transverse: LEFT colectomy sparing the right branch of MCA, ligate IMV, take the flexure
- CHECK retrograde IMA flow before dividing the main MCA – elderly, atherosclerotic
- omentectomy inferior to the gastroepiploic vessels, preserve them unless involved

SMV-First (robotic):

- incise below the ileocolic vessels, find the SMV, clear all tissue off its anterior surface cephalad
- then ICV, then ICA off the SMA, then RCA / MCA, then the GCT along the right anterolateral SMV

Retrofascial Uncinate-First (Benz):

- open-book model – mesogastric, mesocolic and ileocolic “pages”, GCT the spine, pancreas the back cover
- the avascular plane b/n pages MUST be opened before any vessel is divided. 9 steps, each with a critical view
- 1: incise at the duodenojejunal flexure, retromesenteric dissection caudally – view: D3 & uncinate process
- 2: tension the ileocolic pedicle – the V b/n ileocolic & superior mesenteric vessels
- 3: incise 3 cm distal to the ileocolic root, dissect SMV 180-270°, ICV 360°, divide ICV

- 4: ICA 360° at the level of the SMV, clear all tissue right of the SMA, divide
- 5: enter the lesser sac 5 cm LEFT of midline – it is obliterated right of the MCA – preserving the gastro-omental arcade
- 6: separate mesogastrium from mesocolon, find the sulcus. SRCV runs OUTSIDE the axis – the bleeding point
- 7: from below, incise transverse mesocolon anterior to the SMV, take the right branch of MCA at its origin, spare the left
- 8: expose the trunk of Henle, divide all mesocolic veins, PRESERVE RGEV & ASPDV
- 9: lateral attachments, then intra- or extracorporeal anastomosis

Lateral to Medial:

- for when the ileocolic base can't be identified. Toldt's
→ Gerota's, protect ureter, kidney, gonadals

Cranial to Caudal (Asia):

- open the omental bursa first, GCT found via RGEV & SRCV, MCV at its root, then work down the SMV

TECHNIQUE – LEFT COLON D3

Medial to lateral, incise just above the sacral promontory
Skeletonize the IMA at its base, clear all nodal & areolar tissue at the root

- protect the superior hypogastric plexus at the aortic bifurcation, the left ureter and gonadals
- proximal left colon tumor: LEFT COLIC at its origin, preserve IMA root & superior rectal if anastomosing to distal sigmoid
- mid-left, sigmoid, rectosigmoid: IMA at its origin on the aorta

IMV divided just inferior to the pancreas – ligament of Treitz is the landmark

- CHECK for an artery beside the IMV first – dominant flow can run in the left branch of the MCA at the root of the mesentery. Divide it and the conduit is ischemic

OUTCOMES & THE TRADE-OFF

Short-term:

- pooled morbidity CME 22.5% vs non-CME 19.6%.
Longer OR time
- Danish DCCG (529 CME/D3): intraop ORGAN INJURY 9.1% vs 3.6% – splenic & SMV. Lap attempted 48.8%, conversion 19.9%
- conversion in the literature 5-22%, right colectomy the worst
- Freund: vascular injury 1.6% over 304 right colectomies – misperceived anatomy, MCV avulsion
- Melich: OR time fell only from 250 to < 200 min over the first 81 lap CME cases
- also chyle leak, diarrhea from superior mesenteric plexus injury, GU dysfunction from left-sided plexus injury

JCOG0404 (open vs lap CME + D3, 521 vs 527, stage

II/III):

- conversion 5.4%; organ injury 3.6% lap vs 1.7% open, NS – duodenum, arterial, GCT
- lap: less blood loss, faster ileus resolution, shorter LOS, morbidity 14.3% vs 22.3%
- 5-yr OS 91.8% lap vs 90.4% open, DFS 79% vs 80%

COLD trial (D2 vs D3, first 100 pts):

- no difference in OR time, blood loss, conversion (12 vs 13%), or 30-d complications
- experienced D2 surgeons CAN transition to D3

Long-term:

- Hohenberger, Erlangen 1329 pts: 5-yr locoregional recurrence 6.5% → 3.6%, cancer-related survival 82.1% → 89.1%. Only 5.6% got adjuvant chemo
- updated Erlangen, stage III: LR 14.8% → 4.1%, 5-yr CSS 61.7% → 80.9%, OS 53.1% → 68.6% – but adjuvant use went 0 → 79%
- Danish population data: higher 4-yr DFS with CME, esp. stage I/II. Right colectomy 5.2-yr recurrence 9.7% vs 17.9%
- CME + D3 vs D2, stage I-II: 3-yr OS 88.1% vs 79%, DFS 82.1% vs 74.3%
- pooled analysis: 5 studies show NO difference in 3- or 5-yr OS. Evidence does NOT consistently support oncologic superiority
- the criticism: historical controls, non-CME cohorts drawn from other hospitals, implausible control recurrence rates (14.8%, 20.6%), no quality indicator beyond node count

JAPANESE VS WESTERN FRAMING

Japan: dissection defined by anatomic nodal STATION, mapped onto the specimen, D3 standard for cT3/T4

- adoption 58.4% (2001) → 75% (2010) by photodocumentation

West: quality = ≥ 12 nodes (ASCO, ACS CoC, NCCN) + high ligation + negative margins

- no station mapping, no expectation for mesenteric area or level of tie

Translation: CME + D2 is US STANDARD OF CARE, with SELECTIVE D3 for suspicious central nodes

BOTTOM LINE

Mesocolic-plane, envelope-intact, tumor-specific CME – for everyone

D3 – selective: cT3/T4, suspicious central nodes on imaging, probably left-sided stage III

Right-sided D3 is where the injuries live. Know the trunk of Henle before you start

Ongoing: COLD, RELARC, SLRC, LCME – all with video-based technique QC and photo-based specimen morphometry

38 BENIGN COLORECTAL DISEASE: COLONIC

DIVERTICULAR DISEASE

DEFINITIONS

Uncomplicated: no abscess, fistula, stricture, obstruction, or free perforation

- microperforation w/ small contained extraluminal gas and NO systemic inflammatory response is still UNCOMPLICATED

Complicated: abscess, fistula, stricture, obstruction, or free perforation

SUDD: diverticulosis + chronic abdominal pain, no overt colitis

- persistent, boring, constant over long periods – NOT crampy. Hard to separate from IBS
- QOL stays impaired after the acute episode clears, often with no imaging correlate

INCIDENCE

- 5% by 40, 80% by 80
- hospitalization 74.1/100,000 (2000) → 96.0/100,000 (2008)
- 1.92 million ambulatory diagnoses in 2014, > 340,000 ED visits in 2012, ~\$1.6 billion/yr
- 10% of those admitted develop a complication needing further mgmt

PATHOPHYSIOLOGY

High intraluminal pressures – 90 mmHg at peak (9x wnl)

Herniation at the vasa recta brevia – where the vessels penetrate muscle to reach mucosa

- most b/n mesenteric & anti-mesenteric tinea
- muscular layer does NOT herniate
- *acquired or pulsion diverticula for most; diffuse disease may be a connective tissue abnormality*

Histology mimics IBD – lamina propria thickening, mucin depletion, Paneth cell hyperplasia, crypt abscesses

Genetics:

- twin studies: heritability accounts for 40-50% of the risk
- GWAS: COLQ, ARHGAP15, FAM155A, TNFSF15 SNPs in surgical pts – also implicated in Crohn's
- LAMB4 (colonic myenteric plexus) a/w early-onset dz

Microbiome:

- ↓ fiber-metabolizing SCFA producers, ↓ Clostridial organisms, ↑ Proteobacteria diversity
- association only – causality NOT established

RISK FACTORS

Diet: high red meat & low fruit/veg increase dz. Veggies & brown bread protective

- Western diet HR 1.55 vs a "prudent" diet
- NUTS, SEEDS, CORN & POPCORN: MYTH – inversely correlated w/ diverticulosis and its complications. Do NOT restrict

- low-risk lifestyle: red meat < 51 g/d, fiber in the top 40% (~23 g/d), 2 h exercise/wk, normal BMI, never smoked

Age/Sex: M:F 3:2 to 3:1. Young men present w/ more severe CT findings; women fail outpatient mgmt more often

- young pts recur MORE but do NOT have more complicated recurrences and are NOT more likely to need emergency surgery

NSAIDs: 23% higher perforation risk, 4x among the hospitalized – COX inhibition → ↓ prostaglandin

Smoking: current smokers RR 2.54 for

abscess/perforation, ever smokers 1.83

Obesity: RR 1.31 per 5 units BMI for an attack, 1.20 for a complication. Women gaining ≥ 20 kg: 73% ↑

Exercise: inverse, independent of fiber. > 57.4 MET-h/wk → RR 0.75. VIGOROUS activity only

Immunocompromised: more free perforation, but overall only ~1% higher rate of complicated dz

Opiates: ↑ intracolonic pressure, slow transit – both raise complication risk

PRESENTATION

- LLQ pain, fever, leukocytosis. RLQ pain if a redundant sigmoid crosses the midline
- no prodromal epigastric pain, rare n/v
- bleeding is a feature of diverticulOSIS, not diverticulITIS – if present think ischemic colitis, IBD, or CA

Differential: appendicitis, obstruction, ruptured AAA, CA, ischemic colitis, pyelo, IBD, IBS, endometriosis, TOA, PID, stones, volvulus, stercoral ulcer, ovarian torsion

Polycystic Kidney Disease: high association – some transplant centers ask for prophylactic sigmoid colectomy pre-transplant

Crohn's: recurrent diverticulitis after a prior resection should make you consider Crohn's

DIAGNOSTIC TESTS

CT: STUDY OF CHOICE. Stages severity, maps the drain, catches SBO, stricture, fistula, pylephlebitis, liver abscess

Labs mostly unhelpful. CRP > 150 mg/L discriminates complicated from uncomplicated; CRP > 150 + free fluid a/w ↑ mortality

AXR: nonspecific – only to rule out pneumoperitoneum

Contrast studies: water-soluble, for a suspected obstructing stricture. U/S & MRI not routine in the US
Air in the bladder without instrumentation is DIAGNOSTIC of colovesical fistula – no cystoscopy or cystogram needed

Endoscopy: delay 6 weeks after resolution

- classic concern is converting a sealed microperf to a free perf; safe in the acute setting per some series, but often incomplete

- CA found after UNCOMPLICATED -itis 0.5-1.3%; after COMPLICATED 7.9-11%. Pooled prevalence across all diverticulitis 2.1%

CLASSIFICATION

Hinchey – complicated acute diverticulitis:

- Stage I: pericolic abscess
- Stage II: distant pelvic or retroperitoneal abscess
- Stage III: purulent peritonitis
- Stage IV: fecal peritonitis

Modified (CT-based) Hinchey:

- 0: wall thickening, NO pericolonic stranding
- Ia: wall thickening + pericolonic fat stranding
- Ib: pericolic or mesocolic abscess
- II: distant intra-abdominal or pelvic abscess
- III / IV: purulent / feculent peritonitis – CT usually CANNOT separate these two

Ambrosetti – severity on CT:

- mild: wall thickening > 5 mm + pericolic fat inflammation
- severe: the above + abscess, extraluminal air, or extraluminal contrast
- severe → operation 33% vs 15%, recurrence 39% vs 14%
- abscess or extraluminal air > 5 cm predicts failure of nonoperative mgmt

MEDICAL MANAGEMENT

Antibiotics – NOT routine for uncomplicated dz:

- AVOD and DIABOLO RCTs: no difference in complications, recurrence, or need for surgery, short or long term
- DIABOLO included small abscesses. Antibiotic-related complications up to 8%
- ASCRS 2020: “selected patients with uncomplicated diverticulitis can be treated without antibiotics”
- SAGES/EAES could NOT reach consensus – practice has not caught up to the data
- when used: gram negatives & anaerobes – E. coli, Streptococcus, Bacteroides, Peptostreptococcus, Clostridium, Fusobacterium

Outpatient management:

- most uncomplicated pts. Readmission ~7%, rarely need drainage, large cost savings
- candidates: hemodynamically stable, well appearing, no major comorbidity, NOT immunosuppressed

Diet:

- no data that restriction alters the course – NORMAL DIET AS TOLERATED
- maintenance fiber 20-30 g/d. No seed, nut, or popcorn restriction

COMPLICATED DISEASE

Abscess (Hinchey Ib & II):

- < 3 cm: antibiotics alone. > 3 cm: percutaneous drainage
- 447-pt multicenter series: failure HIGHER in the drained group, 36% vs 24% – selection bias, the big ones got drained
- recurrence after antibiotics 25.5%, after percutaneous drainage 15%
- emergent surgery for those who fail nonoperative treatment

Hinchey III – nonoperative trial:

- free air + free fluid on CT. If stable, afebrile and non-toxic, antibiotics alone are reasonable
- rectal contrast added to oral/IV excludes an overt lumen-to-peritoneum connection (Hinchey IV)

Laparoscopic Lavage (Hinchey III):

- LOLA/LADIES: closed early – serious short-term AEs 39% vs 19%, reintervention 20% vs 7%, abscess needing drainage 20% vs 0%. BUT 52% never needed any operation, 74% never got a stoma, and 12-mo morbidity/mortality was equal (67% vs 60%)
- SCANDIV: no difference in severe complications at 90 d or 1 yr. Deep SSI 32% vs 13% and reoperation 27% vs 10% w/ lavage; superficial wound infection 1% vs 17% and stoma 14% vs 42% lower. FOUR lavage pts proved to have colon CA
- DILALA: 30-d reintervention no different (13.2% vs 17.1%); 45% reduction in reoperation at 24 months
- pooled: initial success ~3 in 4, ~3/4 stoma-free at 1 yr, 1 in 4 needs a second operation. ↑ abscess, peritonitis, emergency reoperation. More cost-effective
- THE TRADE-OFF: higher early reintervention, equal morbidity, mortality and further surgery at 1 yr

Technique: lavage the ENTIRE cavity until clear. Take fibrinous exudate only if it lifts easily. Elevate the phlegmon and inspect the pelvis to be sure it is not Hinchey IV – endoscopic insufflation finds a patent perforation. Leave densely adherent omentum alone

- early failure = missed Hinchey IV or perforated CA. Both need resection

Hinchey IV:

- resection is MANDATORY to control sepsis
- formal mesenteric resection NOT necessary acutely unless CA is suspected

Primary Anastomosis vs Hartmann's (Hinchey III & IV):

- in stable, immunocompetent pts, PA ± diverting loop ileostomy is SAFE and far more get closed
- 4 RCTs (Oberkofler, Binda, Bridoux, Lambrichts): stoma closure 65-96% PA vs 57-72% HP
- pooled cohort, 529 PA / 366 HP: mortality 7.9% vs 19.5%, deep SSI 3% vs 12%, reoperation 6% vs 16%, closure 86% vs 68%
- if PA, do a PROPER diverticular resection. If Hartmann's, resect only the perforated segment and complete the sigmoidectomy at takedown

ELECTIVE RESECTION

The “resect after two attacks” rule is DEAD

- complications occur most often at the FIRST episode – prophylactic resection does not prevent them
- 75-96% of pts presenting w/ peritonitis had no prior Hx of diverticulitis
- INDIVIDUALIZED, shared decision – symptom severity & QOL against operative risk
- CT confirmation required. IBS mimics recurrent attacks

DIRECT RCT: surgery beat optimal conservative mgmt on QOL for ongoing complaints or recurrent attacks, despite a high complication rate

Young: not more virulent. Higher lifetime recurrence only from more years at risk. Emergency operation 7.3% vs 4.9%

Immunocompromised: same individualized approach – NOT an automatic indication

Fistula – essentially always an operative indication:

- Bladder (MC): pneumaturia, fecaluria, recurrent UTI. Operate on anyone medically fit – recurrent urosepsis
- Vagina: after hysterectomy. Repair for the symptoms
- Cutaneous: from a chronic draining abscess. Expectant at first

Obstruction: operate. Stenting is NOT successful in benign dz. Exclude CA first

TECHNICAL POINTS

Laparoscopy within ERAS is PREFERRED – 2 of 3 RCTs superior, earlier recovery, fewer complications. Pfannenstiel extraction, lowest hernia rate

- evidence now accumulating for laparoscopy in the emergency setting as well

Margins:

- distal margin MUST be the PROXIMAL RECTUM – colorectal beats colocolic for recurrence
- proximal margin where the bowel is not thickened. Diverticula themselves DO NOT set the margin
- retained distal sigmoid = the MC reason for recurrence. Recurrence after elective resection 3-13%

IMA:

- PRESERVE it when CA is not suspected – better bowel function, leak rate lower or equal
- high ligation ONLY when malignancy cannot be excluded

Splenic flexure: individualized, not routine. Mobilization a/w more superficial SSI (10.6% vs 8.4%)

Ureteral stents: SELECTIVE. Routine use adds OR time, LOS and cost with unproven protection

Leak test: air insufflation under saline. A positive test flags a high-risk anastomosis – repair, revise, retest. RCT: testing reduced clinical and radiologic leaks

RIGHT-SIDED DIVERTICULITIS

TRUE diverticula – all layers – unlike the false diverticula of the left

More common in Asia, presents younger, often mistaken for appendicitis

Less often complicated. Management otherwise the SAME as left-sided

- take the appendix regardless, to prevent future confusion

39 BENIGN COLORECTAL DISEASE: LARGE BOWEL

OBSTRUCTION

PATHOPHYSIOLOGY & WORKUP

Ileocecal valve competent in ~75% → CLOSED LOOP, cannot decompress retrograde

Laplace: pressure needed to distend is inversely proportional to radius → the CECUM distends and perforates first

- villous necrosis within 4 hours of ischemia, then transmural infarct and perforation
- ACUITY of distention matters more than absolute cecal diameter – a chronically obstructed cecum accommodates

Partial vs complete (no gas or fluid passes); simple vs complicated (visceral blood flow compromised)

Differential: intraluminal (intussusception, stercoral, foreign body, bezoar, gallstone), intramural (CA, diverticular, IBD, radiation, anastomotic stricture, hematoma), extrinsic (mass, abscess, hernia, endometriosis, pregnancy, pseudo-obstruction, volvulus)

Cessation of flatus 90%, of feces 80.6%, distention 65%.

Vomiting is LATE compared to SBO

Ischemia: constant pain, fever, tachycardia, peritonitis – but often insidious. Low pH, low bicarb, ↑ lactate support it; normal values do NOT exclude it

Normal colon 3-8 cm. Dilated = cecum > ~9 cm, remainder > ~6 cm

AXR 84% sens / 72% spec. CT 96% / 93%, and for perforation 95% / 90% vs AXR 53% / 53%

- pneumatosis suggests but is NOT pathognomonic for ischemia. Free air or portal venous gas is worse

Water-soluble enema 96% / 98% for LBO but rarely gives the etiology

1/3 of clinically diagnosed LBO have NO mechanical obstruction. 20% of presumed pseudo-obstruction ARE mechanical

Initial mgmt in every case: NPO, NGT, resuscitate with electrolyte repletion, follow urine output

MALIGNANT LARGE BOWEL OBSTRUCTION

Up to 20% of CRC obstructs. CRC accounts for 50-60% of all LBO

Emergency presentation carries 3-5x the 30- and 90-day mortality of an elective one

MC obstructing sites: sigmoid & splenic flexure. MC perforation site: CECUM. Synchronous lesions 2-7%

Perforation:

- 2.6-12% of CRC. At the tumor from necrosis, or proximal from dilation – DIASTATIC, usually cecal
- right tumor: oncologic right hemi + ileocolic anastomosis, leak 3-15%. Ileostomy is the safe play
- left tumor + local perforation: oncologic resection + end colostomy, if the proximal colon is viable
- left tumor + PROXIMAL perforation or questionable viability: TOTAL ABDOMINAL COLECTOMY

- unstable: damage control – washout, drains, ± ostomy, open abdomen, come back

Right-sided (cecum to distal transverse):

- 30-40% of malignant LBO. Resection + anastomosis is traditional
- modern audit of 776 pts – 92% resected, 82% anastomosed – mortality 10%, LEAK 14%. Temper the perceived safety
- no data to select who needs an ostomy. Intraoperative judgment of blood supply and tissue quality

Left-sided (splenic flexure to rectosigmoid):

- RCT and Cochrane: defunctioning loop colostomy alone vs primary resection – no difference in morbidity or OS
- so RESECT when feasible; reserve loop colostomy for the very frail or unresectable
- Hartmann's remains the most common operation performed
- on-table lavage vs decompression alone (RCT, 49 pts): lavage costs 31 vs 13 min; leak 0/24 vs 2/25, NOT significant
- primary anastomosis leak 2-12% in series, comparable to elective left-sided (2-8%) – confounded, surgeons anastomose when everything looks good
- Kube, 743 pts: no leak or reoperation difference b/n PA with vs without a diverting loop ileostomy
- ACPGBI predictors of in-hospital mortality: age, ASA, operative urgency, cancer stage

Segmental vs subtotal colectomy:

- subtotal for proximal ischemia, an unreparable cecal serosal tear, or a synchronous lesion
- SCOTIA RCT (91 pts): NO difference in leak or mortality, but ≥ 3 BMs/day in 41% of STC vs 9% of segmental, persisting at 4 months
- segmental ± on-table lavage is therefore PREFERRED where the colon allows

STENTING (SEMS)

Two roles: BRIDGE to elective resection, or PALLIATION of unresectable dz

- Stent-In 2: stopped early for 13% PERFORATION in the stent arm; 17% could not pass a guidewire
- Pirlet: stopped – ostomy rate not reduced, because deployment kept failing
- Alcantara: stopped for excess morbidity in the SURGERY arm
- Arezzo meta-analysis, 8 RCTs / 497 pts: 60-d mortality identical (9.6% vs 9.9%), temporary OSTOMY 33.9% vs 51.4%
- CREST, 246 pts, largest: 30-d mortality 5.3% vs 4.4% NS; ostomy 45% vs 69%
- the benefit is the STOMA. The risk is PERFORATION and the tumor cell dissemination it may cause
- outcome tracks the endoscopist – advanced skill is the limiting factor

Covered vs uncovered:

- UNCOVERED: less tumor overgrowth (RR 0.29), less migration (RR 0.29), less reinsertion (RR 0.38)
- no difference in deployment success, perforation, or bleeding

Obstructing rectal cancer:

- rare, and usually locally advanced. AVOID emergency resection – it wrecks the oncologic result
- decompress → stage → neoadjuvant → TME
- SEMS in the extraperitoneal rectum: migration and tenesmus
- loop COLOSTOMY decompresses but may damage the marginal supply for a later restorative operation; loop ileostomy will NOT decompress a competent ileocecal valve

Unresectable at presentation: 10-15%. Diverting ostomy or SEMS to palliate

ACUTE COLONIC PSEUDO-OBSTRUCTION (OGILVIE'S)

Parasympathetic withdrawal / sympathetic excess at the SPLENIC FLEXURE – the midgut-hindgut innervation transition. Dilated proximal, decompressed distal

~100 per 100,000 admissions. Rarely a primary diagnosis

- elderly, hospitalized, comorbid – acute illness, non-operative trauma, metabolic derangement, recent surgery incl. C-section
- drugs: opioids, CCBs, psychotropics

Criteria: cecum $\geq$ ~9 cm or other segments $\geq$ ~6 cm.

MUST exclude mechanical obstruction – water-soluble enema if CT is unclear. Consider toxic megacolon

Perforation risk rises past ~10 cm cecal diameter and past ~6 days of distention. Mortality 10%, 30-40% w/ perforation

Supportive – FIRST LINE, resolves 77-96%:

- bowel rest, stop the offending drugs, correct electrolytes, ambulate & reposition, NGT and rectal tube
- oral laxatives are CONTRAINDICATED – they raise intraluminal pressure
- escalate if no improvement by 48-72 h, or cecum ~12 cm

Neostigmine:

- 2 mg IV over 3-5 min. Effective in 85-94% in placebo-controlled RCTs. Off-label
- decompresses in 10-30 min, half-life ~60 min. Redosing works in 40-100%
- MONITORED setting, atropine at the bedside. Glycopyrrolate blunts bronchospasm & hypersalivation
- contraindicated in mechanical bowel or urinary obstruction. Caution: bradycardia, asthma, renal failure, PUD, recent MI
- failure predicted by male sex, younger age, post-op status, electrolyte imbalance
- daily polyethylene glycol afterward ↓ recurrence. SQ and infusion protocols work with fewer side effects

- refractory: pyridostigmine, methylnaltrexone, metoclopramide, erythromycin

Colonoscopic decompression – SECOND LINE:

- success up to 95%, but neostigmine is cheaper and safer – reserve the scope
- water immersion or minimal CO₂, NOT ambient air. Non-narcotic sedation
- goal is the ASCENDING colon, not the cecum, then deploy a long decompression tube
- repeat scope needed in up to 40% if no tube is left. Resolution usually within 48 h

Surgery – LAST:

- mortality escalates with each step (NIS): medical 7.3%, colonoscopy 9.0%, surgery 12.3%. Operative mortality reported ~30%, up to 60%
- viable colon: explore, decompress, vent through a colostomy. Colectomy is of questionable value
- ischemic or perforated: resect, usually end stoma

COLONIC VOLVULUS

10-15% of LBO in the US, 3-5% of all bowel obstruction.

Up to 40% in the “volvulus belt” – Africa, Middle East, India, Brazil, South America

Incidence: Sigmoid ~60%, Cecal ~40%. Transverse & splenic flexure rare

Needs a redundant segment on a NARROW mesentery.

Also constipation/dysmotility, prior abdominal surgery, prior volvulus

Sigmoid twists COUNTERCLOCKWISE, cecal CLOCKWISE

CT w/ multiplanar reconstruction: ~100% sensitive, > 90% specific – has supplanted plain films and enemas

- coffee bean / bent inner tube; northern exposure sign (loop apex cranial to the transverse colon); bird's beak on enema; whirl sign on CT

Emergency presentation mortality up to 33%. Ischemia & peritonitis are the drivers

SIGMOID VOLVULUS

Male, > 70, African American, diabetic, institutionalized, on psychotropics

Shrinking mesosigmoiditis: scarring patches and bands from a previous volvulus that resolved

- $\geq 180^\circ$ to be clinically significant, 360° = closed loop
- 3 patterns of necrosis: at the neck of the volvulus; anywhere in the closed loop; proximal descending colon from retrograde mesenteric thrombosis

Nonoperative detorsion – for the stable pt without peritonitis:

- rigid proctoscopy at the bedside, left lateral, no sedation. PINWHEEL mucosa. Insufflate, untwist, intubate the segment, open the window and vent
- flexible sigmoidoscopy if the twist is beyond proctoscope reach
- success 60-95%. ~3% mortality – reflects the population, not the procedure

- INSPECT THE MUCOSA. Ischemia → colectomy
- leave a transrectal tube – a semi-rigid chest tube sutured to the perianal skin – to STENT the segment, not to vent (it clogs)
- post-detorsion film for tube position, decompression, and absence of free air
- scope the whole colon before resecting, to exclude a neoplasm

Resection:

- recurrence after a first admission 45-70%, most within a few months → SIGMOIDECTOMY, semi-elective, ideally the same admission
- individualize – this population is elderly and comorbid
- redundancy elsewhere is NOT by itself an indication to extend the resection
- extend to SUBTOTAL for cecal perforation, patchy proximal ischemia, or megacolon
- GANGRENOUS LOOP: DO NOT DETORSE. Resect it twisted – avoids the potassium, bacterial and endotoxin load, and avoids perforating it
- laparoscopy is hard – the loop stays dilated and prolapses out of a short midline anyway. Splenic flexure rarely needs release
- ligate the main pedicle rather than nibbling the mesentery; a mid-mesenteric plane keeps you off the retroperitoneum and ureter
- wide colonic end – staple it closed and do a side-to-end Baker anastomosis

Non-resectional options – all with much higher recurrence:

- mesosigmoidoplasty: incise the elongated mesentery vertically, close transversely – shortens and broadens it
- simple operative detorsion, sigmoidopexy

PEC (percutaneous endoscopic colostomy): for the prohibitive-risk pt, under conscious sedation, ± laparoscopic guidance. 1 vs 2 points of fixation unsettled; early device removal a/w recurrence. Complications from site infection to peritonitis from migration

CECAL VOLVULUS

Younger, more often FEMALE

Cecal volvulus: axial torsion of ileum and colon – a closed loop plus an SBO

Cecal bascule: caput folds anterocephalad over the ascending colon, NO axial torsion

- congenital lack of fixation; prior surgery is a risk factor
- coffee bean toward the LUQ; bird's beak on contrast

NONOPERATIVE DETORSION RARELY WORKS – a surgical emergency even without sepsis

Rx: ILEOLECTOMY with anastomosis. End ileostomy ± mucous fistula if anastomosis is too risky

- unstable: resect, leave in discontinuity, second look
- cecopexy & cecostomy: 12-14% recurrence. Resection ~0%

- extraction incision is unavoidable – laparoscopy struggles with the dilated displaced colon

Mobile cecum syndrome: absent lateral peritoneal attachments. "Pre-volvulus" is NOT an established entity – do NOT operate for it

TRANSVERSE & SPLENIC FLEXURE VOLVULUS

Transverse: 1-4% of volvuli

- constipation, laxative use, congenital
- plain film rarely diagnostic; enema shows a bird's beak at the distal T-colon
- Chilaiditi sign: hepatodiaphragmatic interposition of bowel – historical
- Rx: segmental transverse colectomy or extended right hemi

Splenic flexure: rarest, < 50 case reports, young women

- 3 ligaments normally fix it – gastrocolic, splenocolic, phrenocolic. 2/3 had prior surgery
- film: air to the splenic flexure, air-fluid levels in cecum & T-colon, empty descending colon
- Rx: segmental or extended left hemi. May need ileosigmoid or ileorectal if the colon is very dilated

ILEOSIGMOID KNOTTING

Rare in the US, seen in the volvulus belt. Men, mostly < 50. A single large meal

Ileum around sigmoid or vice versa – DOUBLE LOOP obstruction

Fulminant. Shock, gangrene in 70-100%, mortality up to 73%

Suspect it when sigmoid volvulus + SBO cannot be reduced endoscopically

Rx: vascular control BEFORE detorsion. Usually a double resection; segmental sigmoid to prevent retrograde mesenteric thrombosis

RARE CAUSES

Intussusception: 1-3% of adult mechanical obstruction, lead point found in 80%. Malignant in 65-87%. CT target/sausage sign. Resect oncologically – do NOT reduce it endoscopically

Hernia: rare for colon – femoral, inguinal, umbilical, incisional, Spigelian, lumbar, diaphragmatic

Endometriosis: < 1% of obstruction. Rectosigmoid is the MC intestinal site

Other malignancy: ovarian, gastric, pancreatic, bladder. Lymphoma < 0.5% of colorectal malignancy

Infectious: TB, schistosomiasis, helminths

40 BENIGN COLORECTAL DISEASE: LOWER GI

HEMORRHAGE

DEFINITION & EPIDEMIOLOGY

Bleeding distal to the ligament of Treitz

30-40% of all GI bleeds – MC cause of GI-related hospitalization in the US

Incidence 33-87/100,000; acute colonic bleeding 36/100,000

Acute LGIB = sudden hematochezia – 20% of all GI bleeds worked up

Chronic = slow loss → Fe deficiency anemia, + FOBT

Mortality 1.3-3.9% – usually 2/2 comorbidities, not exsanguination

UGI bleed is DECREASING, LGIB is INCREASING – 22% ↑ b/n 2000-2009

- aging population + anticoagulants
 - median age 74; > 80 yo have a 200-fold ↑ risk vs. 20 yo
 - 75% of pts are on a predisposing medication
- ~15% of pts w/ hematochezia have an UPPER GI source
80% stop spontaneously

ETIOLOGY

Anatomic:

- Diverticulosis 30-65% – MC cause, and MC cause of SEVERE acute hemorrhage
- vasa recta penetrate the wall, mucosa apposed to the vessel
- 50% of the population has sigmoid/descending diverticulosis
- cumulative bleeding 2% at 5 yrs, 10% at 10 yrs
- Anorectal 5-20% – 2nd MC. Hemorrhoids & fissure

Vascular: ischemic colitis 5-20%, angioectasia 5-10%

Inflammatory: IBD 3-5%, infectious colitis 2-5%

Neoplastic: 3-11%

Other: post-polypectomy 2-7%, radiation proctitis 0-2%,

NSAID ulcer 0-2%, Dieulafoy 0-2%

Small bowel: 3-13% – Meckel's, IBD, angioectasia, neoplasm, NSAID ulcer, Dieulafoy

Angioectasia: age > 70, RIGHT colon, a/w multiple lesions, comorbidities, anticoagulation

Heyde syndrome: angioectasia + aortic stenosis – consider AVR

PRESENTATION & INITIAL EVALUATION

Hx: color, amount, frequency, duration. Wt loss, pain, diarrhea, obstipation

- meds – NSAIDs, antiplatelets, anticoagulants
- prior XRT, recent endoscopy, vascular surgery, liver dz, IBD, family Hx

Melena → UGI source until proven otherwise

Hematochezia + instability → also think UGI – brisk bleed transits too fast to oxidize

Vitals w/ postural changes, abd exam, DRE

NGT lavage – quickly excludes a gastric source

ANOSCOPY IS MANDATORY – hemorrhoidal source in < 20%

- hemorrhoidal bleeding causing instability is rare – keep looking

RISK STRATIFICATION

Shock Index (HR / SBP):

- SI > 1 = unstable, predicts active bleeding & extravasation on CTA
- a rising SI is a/w mortality

Risk factors for severe bleed: instability, ongoing bleeding, comorbidities, age > 60, Hx diverticulosis or angioectasia, ↑ Cr, anemia

- risk rises with the NUMBER of factors

Velayos:

- instability > 1 hr + Hct < 35% + active gross rectal bleeding → 79% ongoing/recurrent bleed

Oakland Score:

- the ONLY score designed and externally validated for LGIB
- 7 variables: age, gender, prior LGIB admission, blood on DRE, HR, SBP, Hgb
- ≤ 8 → 95% probability of safe discharge – manage as OUTPATIENT
- validated in the UK only, likely underreports who can go home

Others: BLEED (inpatient morbidity), NOBLADS (0 = 0% → 5 = 92.9% severe bleed), Strate (0 low / 1-3 intermediate / > 3 high), Sengupta (30-day mortality, 2.6% → 26% by quartile)

RESUSCITATION & ANTICOAGULATION

Goal = normalize hemodynamics. No difference colloid vs. crystalloid

Transfuse to Hgb 7 – or 9 if significant cardiovascular dz

- extrapolated from UGI bleed RCTs, no LGIB trials exist
- Platelets < 50 x 10⁹/L → transfuse. Massive transfusion → trauma MTP ratios

Warfarin:

- ACG: reverse for INR > 2.5
- INR > 1.5 predicts MORTALITY but NOT rebleeding – a marker of comorbidity

DOACs (rivaroxaban, dabigatran, apixaban):

- OR 1.45 for bleeding – meta-analysis of 43 RCTs
- degree of anticoagulation NOT reflected in the coag panel
- half-lives 5-19 hrs. aPCC if life-threatening
- no high-level evidence for any reversal agent – consult heme

Balance reversal against the thrombotic indication – call the prescriber

ENDOSCOPY

PREFERRED initial modality – diagnostic AND therapeutic. Yield 42-90%

Timing: within 24 hrs in high-risk pts (ACG)

- bowel prep recommended, NGT to facilitate
- intubate the terminal ileum to exclude a small bowel source
- water-jet irrigation + large working channel. CO₂ insufflation
- TATTOO the site in case of rebleed

Urgent (< 8 hrs) vs. standard: better definitive diagnosis (42% vs. 22%, OR 2.6) – NO difference in rebleeding, surgery, or LOS

- *Laine, < 12 hrs vs. 30-60 hrs: no difference in anything*
- *earlier scope buys diagnosis, not outcomes*

High-risk stigmata: active bleeding, non-bleeding visible vessel, adherent clot

Technique:

- injection, contact & non-contact thermal, topical sprays/powders, clips
- adverse events 0.3-1.3%
- best benefit: diverticular, angioectasia, post-polypectomy
- ischemia, IBD, neoplasm – NOT durably treatable endoscopically

Diverticular Bleed:

- Strate, 847 pts: 80% hemostasis. Early rebleed 8%, late 12%
- monotherapy = combination therapy
- CLIPS PREFERRED over cautery – thin wall, delayed perforation
- epinephrine first to slow it, then clip the vessel or close the diverticulum
- cap-assisted eversion, or endoscopic band ligation (left colon)

Angioectasia: APC is the treatment of CHOICE

Post-Polypectomy:

- 95-100% success. CLIPS PREFERRED – avoid more energy
- occurs in 0.6-9.7%. Risk: polyp > 2 cm, thick stalk, right colon, anticoagulation

RADIOLOGIC EVALUATION

CT Angiography:

- FIRST radiologic test – fast, no prep, universally available
- 3 phases: non-contrast, arterial, portal venous
- detects 0.25-3 mL/min. Sens 85.2%, spec 92.1%
- critical finding = active extravasation into lumen. Subtle = clot on MIP images
- fluid-distended bowel dilutes contrast → false negative
- select with SI ≥ 1, or ≥ 5 U PRBC in 24 hrs
- NEGATIVE CTA in a stable pt = good prognosis, 77.4% will not rebleed
- watch contrast nephropathy

Tagged RBC Scintigraphy:

- historic gold standard – MOST SENSITIVE, detects 0.04-0.05 mL/min

- 99mTc-labeled RBC superior to 99mTc-sulfur colloid
- sulfur colloid: immediate, no labeling – but short half-life, no delayed imaging
- labeled RBC images up to 24 hrs, can RE-SCAN if the first is negative
- needs a relatively stable pt – takes time to label and reinject
- MISLOCALIZES in 10-25% – do NOT choose a resection off this scan
- 55% false-negative rate
- role now = intermittent or too-slow-for-CTA bleeding. CTA has displaced it before angio

Catheter Angiography:

- the ONLY radiologic modality that also TREATS
- needs ≥ 0.5 mL/min and CONTINUOUS bleeding
- best yield if unstable, > 5 U PRBC in 24 hrs, SBP < 90
- a negative angiogram after a positive CTA is common
- superselective embolization to arteries < 1 mm – microcoils, gelfoam, cyanoacrylate, PVA
- technical success 73-100%, clinical success 63-96%, rebleed 11-50%
- major ischemic complications < 3% – self-limited pain / lactate rise is common
- provocative angio (heparin, nitroglycerin, urokinase, tPA) – EXPERIENCED CENTERS ONLY
- if it fails to control, it still directs the resection

RECURRENT LGIB

Early rebleed (before discharge) 22%; late 16%

Readmission 13.7% at 14 days, 19% at 1 yr

Risk: anticoagulation, comorbidities, malignancy, non-ASA antiplatelet, DAPT, age > 65, diverticular index bleed

REPEAT evaluation has ADDITIVE yield – repeat scope yield 3-60%

- no prior localization: repeat upper + lower endoscopy yields 40-65%

Prevention: stop NSAIDs, stop ASA used for primary prevention

Uncontrolled but localized → angio + embolization

Early rebleed + prior localization → resection

OBSCURE & SMALL BOWEL BLEEDING

Obscure = unlocalized after appropriate endoscopic and radiologic w/u

Most obscure sources are SMALL BOWEL – “suspected small bowel bleeding”

Causes: IBD, angioectasia, neoplasm, NSAID ulcers, Dieulafoy

Only after SECOND-LOOK endoscopy – colonoscopy to the ileum, EGD w/ push enteroscopy to distal duodenum & jejunum

Capsule Endoscopy:

- yield 38-83%, complete small bowel in 70-90%
- changes management in 37-87%. PPV 94-97%, NPV 83-100%

- higher yield if Hgb < 10, bleeding > 6 months, > 1 episode, overt bleeding, CE w/in 2 wks
- NO therapy, poor precise localization
- repeat CE is diagnostic if Hgb drops > 4 g/dL (Viazis)

Double Balloon Enteroscopy:

- reaches 240-360 cm distal to the pylorus
- diagnostic yield 60-80%, therapeutic 40-73%
- perforation/pancreatitis 1.2%. SBE equivalent to DBE
- CE FIRST to select who needs it – but a negative CE does not exclude if suspicion is high

SURGERY

Resection required in only 7-25% of LGIB

Indications: failed endoscopic + radiologic control, instability, transfusion threshold reached

ALL attempts at localization MUST be made first in the stable pt

- historic threshold > 6-7 units PRBC
- < 10 units transfused: 7% mortality; > 10 units: 27%

Run the entire bowel – palpate stomach, small bowel, colon for masses

Intraoperative colonoscopy / enteroscopy – oral, rectal, or via enterotomy

- yield 50-88%, but rebleeding 60%

Segmental Resection:

- ONLY when the source is CONFIDENTLY identified
- better function than subtotal. Rebleed 4-14%

Subtotal Colectomy:

- for colonic bleeding that stays UNLOCALIZED, small bowel excluded
- rebleed ~4%
- anastomosis vs. end ileostomy per hemodynamics
- in a stable pt consider a temporary loop ileostomy to sort small vs. large bowel

NEVER do a blind SEGMENTAL resection – localize it or take the whole colon

Malignant source → oncologic resection. Rectal CA → consider urgent XRT for hemostasis

ALGORITHM

Hematochezia → assess, stabilize, labs, ANOSCOPY

Shock index + risk score → low risk → outpatient

Bloody NGT lavage or suspicion of UGI → EGD first

Stable → prep → COLONOSCOPY → endoscopic treatment + tattoo

Unstable, or scope non-diagnostic → CTA (tagged RBC if intermittent/slow) → angio + embolization

Still bleeding, colonic, unlocalized → SUBTOTAL COLECTOMY

Small bowel suspected → capsule → enteroscopy → targeted resection

41 BENIGN COLORECTAL DISEASE: ENDOMETRIOSIS

DEFINITION & EPIDEMIOLOGY

Viable endometrial-like glands & stroma OUTSIDE the uterine cavity

Cyclical pain/bleeding from any site that coincides w/ menses

Up to 15% of reproductive-age women – ~200 million worldwide

- 6.2% of premenopausal women (Houston, only population-based study)
- infertility 25-50%, chronic pelvic pain 71-87%
- \$69.4 billion/yr; direct cost > \$12,000/pt/yr – on par w/ DM, RA, IBD
- 50% of working women lose ~18 days/yr

No racial predilection except JAPANESE – double the Caucasian rate

Uninterrupted menstrual cycles predispose

- ↓ w/ OCPs and multiple pregnancies; ↑ w/ time since last childbirth
- now seen postmenopausal 2/2 exogenous estrogen & obesity
- progressive dz in primate studies, though spontaneous regression occurs

Diagnostic Delay: 7-12 yrs from symptom onset – THE central problem

- pts wait > 2 yrs before making an appointment
- ~7 PCP visits before referral; 3/4 are MISDIAGNOSED
- half of PCPs can't name 3 main symptoms

ETIOLOGY

Precise cause unknown. Familial component. 2 classic theories:

- (1) *Coelomic Metaplasia (Meyers): coelomic epithelium undergoes metaplasia into endometrial tissue*
 - explains dz in men and non-menstruating women
 - FAILS to explain the pelvic distribution, and it should worsen w/ age – it doesn't
- (2) *Retrograde Menstruation (Sampson, 1921): MOST PLAUSIBLE – reflux through the tubes into the peritoneum*
 - viable tissue shown in menstrual effluent; induced in primates and in volunteers
 - but retrograde flow occurs in virtually ALL women and only a minority get the dz

Genetic:

- Simpson: 7% relative risk in blood relatives vs. 1% controls, and more severe dz
- polygenic, or genetic + environmental

Immune (Dmowski):

- depressed cellular immunity – ↑ activated macrophages, ↓ NK cell function
- humoral: autoantibodies to cellular components
- present in peritoneum AND systemic circulation → may be a systemic dz

Probably a GROUP of disorders – superficial peritoneal, ovarian endometrioma, deep infiltrating, and extrapelvic all behave and respond differently
Lesion size & number correlate POORLY w/ symptoms – except dyspareunia in rectovaginal septal dz

CLINICAL MANIFESTATIONS

Triad: pelvic pain, pelvic mass, infertility

Pain:

- deep, constant, dull. Before or during menses
- dyspareunia w/ DEEP penetration, dyschezia, pain w/ rectal filling
- pain on palpation of the uterosacral ligaments at rectovaginal exam
- teens w/ pelvic pain, worsening dysmenorrhea, rectal pain, dyspareunia → work up

Sites:

- Common: ovaries, uterosacral ligaments, cul-de-sac, uterus, rectosigmoid
- Less common: appendix, ureter, bladder, vagina, cervix, rectovaginal septum, cecum, ileum, inguinal canal, perineal & C-section wounds
- Rare: diaphragm, lung, liver, brain

Thoracic endometriosis syndrome: catamenial

pneumothorax, perimenstrual hemoptysis, dyspnea, chest pain

Brain: perimenstrual headache & seizures

Bladder: dysuria, cyclic hematuria – cystoscope DURING menses. Ureter → obstruction & renal loss, rarely bilateral

GI Involvement:

- rectosigmoid MC, then small bowel, then appendix
- change in BM, ↓ caliber, tenesmus, dyschezia, bleeding around menses
- large/deep lesions → obstruction, perforation, bleeding
- do NOT resect an asymptomatic pt – benign natural history

Endometrioma:

- fluid-filled endometriotic cyst, MC in the ovary. "Chocolate cyst"
- occurs in 17-44% of cases; can grow large and stay silent

"Kissing ovaries" – bilateral endometriomas fused at the midline. PATHOGNOMONIC

Infertility:

- MC presenting complaint. Up to 30% of women w/ endometriosis
- Extensive dz: tubo-ovarian adhesions blocking ovum pickup, blocked tube, ovarian destruction
- Mild dz: anovulation, abnormal LH surge, LUFS, luteal phase inadequacy, hyperprolactinemia, immune dysfunction, altered peritoneal fluid (prostaglandins, macrophages), spontaneous abortion

ENDO study: infertility Hx ↑ odds (AOR 2.43).

Dysmenorrhea AOR 2.46, pelvic pain AOR 3.67.

Gravidity, parity, and BMI all ↓ the odds

DIAGNOSIS

High index of suspicion – there is NO validated noninvasive test

Serum markers:

- CA-125: poor sens & spec. Better for surveillance than diagnosis
- investigational: CA-125 + prolactin (77/88), follistatin (92/96), IL-6, IL-8, TNF- α , CRP, miRNA, cell-free DNA – none validated

Pelvic Exam: irregular or painful nodule, uterosacral induration, cul-de-sac nodularity

Transvaginal U/S:

- best for ovarian endometrioma. Hypoechoic mass in the rectovaginal septum
- direct rectal involvement: sens 26.7%, spec 85.7%, accuracy 52.7% – POOR
- *Negative Sliding Sign: cervix/uterus does not slide freely against rectosigmoid → pouch of Douglas obliteration. Sens 83.3%, spec 97.1% (Reid & Condous)*
- far better than palpating a nodule (sens 24.4%)
- predicts longer, harder surgery – get it PREOPERATIVELY

MRI: sens 73.3%, spec 92.9%, accuracy 82%

Endorectal U/S: sens 86.7%, spec 85.7%, accuracy 86.2% – best for deep rectal wall involvement

Colonoscopy:

- role is to EXCLUDE other causes – findings suggestive in only 4%, misses dz in > 92%
- only diagnostic when mucosa is involved
- polypoid mass is the MC finding; MC preop diagnosis is COLORECTAL CA
- mimics IBD, solitary rectal ulcer, cancer
- IHC panel: CK7 & ER (ectopic glands), CD10 & ER (stroma) vs. CK20 & CDX2 (rectal glands)
- ciliated (tubal) metaplasia is the MC metaplastic change – intestinal glands have NO cilia

Laparoscopy: GOLD STANDARD – direct visualization + histology

- endometrial glands & stroma w/ inflammation and fibrosis = definitive
- inspect: ovaries, both cul-de-sacs, broad & round & uterosacral ligaments, tubes, uterus, sigmoid, appendix, ureters, small bowel
- lesions red, brown, black, white, clear, yellow, blue
- “Powder burn” blue-tinted implants – highly suspicious
- subtle forms: fibrotic peritoneal patches, convalescence of small vessels on the side wall
- narrowband imaging adds 86% predictive value ONLY if white light is already positive – 0% if white light is negative

CLASSIFICATION

Deep Infiltrating Endometriosis (DIE): invasion > 5 mm beyond the peritoneum. Posterior cul-de-sac, uterosacrals, rectosigmoid

r-ASRM (revised American Society for Reproductive Medicine):

- MOST WIDELY USED. Scores peritoneum, ovaries, tubes, cul-de-sac
- size & depth of lesions, adnexal adhesions, severity of cul-de-sac obliteration
- Stage I minimal 1-5 / II mild 6-15 / III moderate 16-39 / IV severe ≥ 40
- Symptomatic Stage IV REQUIRES surgery

ENZIAN: for DIE. Pelvis in 3 compartments A, B, C + F (“far” – bladder, ureter, bowel), each graded by depth

Endometriosis Fertility Index (EFI): historical factors (age, yrs infertile, prior pregnancy) + surgical factors (Least Function score, AFS scores) – predicts pregnancy after surgical staging

NONE of these predict pain, response to medication, recurrence, or QOL

ENDOMETRIOSIS & CANCER

Benign dz, but ↑ risk of Type I ovarian CA – CLEAR CELL and ENDOMETRIOID

- Saavalainen: SIR 1.76 overall for ovarian CA
- endometrioid 3.12, clear cell 5.17, serous 1.37
- OVARIAN subtype of endometriosis is the driver – EOC 4.72, CCOC 10.1
- peritoneal subtype → endometrioid only (2.03). DIE: NO association
- NO ↑ in endometrial, vaginal, or vulvar CA

Barreta: endometriosis found in 80% of CCOC/EOC. 42% nulliparous, 42% premenopausal

- CA-125 elevated in early and late stage
- endometriosis-associated CCOC presents EARLIER and does well; late-stage CCOC recurs early w/ short OS

MEDICAL MANAGEMENT

FIRST LINE. Goal = pain control + arrest growth of implants

Pathophysiology targeted: ↑ estradiol, ↑ intrinsic aromatase activity, ↑ inflammatory markers, PROGESTERONE RESISTANCE

NSAIDs: ↓ inflammatory mediators

OCPs: hormone suppression. VTE risk – avoid in older pts, migraine w/ aura, lupus, HTN, liver dz

GnRH agonist/antagonist: blocks ovarian estrogen production

- neoadjuvant 3-6 months ↓ burden and allows a more localized resection
- suppresses ovulation – limited use if fertility desired
- menopausal symptoms, bone loss

Progestins: inhibit angiogenesis, estrogen-induced mitosis, alter estrogen receptors

Aromatase inhibitors: ↓ peripheral conversion of steroid precursors in adipose tissue

Will NOT cure – relapse once stopped

Recurrence after surgery should be managed MEDICALLY first

- Vignali: pain 20.5% at 3 yrs / 43.5% at 5 yrs; clinical 9% and 28%
- COMPLETENESS OF THE FIRST OPERATION is the prognostic factor
- other risks: younger age, laterality, rAFS stage & score > 70, cyst size, high preop pain, absence of pregnancy, prior medical treatment

SURGICAL MANAGEMENT

LAPAROSCOPIC approach is PREFERRED

Goal: excise/ablate ALL implants, preserve ovarian function, optimize fertility

- incomplete excision → persistent symptoms and no fertility benefit
- implants run deeper than they look – ureteral stents advised

MULTIDISCIPLINARY w/ gynecology ± urology for advanced dz

Ovarian:

- Cystectomy PREFERRED in the young pt – preserves ovarian function
- Oophorectomy if older or the cyst may be malignant
- cystectomy still feasible in large multiloculated cysts
- contralateral recurrence up to 24.75% at 5 yrs after unilateral oophorectomy (Hidari)

Superficial peritoneal: excise or ablate – cautery, harmonic, laser

Shaving: nodule excision WITHOUT entering the bowel lumen

Disc Excision: full-thickness excision of the bowel wall

- lesion < 3 cm, UNIFOCAAL, < 60% of the circumference

Segmental Resection:

- for more extensive wall involvement – LAR w/ diverting stoma
- if rectosigmoid MUCOSA or submucosa is involved, > 40% of the circumference is already involved
- margins: grossly normal bowel

Appendix: appendectomy. Small bowel: wedge or resection

Complications:

- major (needing reintervention): 2.9-8.4% for DIE WITHOUT bowel resection, 7.4-25% WITH
- rectovaginal fistula, pelvic abscess, anastomotic leak, stricture
- anastomotic stenosis – endoscopic dilation is a valid first option
- protective ileostomy is a MODIFIABLE risk factor for stenosis

Lermann: sexual function improved to control levels after surgery WITHOUT bowel resection – the bowel resection group did NOT improve and scored worse than controls. Counsel accordingly

Recurrence:

- 6-67% depending on the definition – relapse of pain, no fertility improvement, or re-visualized lesions

42 BENIGN COLORECTAL DISEASE: TRAUMA OF THE

COLON AND RECTUM

HISTORICAL ARC

Pre-WWI: nonoperative, mortality ~100%. Laparotomy was CONDEMNED. WWII: laparotomy + primary repair
Ogilvie (N. Africa, 1942): divert everything – the US Surgeon General made proximal diversion POLICY
Korea/Vietnam: anatomic – right-sided anastomosis, left-sided colostomy. Rectal injuries got washout + diversion → the “4 Ds”
Iraq/Afghanistan: primary repair or anastomosis for the MAJORITY – but ~1/3 still diverted
Mortality 60% in WWI → < 10% now. Modern civilian colon-related mortality 1.3%. Abdominal sepsis still 16-33%

COLON TRAUMA – EPIDEMIOLOGY & DIAGNOSIS

MC mechanism PENETRATING – GSW > stab > impalement

Colon = 2nd MC injured organ in penetrating abdominal trauma (after small bowel)

Blunt: < 10% of injuries found at laparotomy

- lap belt WITHOUT shoulder harness ↑ visceral injury
- most are serosal tears/hematoma – but 1/3 are FULL THICKNESS
- rapid deceleration → mesenteric tear & ischemic necrosis
- blast hits HOLLOW viscus more than solid, colon most susceptible – may have no external signs

Anterior GSW crossing the midline or A → P: highest suspicion

Trans-pelvic or perineal GSW = RECTAL INJURY until proven otherwise

Flank & back wounds → colon injury w/o peritonitis or instability

- triple-contrast CT sens 90%, spec 96% – but standard IV-contrast CT is usually adequate and avoids delay

Blunt: CT is the study of choice – free air, unexplained free fluid, thickened colon wall

- free intraperitoneal AIR mandates exploration
- free FLUID w/o solid organ injury → explore or serial exams + repeat imaging

DPL has no role. Laparoscopy only in the stable pt w/ back/flank/pelvic wounds

Paracolic / Retroperitoneal Hematoma:

- penetrating → EXPLORE and evaluate the colon
- blunt → do NOT routinely explore

AAST Colon Injury Scale:

- I: contusion/hematoma w/o devascularization, or partial-thickness laceration
- II: laceration ≤ 50% of circumference
- III: laceration > 50% of circumference
- IV: transection
- V: transection with segmental tissue loss

COLON – REPAIR, RESECT, OR DIVERT

Destructive: > 50% of circumference OR devascularized → RESECT

Nondestructive: → PRIMARY REPAIR

Favors Primary Repair:

- small, single or widely spaced, clean margins after debridement, minimal mesenteric injury
- tension-free, healthy bowel, widely patent lumen, low-velocity wound, short-gut risk if resected

Favors Resection:

- multiple closely spaced injuries, inflamed or necrotic edges, large mesenteric hematoma
- edema or wall hematoma, closure would narrow the lumen > 25%
- high-velocity wound, adjacent pancreatic injury/leak, underlying CA or diverticulitis

“Bucket-handle” deformity: large mesenteric tear w/o colonic wall injury, from blunt deceleration. Bowel looks fine – RESECT IT anyway for delayed perforation and leak

THE EVIDENCE

Stone 1979 (n = 268): primary repair → TENFOLD ↓ complications, shorter stay, lower cost

Chappuis, Sasaki, Gonzalez (> 300 randomized): FEWER complications than diversion

- pooled abdominal septic complications: repair 13.1% vs. diversion 21.7%

Kamwendo 2002 RCT: primary repair superior to colostomy EVEN in high-risk pts – delay, contamination, associated injuries, shock

AAST multicenter RCT 2001 (n = 297) – DESTRUCTIVE injuries:

- 66% anastomosis, 33% diversion
- colon-related mortality 1.3%, ALL in the diversion group
- anastomotic leak 6.6% with ZERO mortality
- RFs for abdominal complications: severe fecal contamination, > 4 U PRBC in 24 hrs, inappropriate antibiotic choice
- all 3 present → 60% complication rate
- CONCLUSION: resection + anastomosis for ALL destructive injuries regardless of severity

Those risk factors raise complications REGARDLESS of whether an ostomy was made – do NOT use them as independent criteria to divert

Right vs. left is DEAD dogma – they heal equally. Left-sided injury does not mandate a colostomy

NOT risk factors / NOT contraindications: shock, massive transfusion, ISS > 15, associated abdominal injuries, any specific organ injury, time to operation, a retained missile

EAST Guidelines (updated 2018):

- LOW RISK (no shock, hemorrhage, severe contamination, or delay): repair, or resection + anastomosis

- HIGH RISK (delay > 12 hrs, shock, associated injury, > 6 U PRBC, contamination, left-sided):
CONDITIONALLY still repair or anastomose rather than mandatory colostomy – except the most severe injuries

DAMAGE CONTROL

Abbreviated laparotomy: stop bleeding, control contamination, LEAVE THE COLON IN DISCONTINUITY

Indications: acidosis, hypothermia, coagulopathy – also multiple complex injuries, questionable bowel

Decide EARLY in the case. No predetermined interval, but AVOID > 48 hrs w/ stapled-off bowel – proximal dilation, edema, fluid demand

Close the abdomen as EARLY as possible – earlier closure ↓ complications

Anastomosis after DCL:

- Weinberg 2009: ↑ complications vs. colostomy. Ott 2011: 27% enteric leak, worse w/ transfusion & left-sided
- Tatebe 2017 (3 Level I centers): DCL NOT a/w ↑ leak, fistula, SSI, or abscess despite 2/3 delayed repair
- caution if still on pressors

COLON – TECHNIQUE & PERIOPERATIVE

Prep WIDE, include the lateral abdominal wall for a possible stoma. Abandon a minilaparotomy if hemoperitoneum is found

Debride high-velocity GSW edges to healthy tissue – missiles cause THERMAL injury and closures break down

Perforations within a few cm: take down the intervening bridge and do ONE repair, or resect the segment
Single- vs. double-layer: no difference. Stapled = hand-sewn for leak, complications, function

NO colonic lavage needed even for a left-sided anastomosis

Ileocolostomy leaks LESS than colocolostomy

- overall leak 2-3%; colocolostomy 13% vs. ileocolostomy 4% in older series
- enterocutaneous fistula – most heal after percutaneous drainage
- re-explore ONLY for generalized peritonitis or failed drainage

No routine drain on an anastomosis – drain adjacent pancreas or bladder injuries widely

Antibiotics:

- give within 30-60 min of incision, redose if prolonged or ~1 blood volume transfused
- 24 HOURS ONLY – no benefit beyond, EVEN with large-volume contamination
- exception: delayed presentation w/ established intraabdominal infection

Wound: ~11% infection rate – delayed primary closure at 3-5 days

Ostomy takedown: ~15% complication rate. Can be closed early, even same admission

TIMING & DELAY

Most present within hours – a 2-8 hr delay should NOT change management

Delay > 8-12 hrs w/ fecal contamination alters anatomy AND physiology → be LIBERAL with diversion

- septic shock, staple line compromise from induration & edema, bowel distension
- mesenteric thickening/shortening limits colostomy creation

RECTAL TRAUMA

> 80% from GSW. Also impalement, iatrogenic endoscopic/urologic, foreign bodies

Military injuries more destructive – high-velocity missile and blast

Missed or delayed diagnosis drives the morbidity and mortality

Suspect With:

- penetrating buttock, groin, proximal thigh, perineum, sacrum
- ANY trans-pelvic GSW – assume rectal injury
- injury to bladder, uterus, vagina, iliac vessels
- blunt: isolated anorectal injury is rare – think “open-book” pelvic fracture, posterior/sacral disruption, straddle, perineal impalement

Diagnosis:

- gross blood on DRE is highly suggestive but NOT sensitive or specific enough to rule in or out
- Rigid or flexible sigmoidoscopy – accuracy 80-95%. GOLD STANDARD exam
- do NOT worsen the defect w/ aggressive advancement or insufflation
- full 360° inspection – a small HEMATOMA is often the only finding, a visible defect is uncommon
- triple-contrast CT (IV, oral, rectal) improves accuracy when rectal injury is suspected

AAST Rectal Injury Scale:

- I: contusion/hematoma w/o devascularization, or partial-thickness laceration
- II: laceration < 50% of circumference
- III: laceration ≥ 50% of circumference
- IV: full-thickness laceration extending into the perineum
- V: devascularized segment

Destructive = > 50% circumference or mesenteric injury compromising perfusion

Anatomy:

- upper 2/3: anterior & lateral peritonealized, POSTERIOR extraperitoneal

- distal 1/3 entirely EXTRAPERITONEAL – thick connective tissue & fat carrying the neurovascular supply
- bladder ant., sacrum & sacral venous plexus post., iliacs & ureters posterolateral
- exposure of the extraperitoneal rectum is far harder – this is why management differs

INTRAPERITONEAL RECTAL INJURY

Treat it LIKE A DISTAL LEFT COLON. Diverting ostomy is NO LONGER standard of care

Nondestructive + stable → PRIMARY REPAIR without diversion

Destructive → segmental resection, then anastomosis vs. ostomy by the same risk scaling

- low risk & select moderate → primary anastomosis
- high risk & select moderate → ostomy, or anastomosis w/ proximal protecting stoma

Resection carried to the mid or lower third → anastomosis + PROTECTING LOOP ILEOSTOMY

- reverse as early as 4-6 wks, almost always locally w/o laparotomy

AAST 2017 multicenter (758 rectal injuries, largest series): diverting colostomy had DOUBLE the complication rate of primary repair/anastomosis without diversion – 22% vs. 10%

EXTRAPERITONEAL RECTAL INJURY

Classic “4 Ds”: Direct repair, Divert, Drain (presacral), Distal washout

- very few pts need all four, or even three – the dogma has COLLAPSED

What Remains:

- PROXIMAL DIVERSION is the most important D
- direct repair ONLY if easily accessible w/o significant dissection, or if already exposed
 - proximal extraperitoneal → abdominal mobilization. Very distal → transanal
- LOOP colostomy provides adequate diversion and avoids a complicated takedown
- alternative: resect, anastomose, protect w/ loop ileostomy or a stapled END-LOOP (distal end buried subcutaneously) – full diversion and no future laparotomy

Presacral Drainage – NOT indicated:

- 1998 RCT: NO reduction in infectious complications
- AAST 2017: independent RF for abdominal complications, OR 2.6
- consider ONLY for inaccessible unrepairable injury or heavy presacral contamination, always w/ diversion
- technique: curved transverse incision posterior to the anus, blunt dissection anterior to Waldeyer’s fascia to the level of injury

Distal Rectal Washout – NOT indicated:

- AAST 2017: independent RF for abdominal complications, OR 3.4
- forceful irrigation pushes stool into uninvolved planes
- consider only for a large retained stool burden once the injury is controlled or excluded

EAST Guidelines (nondestructive extraperitoneal):

- FOR proximal diversion
- AGAINST presacral drainage and AGAINST distal rectal washout
- evidence graded “very low”

Associated injuries in 77%; bladder in 30-40% –

INTERPOSE OMENTUM, 24% develop rectovesical fistula

ANAL & IMPALEMENT INJURY

Rare outside obstetrics. Ground-level IEDs, penetrating perineal wounds, straddle, impalement, open pelvic fracture

Diagnosis is visual on the secondary survey. DRE for tone and voluntary squeeze, vaginal exam

- OBESITY hides major injuries – retract the buttocks fully
- EUA + gentle anoscopy/proctoscopy for the canal and any associated rectal injury
- get the PRE-EXISTING continence history – it drives management

Management:

- minor: local wound care, or transanal debridement + primary suture repair
- PRESERVE the sphincter mechanism, avoid narrowing the canal
- simple sphincter lacerations: repair primarily w/ absorbable suture
- document size, distance from the verge and dentate line, and every associated structure – urethra, vagina, bladder, pelvic nerves, sacrum, cord
- massive wounds: debride nonviable tissue but do NOT over-debride at the first operation
- TAG the cut sphincter ends for later repair
- COLOSTOMY EARLY for complex perineal wounds involving the sphincters
- proctectomy for large destructive anal + extraperitoneal rectal injury to control pelvic sepsis
- perineal wound VAC between serial debridements. Investigate the GU tract
- delayed sphincter repair has good functional outcomes
- MANOMETRY before colostomy reversal – it predicts function
- pelvic floor PT and biofeedback for minor injuries
- nonfunctioning sphincter or pre-existing incontinence → PERMANENT colostomy

43 BENIGN COLORECTAL DISEASE: INFLAMMATORY

BOWEL DISEASE — PATHOBIOLOGY

OVERVIEW

Chronic relapsing intestinal inflammation – multifactorial

- genetics + microbiome + immunity + environment, no single cause

Prevalence ~0.5% in North America & Europe – highest rates worldwide

Incidence rising fast in newly industrialized nations

Ashkenazi Jews: ↑ CD risk regardless of geography or era
→ ethnic genetic predisposition

HOST FACTORS

GENETICS

8-10x greater risk of IBD among relatives

15% of Crohn's pts have an affected family member

Twin concordance:

- CD: 50% monozygotic vs. < 10% dizygotic
- UC: only 10-15% monozygotic → non-genetic factors matter MORE in UC

Sibling with the dz: 26x risk for CD, 9x for UC

Polygenic – hundreds of loci, each contributing very little

- identified loci explain only 13.1% of CD and 8.2% of UC disease variance
- > 50% of IBD loci shared with other inflammatory & autoimmune dz

NOD2 / CARD15:

- FIRST susceptibility gene for CD – found 2001, chromosome 16
- intracellular receptor for muramyl dipeptide (bacterial wall) in monocytes → NFκB activation
- activation by MDP induces autophagy in dendritic cells
- NOD2-defective DCs: deficient autophagy, ↓ localization of bacteria to autophagolysosomes
- also a/w severe pouchitis

Other loci:

- autophagy genes: IRGM, ATG16L1
- IL23R, PTPN2 – shared with autoimmune dz
- the CD loci point to innate immunity, autophagy, and phagocytosis

Monogenic IBD:

- rare – early onset (< 5 yrs) and very early onset (< 2 yrs), severe phenotype
- IL-10 or IL-10 receptor defects → extensive colonic + perianal inflammation
- mechanisms: epithelial barrier disruption, poor bacterial clearance by phagocytes, disrupted T- and B-cell selection
- found by NGS / whole exome sequencing
- monogenic dz is the ONLY setting where genetic testing is indicated (plus TPMT / NUDT15 before thiopurines)

Epigenetics:

- DNA methylation, histone modification, RNA interference, nucleosome positioning

- promoter hypermethylation a/w IBD; methylation differs b/n normal & inflamed tissue
- miRNA profiles unique to CD and UC – in target organ AND peripheral blood

IMMUNITY

Layers of defense:

- epithelium – Paneth cells make antimicrobial peptides
- lamina propria – macrophages & dendritic cells = INNATE
- T & B cells, Peyer's patches = ADAPTIVE

GALT = Peyer's patches + lymphoid follicles + mesenteric nodes

DCs interdigitate b/n epithelial cells to sample lumen, then present to T cells

Pattern recognition receptors: toll-like receptors (TLR) and NOD

Even at rest there is controlled inflammation – balance of Th1, Th2, Th3, Th9, Th17, Treg

IBD = adaptive immune response to SELF-antigen → chronic inflammation & damage

UC called *Th2-mediated*, CD called *Th1* – but the pathways overlap

Cytokines:

- IFN-γ, TNF-α, TGF-β are the principals
- IL-1 → T cells make IL-8 and IL-6
- IL-6 → differentiates Th17 and Treg
- IL-12 → Th1 differentiation. IL-23 → drives IL-17, TNF-α, IL-6
- IL-17 & IL-22 initiate and amplify colitis
- TNF-α → macrophage proliferation, inhibits Treg

Autophagy defects → reduced clearance of intracellular pathogens

INTESTINAL MICROBIOME

16S rRNA next-gen sequencing made all of this visible

IBD microbiota has LESS diversity and richness

- ↑ Fusobacterium and Proteobacteria (Enterobacteriaceae, E. coli)
- ↓ Firmicutes (F. prausnitzii, Ruminococcus, Oscillibacter) and Bacteroidetes

Dysbiosis is NOT seen in unaffected twins & relatives → disease or environment, not genes

NO single organism is the driver

- M. avium: granulomatous enteritis in animals, link to CD in humans unproven
- adherent-invasive E. coli colonize ileal CD lesions – but found in 33% of healthy controls

Metagenomics: only 2% of composition differs, but 12% of metabolic pathways do

- ↑ virulence, secretion & oxidative stress pathways; ↓ SCFA and amino acid production

Virome: ↑ Caudovirales bacteriophages, Synechococcus phage S, Retroviridae

Fungi: ↑ S. cerevisiae and C. albicans; Basidiomycota a/w flares

Cause vs. consequence UNRESOLVED – inflammation itself ↓ mucosal oxygen tension and shifts the flora

ENVIRONMENTAL FACTORS

URBANIZATION

Historically a dz of Caucasians of European descent, wealthy Western countries

Now emerging in Asia, Africa, South America, Middle East

- Brazil UC: 1.0 → 4.5 per 100,000
- Hong Kong: barely recognized in the 1960s-80s → 1.3-2.1 per 100,000

Growing up rural is PROTECTIVE (IRR 0.58)

The speed of these shifts is the strongest argument that environment dominates

SMOKING

The classic dissociation – opposite effects in the two diseases

- Crohn's: 2x risk, active or passive (OR 1.76). Also ↑ fistula, stricture, need for surgery
- UC: PROTECTIVE – current smokers OR 0.58

Mechanism unclear – microvascular change, ↓ perfusion, oxidative stress, ↓ barrier & mucus

Nicotine-metabolizing polymorphisms (CYP2A6, GSTP1) a/w CD and protective in UC

APPENDECTOMY

Appendectomy for TRUE appendicitis or mesenteric adenitis before age 20 → protective against UC

- no protection if the appendix was normal – it is the inflammation, not the operation
- modest ↑ risk in Crohn's, likely detection bias

ANTIBIOTICS

Profound ↓ diversity within days of exposure; in some the microbiome never fully reconstitutes

Meta-analysis (11 studies, 7208 pts): a/w Crohn's (OR 1.74) but NOT UC (OR 1.08)

- all agents except penicillin implicated – metronidazole & fluoroquinolones strongest

Childhood otitis media (a proxy for antibiotic use) → 2.8x risk of IBD

NSAIDs

Literature is MIXED

- CD in remission, NSAID > 5x/month: RR 1.65 for active dz. No effect in UC
- NSAID ≥ 15x/month: ↑ risk of developing both CD and UC
- low dose (< 200 mg/day, less than daily): NO effect on activity

Takeuchi: naproxen, diclofenac or indomethacin in

quiescent IBD → 17-28% relapse within 9 days. NO relapse with acetaminophen

- but pooled meta-analysis of 18 studies: no association for CD (OR 1.42) or UC (OR 1.52)

Mechanism: COX-1/COX-2 inhibition → ↓ prostaglandins → ↑ mucosal permeability

DIET

Cochrane: effect of dietary intervention UNCERTAIN

↑ meat & animal products a/w onset of both CD and UC

↑ fruit & vegetables a/w lower incidence

POUCHITIS PATHOBIOLOGY

Incidence tracks the INDICATION, not the operation → implies a genetic basis

- ~50% after IPAA for ulcerative colitis
- rare to never after IPAA for FAP

IL-1 +2018 SNP: RR 3.1 for pouchitis

NOD2 mutations ↑ in SEVERE pouchitis vs. asymptomatic pouches or septic complications

↑ toll-like receptor 2 & 4 expression; aberrant defensin-1, ↑ defensin-5

Fecal stasis → colonic metaplasia of the pouch mucosa and a colonic-like microbiome

UC pouches vs. FAP pouches: ↓ Bacteroidetes, ↑ Proteobacteria, ↓ diversity

Bottom line – immune and microbial change in a genetically susceptible host

44 BENIGN COLORECTAL DISEASE: IBD DIAGNOSIS AND EVALUATION

EPIDEMIOLOGY

Highest prevalence North America & Europe – 0.3-0.7%
Incidence STABLE in the West, rising in newly industrialized countries over the last 30 yrs
Immigration from low- to high-prevalence areas ↑ incidence in the immigrants AND their children
Average age of onset: Crohn's in the 30s, UC in the 40s

CLINICAL PRESENTATION

Change in bowel frequency (up OR down), crampy pain ± blood, tenesmus, urgency, incontinence, food intolerance, anorexia, weight loss
Presentation ranges from subtle & internalized for years to acute severe colitis as the FIRST manifestation
Sx may be entirely extraintestinal
History MUST cover:

- chronicity of Sx, family Hx of IBD
- recent travel, sick contacts, recent antibiotics – infectious mimics
- NSAIDs, prior XRT, immunosuppression
- full ROS for extraintestinal manifestations

Exam: vitals, abdomen, skin, orbits, perineum + DRE

CROHN'S DISEASE

Described 1932 – originally terminal ileum with strictures and fistulas
Mouth to anus, DISCONTINUOUS (skip lesions), FULL THICKNESS
TI is the most common site; ileocecal ~40%
Creeping fat – mesenteric adipose wrapping the inflamed bowel

Mesentery is no longer thought a bystander – an active mediator of stricture formation and post-op recurrence; mesenteric resection ↓ surgical recurrence

Progresses: inflammatory → stricturing → penetrating (fistula, perforation)

ULCERATIVE COLITIS

Starts at the distal rectum, extends proximally, CONTINUOUS

MUCOSAL only – unless fulminant
Spare small bowel, upper GI tract, and anus

Exceptions that are still UC:

- backwash ileitis – short segment TI when the cecum is involved
- cecal patch – non-contiguous cecal inflammation with distal dz
- rectal sparing / patchy dz after topical enema therapy or partial Rx response
- severe acute or fulminant colitis: inflammation beyond mucosa, transmural, skip areas, perforation

COMPLETE rectal sparing in an untreated pt should send you back to pathology – phenotype drives the operation

DISTINGUISHING FEATURES

- Distribution: CD anywhere / UC distal rectum extending proximally
- Ileum: CD often / UC only backwash ileitis
- Perianal dz: CD common / UC rare (cryptoglandular)
- Wall: CD full thickness / UC mucosal unless fulminant
- Creeping fat, fistulas, strictures, deep ulcers: CD. UC only in fulminant dz
- Skip lesions: CD often / UC rare, and usually a consequence of treatment

IBD-UNCLASSIFIED & INDETERMINATE COLITIS

No clear dx in ~10% of adult and up to 30% of pediatric IBD

IBD-U: the label BEFORE colectomy

Indeterminate colitis: the label AFTER colectomy, when the specimen still won't phenotype

Generally a MILD course, less medical therapy than UC
~25% reclassified within 5 yrs of dx – 2/3 of those become UC

PSC-IBD

~3/4 of PSC pts have IBD (predominantly UC); only 2-4% of IBD pts develop PSC

Distinct phenotype:

- pancolitis with RECTAL SPARING
- right- > left-sided inflammation, higher rate of backwash ileitis
- milder Sx despite endoscopic activity, better medical response rate

↑ risk of IBD-associated dysplasia and CRC → surveillance starts AT DIAGNOSIS

Timing of colectomy a/w risk of liver transplant; IPAA may worsen graft survival after transplant

EXTRINTESTINAL MANIFESTATIONS

Occur in 1/3 to 1/2 of all IBD pts

1/4 of pts with EIMs present with the EIM BEFORE the IBD dx

a/w HLA subtypes and dysregulated adaptive immunity

Musculoskeletal:

- MOST COMMON EIM – up to 40%
- peripheral arthropathy 5-20%: relation to bowel activity unclear
- ankylosing spondylitis 5-20%: INDEPENDENT of bowel activity, HLA-B27
- osteopenia (50%) and osteoporosis (15%) common
- anti-TNF beneficial

Cutaneous:

- oral aphthous ulcers 10% and erythema nodosum 10-15% – TRACK bowel activity
- pyoderma gangrenosum 1-5% – does NOT track activity
- pyostomatitis vegetans

Ophthalmologic:

- episcleritis 2-3% – tracks activity; injection, burning, tearing

- uveitis / iritis 2-6% – independent of activity, needs PROMPT treatment

Hepatobiliary:

- PSC 5-8% – INDEPENDENT of bowel activity

Coagulopathy:

- ↑ DVT, PE, mesenteric thrombosis – prophylax every admission
- ↓ protein S and antithrombin III, ↑ factors V and VIII

Others to look for:

- anemia, cholelithiasis, nephrolithiasis, tubulointerstitial nephritis

DIAGNOSTIC EVALUATION

LABORATORY & STOOL

CBC, lytes, Cr, LFTs, bilirubin, iron studies

CRP correlates with severity – more in CD than UC

- NOT specific enough to exclude infection
- NORMAL in up to 30% of pts with active CD

ESR: extensively used, LITTLE clinical utility for diagnosis

First presentation of diarrhea → stool culture, O&P, and C. diff toxin if recent hospitalization or antibiotics

Hx of anal receptive intercourse → anal swabs for STI, NAAT for Chlamydia

Serology:

- pANCA and ASCA individually have LOW sensitivity – poor for diagnosing IBD
- +pANCA / –ASCA: sens 70%, spec 93% for UC over CD – but only once IBD is established

Fecal Calprotectin:

- neutrophil cytosolic protein released on degranulation – a burden-of-inflammation marker
- also ↑ in infectious colitis, CA, diverticulitis, and with NSAIDs or PPIs
- pooled sens 93-95%, spec 90-96%
- normal values vary by age – higher in children and the elderly
- < 50 mcg/g: high NPV → may spare the pt an invasive test

Genetic testing:

- > 240 loci identified, NO clinical utility for diagnosis
- only for suspected monogenic dz (IL-10R), onset < 5 yrs, aggressive refractory dz, or TPMT / NUDT15 before thiopurines

IMAGING

Plain films: useless EXCEPT in acute severe colitis – toxic megacolon or perforation mandates urgent colectomy

Imaging adds little in UC other than to exclude small bowel CD

Goals in CD: map distribution, separate inflammation from fibrosis within a stricture, exclude abscess & fistula

CT / CT Enterography:

- CTE with large-volume neutral oral contrast + IV contrast
- luminal stenosis + wall thickening + pre-stenotic dilation correlates with FIBROSIS in the specimen

- low-dose iterative reconstruction protocols cut dose 34-74% without losing utility

Ultrasound:

- cheap, no radiation. Transabdominal for small bowel
- extent of small bowel dz: sens 66%, spec 88%. Presence of small bowel dz: sens 92%, spec 91%
- 100% sens / 63% spec for moderate-severe fibrosis
- transperineal & transanal: > 90% sens for fistula and abscess – cheaper f/u than MR

MR Enterography:

- PREFERRED cross-sectional study of the small bowel
- small bowel dz: sens 97%, spec 96%. Extent: 80% / 95% – all better than US
- COLONIC dz: poor sens (64%), extent only 22% – but spec 93-96%
- delayed enhancement, diffusion-weighted, magnetization transfer: 86-96% sens for fibrostenosis

MR Pelvis:

- GOLD STANDARD for perianal Crohn's
- 80-100% sens & spec for tracts and abscesses; only 69% for the internal opening

ENDOSCOPY

Colonoscopy with TERMINAL ILEAL INTUBATION = gold standard diagnostic test

MINIMUM 2 biopsies each from TI, ascending, transverse, descending, sigmoid, and rectum – SEPARATE vials, read by a GI pathologist

Severe acute colitis → flexible sigmoidoscopy with biopsy only. Perforation risk

Upper endoscopy NOT routine in adults unless upper Sx Spared TI with suspected isolated small bowel CD → capsule, double-balloon or push enteroscopy

HISTOLOGY

UC: *crypt architectural distortion, diffuse transmucosal infiltrate, cryptitis, crypt abscess*

CD: *focal & discontinuous chronic inflammation, focal crypt distortion, granulomas*

- Crypt architecture & acute inflammation: UC diffuse / CD focal
- Crypt abscesses: UC common / CD uncommon
- Mucin depletion: UC pronounced / CD mild
- Paneth cell metaplasia: UC. Pyloric gland metaplasia: CD
- Granulomas & serositis: CD (UC only in ruptured crypts or fulminant dz)
- Neuronal hyperplasia & muscular hypertrophy: CD

NO pathognomonic feature for either

Expert GI pathologists discriminate UC from CD on biopsy only 64-74% of the time

Real value of biopsy = excluding CMV, microscopic and collagenous colitis

CLASSIFICATION & GRADING

MONTREAL CLASSIFICATION – CROHN'S

Age at diagnosis: A1 < 17, A2 17-40, A3 > 40

Location: L1 terminal ileum, L2 colon, L3 ileocolon, L4 upper GI (added to L1-L3, i.e. L2+L4)

Behavior: B1 non-stricturing non-penetrating, B2 stricturing, B3 penetrating

- "p" modifier appended for perianal dz (B1p)
- Paris classification is the pediatric version, adds growth delay (G0/G1)
- behavior progresses over time – reclassify

MONTREAL CLASSIFICATION – ULCERATIVE COLITIS

Extent: E1 proctitis, E2 left-sided (distal to splenic flexure), E3 extensive (proximal to flexure / pancolitis)

Severity:

- S0 remission
- S1 mild: ≤ 4 BM/day, no systemic toxicity, normal ESR
- S2 moderate: ≥ 5 BM/day, mild toxicity
- S3 severe: ≥ 6 bloody BM/day, evidence of toxicity

ENDOSCOPIC SCORES

Mayo endoscopic subscore (UC) – the workhorse, 0-3:

- 0 normal / 1 erythema, $\downarrow$ vascular pattern, mild friability / 2 marked erythema, absent vascular pattern, friability, erosions / 3 ulceration & spontaneous bleeding
- weakness: scores only the WORST segment. Moderate inter-rater variability
- MMES sums Mayo across all 5 segments – better, needs validation

SES-CD (Crohn's):

- most used in practice & research; better inter-observer agreement and more responsive than CDEIS
- 5 segments (TI, right, transverse, left, rectum) x 4 domains scored 0-3
- domains: ulcer size, ulcerated surface, affected surface, narrowing

Rutgeerts score – recurrence in the neoterminal ileum after ileocolic resection:

- i0 none / i1 ≤ 5 aphthous ulcers / i2 > 5 aphthous with normal intervening mucosa, or lesions confined to the anastomosis / i3 diffuse aphthous ileitis / i4 diffuse inflammation with large ulcers, nodules, or stenosis
- $\geq i2$ = ENDOSCOPIC RECURRENCE

ACTIVITY SCORES

Truelove & Witts (UC) – what is actually used clinically:

- mild / moderate / severe by BM frequency, blood, fever, tachycardia, anemia, ESR

Mayo score (UC), 0-12: stool frequency + rectal bleeding + physician global assessment + endoscopic subscore

CDAI (Crohn's): < 150 remission, > 450 severe. Research only – needs a 7-day diary

Harvey-Bradshaw: simplified CDAI, highly correlated, used clinically

- well-being, abdominal pain, # liquid stools, abdominal mass, complications

IBD-associated CRC: younger at dx, and higher mortality even stage-for-stage

Higher risk within IBD:

- earlier onset, longer duration, ACTIVE inflammation
- colonic stricture, PSC, family Hx of CRC

Whom & when:

- ALL UC pts and CD pts with colonic involvement. Proctitis alone excluded
- begin 8 yrs after SYMPTOM onset – or AT DIAGNOSIS if PSC
- scope in remission – inflammation makes the pathology unreliable

Interval:

- high risk (PSC, family Hx CRC, stricture, prior dysplasia): ANNUAL
- intermediate (incl. active mucosal inflammation): 1-3 yrs
- low risk: 3-5 yrs

Technique:

- high-definition colonoscopy + chromoendoscopy IF local expertise
- 2 recent RCTs: chromo adds NOTHING over high-definition
- no consensus on random biopsies – until then, 4 random biopsies every 10 cm plus targeted

SCENIC terminology – DALM is DEAD:

- describe by visibility (visible on targeted biopsy vs. invisible on random), morphology, border, ulceration
- polypoid = protrudes > 2.5 mm (pedunculated or sessile)
- non-polypoid = superficially elevated (< 2.5 mm), flat, or depressed

DYSPLASIA SURVEILLANCE

45 BENIGN COLORECTAL DISEASE: MEDICAL

MANAGEMENT OF ULCERATIVE COLITIS

GOALS & SEVERITY ASSESSMENT

GOAL: glucocorticoid-free clinical remission WITH

complete mucosal healing on endoscopy

- histologic remission is emerging as an endpoint, not yet a primary target
- histologic remission = absence of neutrophils in the epithelial crypts

Toxicity is defined by tachycardia, fever and WBC – NOT by a dilated colon

TRUELOVE & WITTS

Mild – ALL six: ≤ 4 BM/day, no or minimal blood, no fever, no tachycardia, no anemia, normal ESR

Severe – ≥ 6 BM/day AND blood, plus fever or tachycardia, and ≥ 4 of the 6 criteria

- fever $\geq 37.5^{\circ}\text{C}$, pulse > 90 , Hgb ≤ 10 , ESR ≥ 30

Moderate – anything in between: 4-6 BM/day, Hgb > 10 , no systemic toxicity, weight maintained

MAYO SCORE – 0 TO 12

Stool frequency + rectal bleeding + physician global assessment + endoscopic subscore, each 0-3

Moderate-severe = steroid-dependent or refractory, ulcers on scope, or Mayo 6-12 with endoscopic subscore 2-3

RISK STRATIFICATION

LOW risk – the pts who get topical 5-ASA alone:

- mild-mod Sx, no systemic Sx, no deep ulceration, normal or mildly $\uparrow$ CRP/ESR
- no EIMs, dx after age 40, normal albumin

Predicts an aggressive course:

- age < 40 at dx, pancolitis, EIMs, deep ulcers, $\uparrow$ inflammatory markers
- # of hospitalizations, # of flares, need for steroids, C. diff or CMV infection
- these pts get escalated early rather than repeated steroid courses

Cumulative colectomy risk 15-20% at 5-10 yrs – 25-30% in the ASUC subset

THE DRUGS

5-ASA COMPOUNDS

Act via PPAR-gamma nuclear receptors – highest in colonic epithelium

Sulfasalazine: 5-ASA + sulfapyridine, cleaved by colonic bacteria

- the SULFAPYRIDINE causes the trouble: GI upset, headache
- rare: marrow suppression, fever, hemolytic anemia, hepatitis, pancreatitis, pneumonitis, rash
- MUST give folate 1 mg daily – depletes folate stores
- nausea/headache are dose-dependent – titrate slowly. STOP for agranulocytosis, pancreatitis, pneumonitis, rash

Mesalamine: safe, side effects rare. Interstitial nephritis → monitor renal function

Formulations:

- Pentasa: controlled release, small bowel and colon
- Asacol: released in TI & cecum at pH 7. Lialda: delayed release, once daily
- Dipentum & Colazal: azo-bonded, colon only
- systemic exposure and efficacy EQUIVALENT among formulations
- suppository reaches the rectum only; an enema reaches the splenic flexure

GLUCOCORTICOIDS

Budesonide – extensive first-pass hepatic metabolism, minimal systemic exposure

- standard capsules release in TI and right colon – wrong target for UC
- budesonide MMX releases through the ENTIRE colon – this is the UC formulation
- oral & rectal budesonide near-zero risk; steroid effects only with chronic high-dose oral

Traditional steroids MORE effective than budesonide or placebo for induction

Resistance and dependence are the problem, not efficacy

- striae, acne, cataracts, glaucoma, fluid retention, hyperglycemia, HTN, insomnia, mood, moon facies
- myopathy, osteonecrosis, osteoporosis; adrenal suppression

THIOPURINES

AZA and 6-MP – steroid-sparing. They do NOT induce remission

Check TPMT genotype or enzyme activity BEFORE starting

- deficient → do NOT give. Heterozygous intermediate → HALF dose
- if you can't assay it, start low and watch the count

Follow 6-thioguanine and 6-methylmercaptopurine metabolite levels

CBC q1-2 weeks initially and after any dose change, then q3 months at minimum

SE: nausea (most common), LFT abnormalities, leukopenia, pancreatitis

- pancreatitis appears in the first 8 weeks – NEVER rechallenge, it will recur
- slight $\uparrow$ lymphoma risk; serious infection +10 per 1000 pts (adjusted OR 1.23, not significant)

METHOTREXATE

12.5-15 mg PO or 25 mg IM/SQ weekly, with folate 1 mg daily

SE: GI upset & stomatitis; leukopenia much less than thiopurines

- rare hepatic fibrosis and hypersensitivity pneumonitis
- NOT effective for induction, and NOT for maintenance in UC

MERIT-UC: relapse 66% on MTX vs. 63% placebo

CALCINEURIN INHIBITORS

Cyclosporine & tacrolimus – inhibit calcineurin → $\downarrow$ IL-2 → $\downarrow$ T-cell proliferation

- CSA 2-4 mg/kg/day IV, 4-8 mg/kg/day PO
- tacrolimus 0.01-0.02 mg/kg/day IV, 0.1-0.2 PO
- IV reaches target trough faster

SE: GI intolerance, gum hyperplasia, hirsutism, HTN, renal dysfunction, tremor, seizure

- 13.5% must discontinue for adverse events

ANTI-TNF

Infliximab 5 mg/kg at 0, 2, 6 weeks then q8 weeks

- loss of response → ↑ to 10 mg/kg or shorten to q6 weeks
- infusion reactions common, rarely require stopping the drug

Adalimumab 160/80 induction then 40 mg SQ q2 weeks – may need weekly

Golimumab 100 mg SQ at weeks 0 and 2, then q4 weeks

Reactive therapeutic drug monitoring is now STANDARD – optimize the level BEFORE switching agents

SE: infection incl. TB, autoimmune reactions, CHF, liver dysfunction, lymphoma, demyelination

- screen for hepatitis B and TB. Active infection = ABSOLUTE contraindication

Biosimilars: CT-P13 – EMA 2013, FDA 2016. 56%

remission at wk 14, mucosal healing at wk 54

- switching from infliximab to CT-P13 is safe and cost-effective

VEDOLIZUMAB

Anti-α4β7 integrin, blocks MAdCAM-1 → ↓ lymphocyte trafficking. GUT SELECTIVE

300 mg at 0, 2, 6 weeks then q8 weeks

STOP if no benefit by 14 weeks, or if steroids can't be stopped by 6 months

Best safety profile of the biologics – low serious infection, malignancy, infusion reaction

USTEKINUMAB

Anti-p40 – the subunit shared by IL-12 and IL-23

Weight-based IV induction (260 / 390 / 520 mg), then 90 mg SQ q8 weeks

TOFACITINIB

ORAL small molecule, nonselective JAK inhibitor. Induces AND maintains

10 mg BID to induce; indication now RESTRICTED to pts who failed or can't tolerate anti-TNF

- RA post-marketing signal of clot and death at 10 mg BID → use 5 mg BID for maintenance
- ↑ infection, especially HERPES ZOSTER, and hyperlipidemia
- no immunogenicity – it is not a biologic

INDUCTION – MILD TO MODERATE

Up to 90% of mild-moderate UC starts on a 5-ASA

Proctitis:

- mesalamine suppository 1 g daily
- no improvement at 2 wks → 1 g BID x 4 wks, then back to daily

- symptomatic relief may come in a week, remission takes 4-6 weeks

Proctosigmoiditis / left-sided:

- mesalamine ENEMA 1 g daily – more effective than a suppository
- oral + topical 5-ASA BEATS oral alone in left-sided dz

Extensive:

- oral mesalamine or diazo-bonded 5-ASA, standard dose 2-3 g/day
- up to 4.8 g/day for more extensive or more active dz
- all doses beat placebo for induction; standard dose beats low dose for maintenance

Topical mesalamine failing at 4 weeks – add one of:

- topical steroid: suppository if dz < 18 cm, enema if beyond. Effect by 3-4 wks, NOT past 8 weeks
- an oral 5-ASA, or an oral steroid

Oral 5-ASA failing:

- do NOT switch 5-ASA class – no difference between formulations
- left-sided → budesonide MMX 9 mg/day
- extensive → oral systemic steroids

INDUCTION – MODERATE TO SEVERE

5-ASA is NOT useful for severe dz – steroids are better

Budesonide MMX 9 mg for moderate dz: remission 17.9% vs. 12.1% for 5-ASA vs. 7.4% placebo

Prednisone 40-60 mg – most respond within a week, then taper 5-10 mg/week

- can't get below 10 mg within 3 months = STEROID DEPENDENT → escalate

Immunomodulators are slow and do NOT induce – ACG &

AGA do not recommend monotherapy for induction

- so most moderate-severe pts go straight to a biologic or tofacitinib

Pivotal induction trials:

- ACT 1 & 2 – infliximab (2005): response 61-69% vs. 29-37% placebo at wk 8
- ULTRA 1 & 2 – adalimumab (2012): remission 16.5% vs. 9.3% at wk 8
- PURSUIT-SC – golimumab (2013): response 51-55% vs. 30% at wk 6
- GEMINI 1 – vedolizumab (2013): response 47.1% vs. 25.5% at wk 6
- UNIFI – ustekinumab (2019): remission 16% vs. 5% at wk 8
- OCTAVE 1 & 2 – tofacitinib (2019): remission 18% vs. 8% and 17% vs. 4% at wk 8

VARSAITY – vedolizumab vs. adalimumab head-to-head:

remission at 52 wks 31.3% vs. 22.5%, endoscopic improvement 39.7% vs. 27.7%

UC-SUCCESS – infliximab + azathioprine beats either as monotherapy at 16 weeks

Where the guidelines land:

- biologic-naïve: INFLIXIMAB or VEDOLIZUMAB preferred over adalimumab (AGA)

- tofacitinib in a biologic-naïve pt only within a trial or registry
- ACG: combine the monoclonal with a thiopurine or MTX rather than monotherapy

ACUTE SEVERE ULCERATIVE COLITIS

Definition: ≥ 6 BM/day PLUS one sign of systemic toxicity

– tachycardia, fever, Hgb < 10.5 , or ESR > 30

Up to 25% of UC pts develop ASUC; 40% of them come to colectomy

On admission:

- ADMIT, and get the surgeon involved on DAY ONE
- C. diff toxin, and flexible sigmoidoscopy for severity plus CMV
- VTE prophylaxis. NO opioids, NO NSAIDs

Steroids:

- methylprednisolone 60 mg/day or hydrocortisone 100 mg 3-4x/day
- NO added benefit above 60 mg/day; single or divided dosing equivalent
- response 67%, colectomy 27% during the index admission

Day 3 – Oxford / Travis criteria:

- > 8 stools/day, OR 3-8 stools/day with CRP $> 45 \rightarrow$ ~85% will come to colectomy
- no response by day 3-5 $\rightarrow$ salvage or operate. More steroid will NOT help
- EXCEPTION: if C. diff or CMV is present, treat the infection first

Rescue – cyclosporine OR infliximab, and nothing else:

- CSA 2 mg/kg/day IV – equal to 4 mg/kg with less toxicity, so 2 is the dose
- original RCT: 9 of 11 responded at 7 days vs. 0 on placebo. But 80% still come to colectomy long term
- infliximab 5 mg/kg – colectomy-free survival 71% / 64% / 59% / 53% at 3, 12, 36, 60 months
- head-to-head RCT: failure 60% CSA vs. 54% IFX; no difference in colectomy-free survival at 1 or 5 yrs
- CONSTRUCT: no difference in QOL, colectomy rate, time to colectomy, adverse events, or mortality
- accelerated infliximab (3 doses in 24 days): colectomy 7% vs. 40% at 1 month – gone by 3 months
- adalimumab, golimumab, vedolizumab, ustekinumab have NO established role in ASUC
- do NOT sequence CSA after IFX or the reverse – 16% severe adverse events, colectomy up to 60% at 1 yr

After a successful rescue:

- responded to infliximab $\rightarrow$ maintain on infliximab
- responded to cyclosporine $\rightarrow$ transition to a thiopurine or vedolizumab
- PCP prophylaxis with Bactrim DS 3x/week while on CSA

MAINTENANCE OF REMISSION

The agent that induced remission generally becomes the maintenance agent

Proctitis with < 1 flare/yr responsive to topical mesalamine needs NO maintenance

Everyone else does – proctitis with > 1 flare/yr, all proctosigmoiditis, all dz proximal to the sigmoid

5-ASA:

- mildly active proctitis: rectal 5-ASA 1 g/day – longer time to relapse than placebo
- left-sided or extensive: oral 5-ASA ≥ 2 g/day. Standard-high dose beats low dose
- NO role after moderate-severe dz, and no utility alongside an immunomodulator, biologic, or tofacitinib

Steroids: NEVER for maintenance. Taper over 8-12 weeks

Thiopurines:

- superior to placebo AND to 5-ASA; also work in sulfasalazine/mesalamine failures
- the answer for the steroid-dependent pt – keep steroids on until the thiopurine is therapeutic

Methotrexate: NOT recommended as monotherapy for maintenance

Biologics & tofacitinib – 44-54 week remission vs. placebo:

- adalimumab 17.3% vs. 8.5%; golimumab 47-50% vs. 31%
- vedolizumab 41.8-44.8% vs. 15.9%; ustekinumab 38.4-43.8% vs. 24%
- tofacitinib 34.3% (5 mg) and 40.6% (10 mg) vs. 11%
- no single anti-TNF superior to another for maintenance

WHEN TO CALL THE SURGEON

Failure of medical therapy is the COMMONEST indication – and it is common

Continuing an ineffective drug lets complications

accumulate and WORSENS the surgical outcome

ASUC: surgical consultation at ADMISSION, not at day 7

No response to salvage by day 5-7 $\rightarrow$ operate

Toxic megacolon or free perforation $\rightarrow$ urgent colectomy

Dysplasia or carcinoma

Patient preference matters – 75% of pts will gamble on escalating medical therapy to avoid a permanent ileostomy, but only 64% to avoid an IPAA (vs. 80% of gastroenterologists and 49% of colorectal surgeons)

46 BENIGN COLORECTAL DISEASE: MEDICAL THERAPY FOR CROHN'S DISEASE

STRATEGY

STEP-UP VS. TOP-DOWN

Step-up: 5-ASA → IMM / steroids → biologic only after failure

Top-down: early biologic for the high-risk phenotype

High-risk features: male, young onset, active smoker, penetrating phenotype, short-interval recurrence, multifocal (diffuse jejunoileitis), granulomas, perianal Dz

- also deep ulcers, extensive GI involvement, severe rectal Dz, prior resection

REACT: early combined immunosuppression ↓ hospitalization, serious complications, surgery – but did NOT beat conventional care for steroid-free remission (the primary endpoint)

LIRC: early lap ileocecal resection = reasonable, cost-effective alternative to starting IFX in limited (< 30 cm) inflammatory, non-stricturing/non-penetrating ileocecal Dz

TREAT TO TARGET (STRIDE)

Old target = symptoms. New target = symptoms AND objective inflammation

Targets: resolution of pain & diarrhea, endoscopic remission (NO ulceration), no inflammation on MRE / CTE

- pt-reported endpoints q3 months while active
- endoscopic endpoints q6-9 months
- adjuncts: histologic remission, normal CRP & fecal calprotectin

Mucosal healing inversely a/w relapse, surgery, hospitalization, steroid dependence

CALM: tight control (escalate for CDAI >150, calprotectin >250, CRP >5, or steroids in the past wk) beat symptom-driven care – better endoscopic remission, fewer hospitalizations

WHAT DOES NOT WORK

Antibiotics: no role for induction or maintenance

- role is septic complications ONLY – abscess, perianal cellulitis, low-dose flagyl to ↓ fistula drainage
- cipro now carries a black box for tendinopathy, worse w/ concurrent steroids

Probiotics, diet, fecal transplant: all ineffective in CD

- FMT works for C. diff, NOT CD. FDA safety alert 3/2020 – 2 deaths from shigatoxin-releasing E. coli

Exclusive enteral nutrition: beats steroids for induction in PEDS, NOT replicated in adults

- adult use = bridge off steroids while awaiting elective surgery

5-ASA

Very limited role – it is a topical anti-inflammatory, and CD is transmural

NO efficacy for induction (ileal, ileocolic, or colonic) and NONE for maintenance

Not FDA approved for CD in the US; approved in Europe, Canada, Australia, Japan

Rectal 5-ASA for Crohn's proctitis – palliates urgency & tenesmus only

- suppository 500-1000 mg qhs, enema 4 g/60 cc qd-bid

Sulfasalazine depletes folate → folic acid 1 mg daily

Excellent safety, resume post-op

STEROIDS

BUDESONIDE

FDA approved for ileum / right colon. 3 mg caps, up to 9 mg/day

90% first-pass hepatic metabolism – minimal systemic exposure

Induces remission vs. placebo, but NOT as good as systemic steroids – and much safer

NO efficacy for maintenance – ACG says not beyond 4 months (ECCO allows it)

Open-capsule technique (sprinkled on food) targets SB proximal to the TI; MMX formulation for the colon

Also the mainstay for microscopic / collagenous colitis

Safe to hold peri-op. NO stress dose. Resumption often unnecessary

SYSTEMIC STEROIDS

Prednisone 40 mg PO daily then taper

INDUCTION ONLY – 60% remission vs. 31% placebo, NNT = 3

NOT for maintenance

Need for systemic steroids = bad omen, more complicated course

- steroid dependency is an INDICATION FOR SURGERY

Steroid-refractory: active Dz despite prednisolone 1 mg/kg/day x 4 wks

Steroid-dependent: can't get below prednisolone 10 mg/day, or relapse w/in 3 mo of stopping

Taper & Stress Dose:

- taper 50% q5-7 days, slower if on them long
- do NOT stop abruptly – Addisonian crisis
- stress-dose steroids NOT beneficial in IBD surgery
- 8 mg intra-op dexamethasone for PONV effectively covers it
- short pre-op course (1-2 wks) → just stop post-op; chronic → continue the same dose

Chronic toxicity: impaired healing, immunosuppression, HPA suppression, osteoporosis & AVN (9-40%), cataracts, glaucoma, hyperglycemia, psychiatric

IMMUNOMODULATORS

THIOPURINES

AZA 2 mg/kg qd, 6-MP 1 mg/kg qd

- start low (50 / 10 mg), CBC + LFTs q2wks, escalate q2wks

- clinical effect takes ~3 months – reassess then NOT for induction. YES for maintenance, as a steroid-sparing agent
- AZA > placebo for maintenance in steroid-dependent pts (RR 1.19)

TPMT – MUST check before starting:

- 80% normal
- 10% hyperactive “shunters” → toxic secondary metabolites, hepatotoxicity, less efficacy
 - overcome w/ allopurinol + 25% of the usual dose
- 10% heterozygous / intermediate – need a higher dose
- 0.3% (1 in 300) homozygous deficient – severe myelosuppression, will NOT respond

Toxicity:

- dose-independent: pancreatitis <15%, flu-like 5%, rash 4%
- dose-dependent: hepatitis <30%, GI intolerance <20%, leukopenia 10%, agranulocytosis 0.3%
- CESAME: ↑ lymphoma & leukemia, non-melanoma skin CA, cervical, urinary tract CA
 - colorectal CA risk may be LOWER – inflammation is controlled
- AVOID in young EBV+ males – hepatosplenic T-cell lymphoma, universally fatal

Not a/w post-op complications – hold post-op, let GI restart

METHOTREXATE

25 mg SQ / IM weekly for active Dz; 15 mg PO/SQ weekly maintenance. Folate 5 mg weekly

Maintenance only, steroid sparing. PARENTERAL preferred

Inhibits TNF, MMPs, JAK 1/2, IL-23

TERATOGEN & abortifacient – contraception MUST. Also affects sperm

Toxicity: mucositis, cytopenias, hepatic steatosis/fibrosis, pneumonitis & PCP

Short half-life – safe peri-op, no ↑ in complications

BIOLOGICS

Moderate-severe CD: CDAI >220, CDEIS >8, or SES-CD >6

Before starting: QuantiFERON-TB, viral hepatitis, selective histo/blasto

- vaccinate: pneumococcus, varicella, HPV, influenza, hep A, zoster

ANTI-TNF

Infliximab 5 mg/kg IV at 0, 2, 6 wks → 5-10 mg/kg q8wks

Adalimumab 160 mg SQ wk 0, 80 mg wk 2 → 40 mg q2wks

Certolizumab pegol 400 mg SQ at 0, 2, 4 wks → q4wks

Response in a few wks, may take 6

IFX = the ONLY agent w/ a phase 3 trial for fistulizing / perianal Dz

Best for perianal or rectal Dz, fistulizing Dz, high post-op recurrence risk, pyoderma

S.E.: psoriasis, arthritis, hepatotoxicity, cytopenia, melanoma & lymphoma, opportunistic infection

Combination Therapy:

- SONIC: IFX + AZA in naïve pts beat either alone – more mucosal healing (RR 1.82) and remission at 26 wks, w/ FEWER serious adverse events (RR 0.56)
- IMM ↑ TNFi trough and ↓ anti-drug antibody (ADA) formation
- cost is ↑ opportunistic infection
- starting IFX → add a thiopurine
- if the pt already FAILED IMM monotherapy, no added benefit to continuing it (remission OR 1.02, fistula closure OR 1.10)
- DIAMOND: ADA + thiopurine NOT superior to ADA alone; endoscopic gain at 26 wks lost by 1 yr

VEDOLIZUMAB

α4β7, GUT SELECTIVE. 300 mg IV at 0, 2, 6 wks → q8wks

Slow – response by 12 wks. TNFi-exposed need longer (10 wks vs. 6)

NO PML risk. Can be FIRST LINE – failure of other agents NOT required

USTEKINUMAB

Anti-IL-12/23 (p40). 6 mg/kg IV induction → 90 mg SQ q8wks. Onset ~6 wks

Works TNFi-naïve and exposed; may be less effective after TNFi failure

Excellent safety – no ↑ infection or malignancy in the psoriasis registry

NATALIZUMAB

α4 integrin, NOT gut selective. 300 mg IV q4wks

PML from JC virus – up to 1 in 100 of JC-antibody positive

- check JC serology before and q6 months

Largely ABANDONED

BIOSIMILARS

No difference in efficacy or safety. The advantage is COST

- same amino acid sequence, glycosylation may differ – NOT exact replicas
- infliximab-dyyb non-inferior to IFX in biologic-naïve CD (69.4% vs. 74.3% response)
- insufficient data to switch b/n biosimilars

THERAPEUTIC DRUG MONITORING

Trough targets: IFX ≥ 5 µg/mL, ADA ≥ 7.5 µg/mL, certolizumab ≥ 20 µg/mL

- perianal Dz may need HIGHER levels
- IFX >5 and adalimumab >7.1 predict mucosal healing (85% specificity); no benefit beyond 8 and 12

Loss of response – check the level, then the antibodies:

- level NORMAL → dose up, or change class
- level LOW + ADA low/absent → confirm compliance, then shorten interval, ↑ dose, or add an IMM
- level LOW + ADA HIGH → SWITCH agent

TAXIT: dose to trough 3-7 – fewer flares and 28% cost savings, but no difference in 1 yr remission

TAILORIX: TDM no better than symptom-guided (underpowered)

PAILOT (peds, ADA): PROACTIVE TDM beat reactive

DE-ESCALATION

Candidates: no high-risk features, limited failure history, deep remission (clinical + biomarker + endoscopic/imaging), or after a surgical “reset of the clock”

Do NOT: upper GI CD, repeat penetrating complications, prior escalated TNFi dosing

Get a drug level first – if LOW, the biologic isn't what's holding remission, so de-escalation will likely succeed

Surveil q12 wks x 1 yr w/ CRP & calprotectin. Risk is ADA formation on restarting

POST-OP PROPHYLAXIS

Off all therapy: endoscopic recurrence ~90% at 3 yrs, clinical up to 60%

50% s/p ileocolic resection need another operation w/in 10 yrs

SCOPE EVERYONE at 6-12 months after ileocolic resection

Rutgeerts score, neoterminal ileum: i0 none – i1 ≤5 aphthous ulcers – i2 >5 aphthae w/ normal mucosa b/n, skip lesions, or lesions confined to the anastomosis – i3 diffuse aphthous ileitis, diffusely inflamed mucosa – i4 diffuse inflammation w/ large ulcers, nodules, or narrowing

- ≥ i2 → escalate

Risk factors: young, male, SMOKING, penetrating/fistulizing, prior resection, short Dz duration

- positive histologic margin (OR 1.7) or myenteric plexitis (OR 2.3)

NO benefit: probiotics, 5-ASA, budesonide. Antibiotics may help a little

BEST: monotherapy w/ a biologic or an IMM

POCER (De Cruz): colonoscopy at 6 mo w/ step-up for recurrence BEAT drug therapy alone – risk-adjusted selective therapy, NOT routine escalation for everyone

- low-risk pts still need surveillance; early remission does not excuse it

Truly low risk (short-segment ileocolic, long duration, elderly non-smoker) may be surveilled OFF therapy

PERI-OPERATIVE SAFETY

5-ASA, thiopurines, MTX: safe. Hold peri-op, restart per GI

Steroids: THE major modifiable risk – dose-dependent infectious & wound complications

- worst > 40 mg prednisolone. Wean to < 20 mg, ideally OFF x 1 wk before elective surgery

- HPA recovery takes 2 days to 1 year – no correlation w/ dose or duration

Anti-TNF: data conflicting – treat as ONE risk factor among several

- 50% of pts on anti-TNF have NO detectable drug at surgery; complications do not track trough
- elective → delay 4 wks (2 half-lives) from the last dose
- can't delay + ≥ 2 risk factors → DIVERT

Vedolizumab: ↑ SSI. Delay 6 wks (2 half-lives), else consider diversion

Ustekinumab: does NOT ↑ post-op septic complications

47 BENIGN COLORECTAL DISEASE: ANORECTAL

CROHN'S DISEASE

GENERAL PRINCIPLES

Perianal involvement in 13-38% of CD pts; > 80% of those need surgery

Risk ↑ w/ Dz duration, severity, and DISTAL location

- Hellers, fistula rate by site: ileal 12%, ileocolonic 15%, colonic sparing rectum 41%, colon + rectum 92%
- a perineal fistula → 3x more likely to have colonic than isolated ileal Dz

Recurrent fistula activity 48% at 1 yr, 59% at 2 yrs

The rules:

- CONTROL SEPSIS FIRST – no immunosuppression until the infection is drained
- treat the luminal Dz, control the diarrhea
- perineal care: sitz baths or shower, barrier cream
- steroids have NO role in perianal CD
- IGNORE asymptomatic Dz
- do NOT operate in the face of active proctitis

Pathogenesis: CD fistulas start as rectal inflammation → ulcer → tract deepened by feces and defecatory pressure. Cryptoglandular infection explains idiopathic fistulas, NOT these

FISTULIZING DISEASE

ABSCESS

Up to 62% of perianal CD pts get an anorectal abscess

Ischiorectal MC – 40% of CD perirectal abscesses

Intersphincteric & supralelevator give few external signs → CT, ERUS, or MRI

Drainage:

- GA except the most superficial
- ischiorectal: incise CLOSE to the sphincter complex → shorter subsequent tract
- intersphincteric: drain INTO the rectum, dividing mucosa + IAS over the cavity
- large cavity → mushroom catheter. Packing impedes drainage, poorly tolerated
- if an internal opening is found, place a SETON – NOT fistulotomy, even for a low tract
- culture (aerobic + anaerobic) + antibiotics if immunosuppressed, cellulitic, or septic

CLASSIFICATION

Parks (1976): the tract's relation to the EXTERNAL sphincter – intersphincteric, transsphincteric, suprasphincteric, extrasphincteric

AGA simple vs. complex – this is the one that drives treatment:

Simple:

- LOW (superficial, low intersphincteric, low transsphincteric)
- single external opening, no pain or fluctuance
- NOT rectovaginal, NO stricture

Complex:

- HIGH (high intersphincteric, high transsphincteric, extra- or suprasphincteric)
- multiple openings, abscess, RVF, anorectal stricture, or active rectal Dz on scope
- 80% of CD fistulas are COMPLEX – higher recurrence, worse healing

DIAGNOSIS

Office DRE & anoscopy limited by pain, rarely therapeutic → EUA favored

Accuracy for tract anatomy: ERUS 91%, EUA 91%, MRI 87%

- ANY TWO combined = 100%

MRI = GOLD STANDARD for telling complex from simple (LR 22.7, vs. 6.2 ERUS and 2.1 exam)

Simple → EUA alone is enough. Complex → map w/ pelvic MRI FIRST

Scope once sepsis is cleared – you must know whether there is proctitis

MEDICAL THERAPY

ANTIBIOTICS

A BRIDGE to immunosuppression, never sole therapy

Cipro 500-1000 mg/day or flagyl 1000-1500 mg/day

- ↓ drainage (RR 0.8, NNT 5) but do NOT close fistulas
- cipro + IFX 73% vs. 39% response; cipro + ADA 71% vs. 47% at 12 wks

Recurrence on stopping is the rule

THIOPURINES

Adjunct to anti-TNF, or for pts who can't take it – NOT first line

Response (closure or ↓ drainage) 54% vs. 21% placebo (OR 4.44)

3+ months to work – they MAINTAIN closure, they don't induce it

CALCINEURIN INHIBITORS

Tacrolimus 0.2 mg/kg/day: ↓ drainage (43% vs. 8%) but NOT closure

Cyclosporine: 80-88% initial response, 44-45% closure – all lost on discontinuation

Nephrotoxic, needs levels. Reserve for severe Dz otherwise headed to diversion or proctectomy

BIOLOGICS

Infliximab = FIRST LINE and best studied

- 5 mg/kg at 0, 2, 6 wks: ≥ 50% of fistulas closed in 68%, ALL closed in 55% (vs. 26% / 13% placebo)
- median response 2 wks, closure lasts only ~3 months without maintenance
- ACCENT II: q8wk maintenance – 39% complete closure at wk 54 vs. 19%; loss of response > 40 wks vs. 14 wks
- WARNING: MORE abscesses on IFX than placebo – the external opening seals over an undrained tract

Adalimumab (CHARM): 30% vs. 13% closure at 26 wks, 33% vs. 13% at 56 wks

Certolizumab (PRECISE): 54% vs. 43%, NOT significant

Vedolizumab (GEMINI 2): 28% vs. 11% at 14 wks, 33% vs. 11% at 52 wks
Ustekinumab: 61% symptom improvement, 31% closure – small retrospective series only
Anti-TNF + seton + cipro + EUA beats any one of them alone

SURGICAL THERAPY

Anatomy, inflammation, and the presence of proctitis or abscess decide the operation
NEVER fistulotomy or definitive repair w/ active proctitis or an undrained abscess

FISTULOTOMY

Only w/o proctitis, and only minimal muscle involvement
Williams: 73% healed at 3 mo, 93% at 6 mo. 12% minor incontinence
CDAI > 150 is a contraindication

DRAINING SETON

The workhorse – loose, thin silastic
Maintains tract patency, ↓ peri-tract inflammation, PREVENTS recurrent abscess
Healing higher and closure more durable when added to anti-TNF
- *de Groof, 10 studies / 305 pts on seton + anti-TNF: closure 13.6-100%, recurrence 0-83%*
May stay months, years, or permanently
Remove after proctitis is controlled and anti-TNF induction is complete – possible in 98% at a median 33 wks

ENDORECTAL ADVANCEMENT FLAP (ERAF)

Requires NO active proctitis
Excise the internal opening + crypt-bearing tissue, close the IAS defect, advance a U-shaped flap of mucosa, submucosa & IAS over it – NO TENSION
Success 60-64%, but recurrence 57% and incontinence 9.4%
Worse w/ concomitant small bowel Dz, and w/ SMOKING
Contraindicated: proctitis, cavitating ulceration, anal stenosis
Advancement rectal sleeve (Marchesa): circumferential mucosectomy dentate → anorectal ring, full-thickness rectal flap sewn to the dentate line. Alternative to proctectomy in severe complex Dz

LIFT

Ligate & divide the tract in the intersphincteric plane
Gingold: 67% healed at 12 mo, NO incontinence
Kaminski: 75% at < 1 yr, only 33% beyond 1 yr
Better in small bowel than in colonic Dz

GLUE & PLUGS

Fibrin glue: 0-100% healing, sphincter-sparing and repeatable, but CD data are case series
Plug: 15-100% healing, 55% pooled. Multicenter RCT – NO better than simply pulling the seton

MESENCHYMAL STEM CELLS

ADMIRE-CD, phase 3, 212 pts w/ complex fistulas: local allogeneic adipose-derived cells – closure 56.3% vs. 38.6% placebo, and faster (6.7 vs. 14.6 wks)

Meta-analysis: better healing at 6-24 wks (OR 3.06)
- best dose 2-4 x 10⁷ cells/mL
- adipose-derived recur LESS than bone marrow-derived
- relapse-free survival 37% at 4 yrs, surgery-free 63% at 5 yrs

RECTOVAGINAL FISTULA

~10% of women w/ CD, median onset 34 yrs. A/w COLONIC rather than small bowel Dz
Drain sepsis, place setons, optimize medically, and define the sphincter (MRI or US) FIRST
- repair a sphincter defect at the same setting
Options: rectal or vaginal advancement flap, Martius (bulbocavernosus) or gracilis interposition, episiotomy, pull-through
- rectal advancement flap 42% on the first try, 83% after up to 3 attempts
- vaginal flap PREFERRED when there is anal stenosis – healthy tissue
- Martius 50-100%, gracilis 33-80%, plug 50%, mesh 50-78%, stem cells 51% vs. 35%
Start w/ an advancement flap, escalate on failure. Overall 50-80% at 5 yrs, proctectomy ~20%
60% need temporary diversion; half of those become permanent
Smoking & steroids ↓ success, immunomodulators ↑ it

NON-FISTULIZING DISEASE

FISSURES & ULCERS

CD pts get ordinary idiopathic fissures too – treat those by the usual algorithm
Atypical: multiple, OFF MIDLINE, granulating base w/ overhanging edges, may extend onto perianal skin. NOT hypertonic
- non-necrotizing granulomas on biopsy in ~75%
- if no known CD, r/o CA, XRT, syphilis, herpes, HIV, GC, chlamydia, TB, leukemia
Control stool frequency FIRST – treat the luminal Dz, fiber, anti-diarrheals
With NORMAL tone do NOT use tone-lowering treatment – won't work, and it costs continence
Topical flagyl 10% or tacrolimus 0.1%
Anti-TNF = GOLD STANDARD – 43% complete healing, maintained in 73% of responders at 175 wks
- local IFX injection adds nothing if already on systemic IFX
LIS ONLY for a single characteristic MIDLINE fissure w/ HIGH tone and a disease-free rectum
- fissurectomy + LIS: 67% short-term / 60% long-term healing vs. 50% / 49% medical
- but ~60% of surgically treated CD fissures have a complication – nonhealing, recurrence, fistula
- 1/4 of unhealed fissures go on to form a fistula

Cavitating ulcer → ~80% chance of eventual anoproctectomy

SKIN TAGS

Cumulative 10-yr incidence 19%

Type 1: edematous, hard, cyanotic, TENDER – sequela of a fissure, ulcer, or hemorrhoid w/ lymphedema

Type 2: soft, painless, broad or narrow, multiple – “elephant ear”

Symptomatic tags may signal ACTIVE luminal Dz – treat that

Excise only in remission, and only narrow-based tags leaving a small defect

HEMORRHOIDS

Uncommon in CD – 3-20% of IBD pts

Generally do NOT operate. The historic proctectomy rates reflect severe distal Dz, not the hemorrhoidectomy

If you must: luminal remission, OFF steroids, CDAI < 150

- 36 excisional hemorrhoidectomies – 4 complications, 8% needed diversion at 31 mo

ANAL STRICTURE

17% of perianal CD. Almost never occurs without other perianal Dz

Hematochezia, constipation, pain, incontinence. DRE or proctoscopy makes the diagnosis

Asymptomatic → NO treatment

Obstructing → manual, balloon, or Hegar dilation, repeated

- needing repeat dilation is NOT failure if the pt is comfortable b/n them

Rectal advancement described w/ some success. Still, ~HALF come to PROCTECTOMY

ANAL CANCER

↑ risk of BOTH adenocarcinoma and SCC in long-standing perianal CD

In the canal itself, or within a chronic fistula tract

- a long-quiet fistula that suddenly becomes symptomatic, or a newly inflamed chronic fissure → BIOPSY

Found late – needs a high index of suspicion. Treat as for sporadic anal CA

DIVERSION & PROCTECTOMY

10-20% of perianal CD need temporary or permanent diversion

- risk ↑ w/ active rectal Dz and anal stricture
- active colonic Dz + anal stricture → permanent diversion AND proctectomy

Staged proctectomy:

- 1st stage: transect at the levators, permanent stoma
- perianal Dz improves substantially once diverted → limited perineal anoproctectomy later, w/ fewer wound problems
- INTERSPHINCTERIC dissection sparing the EAS – smallest wound

The perineal wound:

- complications after primary closure up to 36%
- worse w/ active inflammation and a “watering-can” perineum
- poor healing in up to 1/3; chronic perineal sinus even after prolonged healing
- nonhealing → skin graft or myocutaneous flap

48 BENIGN COLORECTAL DISEASE: CROHN'S DISEASE

— SURGICAL MANAGEMENT

CHANGING NATURAL HISTORY

Surgery does NOT cure CD – it manages complications. It is PALLIATIVE

Historic cohort (1955-89): 73% needed surgery – 44% at 1 yr, 61% at 5, 71% at 10

Modern cohort (2003-04): 29% – 14.6% at 1 yr, 24.6% at 5, 28.5% at 7

- the biggest drop PREDATES biologics – it tracks steroid use

Goals: relieve obstruction & fistula, improve QOL, PRESERVE SMALL BOWEL, minimize interruption of medical Rx. Pts generally wish they'd had it EARLIER

INDICATIONS

Emergencies (rare): free perforation, toxic colitis, major hemorrhage

The rest: failed medical Rx, obstruction, penetrating Dz, CA or dysplasia, growth retardation

FAILED MEDICAL THERAPY

No symptom control despite escalation through all agents, or intolerable side effects. Peds growth retardation counts

Steroid-refractory: active Dz despite prednisolone 1 mg/kg/day x 4 wks

Steroid-dependent: can't get below prednisolone 10 mg/day, or relapse w/in 3 mo of stopping

OBSTRUCTION

~1/4 of all CD surgery. There is NO antifibrotic therapy

The question is FIBROSIS vs. INFLAMMATION – CT or MR enterography

- inflammation: mucosal hyperenhancement, comb sign, mesenteric hypervascularity, T2 hypersignal, ulceration, blurred margins
- mesenteric hypervascularity is the ONLY CT finding that predicted fibrosis on multivariate analysis
- upstream dilation does NOT distinguish them. Most lesions are MIXED

Inflammatory stenosis → medical. Fibrotic stricture → SURGERY, up front if inflammation is minimal

Ileocectomy controls Dz well – over half never need a second operation

Endoscopic balloon dilation:

- ideal: SINGLE short (< 4 cm) fibrostenotic or anastomotic stricture, no angulation, no fistula or abscess
- dilate ≥ 14 mm; 16-18 mm needs less frequent redilation; > 20 mm in SB → perforation & bleeding
- multifocal stricturing = independent risk factor for failure
- Bettenworth, 1463 pts: technical success 89%, but 75% redilated and 50% resected by 2 yrs. Adverse events 2.8%

PERFORATION

Rare. Usually just proximal to a completely obstructing SB stricture – a closed loop. Resect + primary anastomosis if the pt tolerates it

Obstructing COLONIC stricture perforates at the CECUM → TAC + ileostomy

PENETRATING DISEASE

11-16% of adults. Origin is almost always the TI / ileocecal region

By site: enterocolonic 29%, enteroenteric 18-24%, enterocutaneous 6-16%, rectovaginal 4-9%, enterovesical 2-8%

Operate only on SYMPTOMATIC fistulas – resect the diseased segment, primarily repair or wedge-excise the target organ

Psoas abscess: cavities EPITHELIALIZE and recur – resect the bowel, unroof & curette, omental pedicle flap

Phlegmon or small abscess → antibiotics; safe alongside steroids

- Felder: 2/3 of phlegmons resolved completely on high-dose steroids, the rest shrank > 50%

Abscess > 3 cm and accessible → PERCUTANEOUS DRAIN + antibiotics, success > 90%

- Clancy: recurrent abscess far higher after drainage alone (OR 6.54), 70.7% eventually operated vs. 17.9% after primary resection – but 30% avoid an operation altogether
- as a BRIDGE it works – post-op septic complications 25% vs. 69% w/o pre-op drainage
- intra-abdominal SEPSIS, not the penetrating phenotype itself, drives post-op sepsis

CANCER & DYSPLASIA

CRC risk 2-3x general population, mean age 51.5 – ~20 yrs earlier

Cumulative CRC risk 2.9% at 10 yrs, 5.6% at 20, 8.3% at 30. p53 loss is EARLY here; APC loss is late

COLORECTAL STRICTURE: CA in 6.5-6.8%, all SHORT-SEGMENT. Manage dysplasia as in UC

TOXIC COLITIS

Up to 50% of IBD toxic colitis is Crohn's

Truelove & Witt: ≥ 6 bloody BMs/day, temp > 37.8, HR > 90, Hgb < 10.5, ESR > 30 mm/h

- + colonic dilation and lost haustra = TOXIC MEGACOLON

Fails rescue → TAC + end ileostomy, +/- mucous fistula

BLEEDING

Incidence 1-2%. Does NOT track disease activity

Focal erosion into a vessel – NOT diffuse ulceration as in UC, so LOCALIZE. Up to 30% are a bleeding

DUODENAL ulcer – positive lavage → EGD

Colonoscopy w/in 24 h localizes 60-78%; angiography + methylene blue stains a 10-40 cm segment for resection

OPERATIVE PRINCIPLES

Do NOT chase microscopic disease. Complete extirpation is counterproductive

Fazio RCT: 2 cm vs. 12 cm proximal margin at ileocolic resection – NO difference in recurrence, and a positive microscopic margin did not raise it

Resect the grossly involved bowel; strictureplasty to avoid short gut

Isolated Crohn's COLITIS, esp. multifocal w/ perianal Dz, warrants a more aggressive approach

Run the WHOLE small bowel – many strictures are missed pre-op. Size the lumen w/ a calibration sphere or cuffed catheter, CLIP the strictures, record remaining bowel length in the op note

NUTRITION

Serious deficit = weight loss > 10% over 3-6 mo, BMI < 18.5, or albumin < 30 g/L

- pre-op enteral or parenteral support → fewer SSI and septic complications

Malnutrition is an INDEPENDENT risk factor for morbidity & mortality, regardless of biologics

MINIMALLY INVASIVE

Milsom & Maartense RCTs: better pulmonary function, less morbidity, shorter LOS; long term better body image, LESS obstruction and LESS hernia

No evidence lost tactile feedback misses strictures or ↑ recurrence. Robotic has no clear edge

Intracorporeal anastomosis + small (< 4 cm) transverse extraction may eliminate incisional hernia

PERI-OPERATIVE MEDICATIONS

Steroids: dose-dependent infection risk, worst > 40 mg prednisolone

- wean to < 20 mg, ideally OFF x 1 wk pre-op. Stress dosing UNNECESSARY – continue the usual dose

6-MP / AZA and MTX: NO ↑ in complications. Do not stop them

Anti-TNF: ONE risk factor among many. Delay 4 wks (2 half-lives); vedolizumab 6 wks (↑ SSI)

- can't delay + ≥ 2 risk factors → DIVERT. Ustekinumab does NOT ↑ risk

Risk factors for post-op sepsis: steroids, malnutrition, anemia, emergency surgery, anti-TNF, vedolizumab, penetrating Dz or intra-op abscess, recurrent Dz, smoking

ANASTOMOSIS & RECURRENCE

Endoscopic recurrence at the anastomosis in 70-90% at 1 yr; 20-30% reoperated w/in 5 yrs – blamed on fecal stasis, microbiome, local ischemia

CONFIGURATION

Muñoz-Juárez: stapled wide-lumen S-S vs. hand-sewn E-E – clinical recurrence 24% vs. 57%, 5-yr surgical recurrence 11% vs. 20%

Meta-analyses & Cochrane: S-S has a LOWER LEAK RATE, but overall recurrence is NO different

CAST RCT: endoscopic recurrence 37.9% S-S vs. 42.5% E-E (NS); symptomatic 22.7% vs. 21.9%

KONO-S

Hand-sewn ANTIMESENTERIC functional end-to-end, built to stop anastomotic recurrence

- divide the mesentery CLOSE TO THE BOWEL WALL – preserve vascularization and innervation
- divide the bowel TRANSVERSELY, stapler perpendicular to lumen and mesentery, so the mesentery sits in the MIDDLE of the staple line
- sew the two staple lines together = the SUPPORTING COLUMN, which holds the 3-D structure
- longitudinal antimesenteric enterotomy on each stump, starting ≤ 1 cm off the staple line, to a transverse lumen of 7 cm (SB) or 8 cm (colon), closed TRANSVERSELY in two layers

Kono original: 5-yr anastomotic surgical recurrence 0% vs. 15%. At 65 mo f/u, 5- and 10-yr surgical recurrence-free survival 98.6%

Shimada: surgical recurrence 3.4% vs. 24.4% E-E; leak 5.1% vs. 17.3%; 5-yr surgery-free 95% vs. 81.3%

First RCT confirmed lower endoscopic and clinical recurrence

THE MESENTERY

Ulcers begin on the MESENTERIC side, w/ the corresponding creeping fat

Ireland: wide mesenteric excision vs. conventional close division – surgical recurrence 2.9% vs. 40%, but historical control, prophylaxis not standardized. NOT settled

PROPHYLAXIS & SURVEILLANCE

Regueiro: endoscopic recurrence 9.1% on post-op IFX vs. 84.6% placebo at 1 yr. PREVENT missed its clinical endpoint but had lower endoscopic scores

De Cruz (POCER): early colonoscopy w/ treatment step-up BEAT drug therapy alone

- RISK-ADJUSTED selective therapy, NOT routine escalation for everyone
- low-risk pts still need surveillance; early remission does not excuse it

SPECIFIC LOCATIONS

GASTRODUODENAL

Clinically significant in 0.5-4%. Macroscopic UGI Dz 30-50%, histologic 40-70%; 2/3 asymptomatic, > 90% have Dz distally too. Calprotectin NOT reliable here

Gastric: aphthous erosions, longitudinal ulcers, "bamboo joint" cardia. Duodenal: longitudinal and NOTCH-like erosions of the Kerckring folds

STRICTURE is by far the commonest problem. Rx mirrors distal Dz + ACID SUPPRESSION

Surgery is < 1% of CD operations. Indications: obstruction and fistula

- nearly every gastroduodenal fistula ORIGINATES ELSEWHERE (TI, transverse colon) and penetrates in

– Kocherize widely, close in 1-2 layers, jejunal serosal patch for a large defect

Strictureplasty (HM or Finney) after Kocherizing – float a 20 mm Foley to trawl for distal strictures

BYPASS if dense scarring, stricture in D1 or D4, or > 2 strictures

- 1/4 to 1/3 reoperated for marginal ulcer. VAGOTOMY does NOT help – don't do it

Whipple only for severe ampullary dysfunction or cholangitis

JEJUNOILEAL

Non-terminal jejunum & ileum in 3-10%. Indications: obstruction and sepsis

Reoperation in up to 30%; short bowel syndrome in 8.5% at 20 yrs

Fibrostenotic stricture w/ lumen < 20 mm = resect or strictureplasty; lesser ones may be left alone in the biologic era

Resection for the INFLAMMATORY or PENETRATING phenotype; strictureplasty for QUIESCENT STRICTURING Dz and pts at risk of short gut

COLON & RECTUM

Toxic colitis → TAC + end ileostomy regardless of rectal involvement

- inflamed stump: rectal tube if intraperitoneal, bury it extraperitoneally in the LLQ or above the fascia in the midline (a leak then presents in the abdominal wall, not the pelvis), or mature a mucous fistula – SUBCUTANEOUS placement has the lowest morbidity

Rectal involvement → no distal target → TPC + end ileostomy

- poor nutrition or high-dose steroids → near-total proctocolectomy w/ ultralow Hartmann, completion perineal proctectomy later

Rectal-sparing short-segment colitis → SEGMENTAL RESECTION

- two contiguous diseased segments → resect IN CONTINUITY, not as two separate resections

Recurrence / further surgery / permanent stoma:

- segmental resection: 26-55% colorectal recurrence / 11-66% / 5-44%

- total abdominal colectomy: 24-66% / 30% / 25-50%

- total proctocolectomy: SB recurrence up to 39% / 9-32% / up to 100%

TPC is the ONLY operation that minimizes recurrence – and still 39% recur in small bowel

Risk factors for recurrence & permanent stoma:

SMOKING, isolated distal Dz, perianal Dz, young age, female sex

IPAA IN CROHN'S

Older series: up to 56% pouch excision. Modern: pouch failure 13.3% vs. 5.1% UC, indefinite diversion 15.5%

Pre-op hints of Crohn's (subtle perianal Dz, discontinuous inflammation) do VERY poorly

Consider ONLY in highly selected Crohn's COLITIS w/ NO perianal and NO small bowel Dz

ILEOSIGMOID FISTULA

Inflamed TI adheres to an otherwise NORMAL sigmoid

Divide, debride the sigmoid defect, close primarily – works in 75%

Resect the sigmoid if it is itself diseased, the opening is large, there is extensive fibrosis, or the tract enters near the mesentery

STRICTUREPLASTY

Indications:

- multiple diffuse SB strictures; rapid recurrence w/ obstructive Dz

- stricture after > 100 cm of prior SB resection, or existing short bowel

- nonphlegmonous FIBROUS stricture

Contraindications:

- perforation, phlegmon, or any fistula at the site

- multiple strictures in a short segment; stricture close to a planned resection margin

- albumin < 2

Heineke-Mikulicz: < 10 cm – the workhorse

- single longitudinal antimesenteric incision, carried 2 cm beyond the diseased bowel both ways, closed TRANSVERSELY

Finney: 10-25 cm

- fold the segment on itself, long antimesenteric enterotomy, sew anterior and posterior walls separately

- > 25 cm gives a functional blind loop → bacterial overgrowth

Jaboulay: > 10 and < 25 cm, side-to-side internal bypass – leaves a lateral diverticulum, blind loop and stasis

Michelassi – side-to-side ISOPERISTALTIC: > 20 cm, or many short strictures in tandem

- divide bowel & mesentery at the MIDPOINT of the strictured segment

- longitudinal enterotomy on both loops, TAPER the ends – no blind stumps

- documented radiographic, endoscopic & histologic REGRESSION of Dz at 7.5 yrs

Outcomes:

- early complications (bleeding, sepsis) 8-15%

- overall recurrence 18-29%, but only 4.6% at the strictureplasty site itself

- Reese: surgical recurrence more likely and the recurrence-free interval SHORTER than after resection

- BIOPSY the mucosa and CLIP the site – CA has been reported at strictureplasty sites

49 BENIGN COLORECTAL DISEASE: ULCERATIVE

COLITIS — SURGICAL MANAGEMENT

INDICATIONS FOR SURGERY

OVERALL

~30% of UC pts will have surgery in their lifetime
- 90% elective, 10% emergent

ELECTIVE

Medically refractory colitis + its complications = 70% of the surgical cohort

Med side effects, extraintestinal manifestations, growth retardation in children

Over 50% of operated pts felt they should have had surgery EARLIER

Dysplasia / CA – overall CRC rate in UC 3.7%

- 2% at 10 yrs, 8% at 20 yrs, 18% at 30 yrs
- risk ↑: young age at dx, pancolitis, mod-severe dz, FHx CRC, PSC
- surveillance q1-2 yrs after 8 yrs of dz – chromoendoscopy or HD scope
- random quadrant bx q10 cm + targeted bx of raised lesions

SCENIC:

- distinguishes visible vs invisible (random), polypoid vs non-polypoid
- endoscopically resected polypoid dysplasia → surveillance, NOT colectomy
- non-polypoid (flat) dysplasia → surveillance – conditional & still controversial
- challenges routine random quadrant biopsies
- invisible dysplasia → confirm w/ GI pathologist, refer to an IBD center
 - invisible HGD, no visible lesion → COLECTOMY
 - invisible LGD → surveillance. Multifocal LGD → surgery
- old low-def data: CA in the specimen w/ invisible LGD 22%, invisible HGD 32-42%
- invisible dysplasia now 10% of dysplastic bx vs 87% pre-HD/chromo era

EMERGENT

ASUC failing medical Rx, sepsis, toxic megacolon, perforation, hemorrhage

~25% of UC pts will be admitted with ASUC

Truelove & Witts (modified):

- bloody stools ≥ 6/day PLUS ≥ 1 systemic sign
- HR > 90, T > 37.8 °C, Hgb < 10.5 g/dL, ESR > 30 mm/hr

Initial Rx: IV steroids, fluids, electrolytes, VTE ppx, nutrition

- MUST rule out C. difficile and CMV
- 30-40% have partial or no response

Rescue IFX or cyclosporine – colectomy still 13-25% in-hospital, ~50% at 1 yr

Le Baut colectomy risk score – 1 pt each:

- prior thiopurine/anti-TNF (HR 3.86), C. diff (HR 3.73), CRP > 30 (HR 3.06), albumin < 3.0 (HR 2.67)

- 1-yr colectomy by score 0/1/2/3/4 = 0% / 9% / 11% / 51% / 100%

Waiting until all medical options are exhausted → morbidity > 50%, in-hospital mortality 8%

Toxic megacolon = ASUC + radiographic dilation, total or segmental

- dilation WITHOUT toxicity → trial of bowel rest + serial exams
- fever, tachycardia, progressive pain → URGENT colectomy

THE EMERGENT OPERATION

Total abdominal colectomy + end ileostomy = PREFERRED

Leave the rectum as a Hartmann stump, even if diseased

- staple line integrity in question → mucous fistula or subcutaneous rectal stump

Do NOT dissect the pelvis

- hinders future restoration, risks autonomic nerve injury and bleeding, now and later

Removing the colon alone lets the pt come off immunosuppression and regain nutrition

Completion proctectomy ± IPAA later, electively

PREOPERATIVE PLANNING

Stoma marking + enterostomal therapist

Small bowel eval to exclude SB inflammation and confirm the dx of UC

Steroid taper as tolerated

NO peri-op stress-dose steroid – ↑ infectious complications (Zaghiyan RCT)

- exception: documented adrenal insufficiency
- continue the home dose peri-op, taper at discharge

Nutritional optimization, dietician, prehabilitation

Mechanical bowel prep + oral antibiotics

ILEAL POUCH-ANAL ANASTOMOSIS

Parks & Nicholls 1978 – complete mucosal stripping + triple-loop S-pouch

Now: preserve the ATZ, stapled J-pouch to the anal canal

STANDARD operation for medically refractory dz, dysplasia, or CA

Sphincter integrity MUST be assessed before offering it

Laparoscopic PREFERRED electively – fewer minor complications, shorter LOS

- ↓ adhesions, improved fertility long term
- nuances identical for open, hand-assisted, or robotic

TECHNIQUE

Modified lithotomy. 11 mm umbilical, 5 mm suprapubic, 12 mm at the future ileostomy site

Run the small bowel for Crohn's, inspect for perforation
Colectomy right → left, mesenteric dissection CLOSE to the bowel

- PRESERVE the ileocolic artery – it perfuses the pouch

Assess mesenteric reach at the pelvic brim – the decision point to proceed or abort

- foreshortened or fatty mesentery → clear the upper rectum, staple, abort the pouch

Specimen out through the ileostomy site – cruciate fascial opening to two fingers, wound protector

- transect ileum flush with the cecum EXTRAcorporeally – prevents staple line blowout
- too thick to deliver → small Pfannenstiel

Proctectomy: divide the superior rectal a., enter the presacral space

- close-rectal / intramesorectal dissection is the alternative to the TME plane – less autonomic injury
- anterior: preserve rectoprostatic fascia / rectovaginal septum, to prostate or mid-vagina
- lateral stalks close to the rectum. Posterior past the coccyx

Transect at the PUBORECTALIS, leaving a 1-2 cm rectal cuff

Pouch:

- J limbs 15-25 cm, exact length set by reach
- apex MUST reach beyond the pubic symphysis for a tension-free anastomosis
- lengthening: selective division of mesenteric vessels to the apex; ant. & post. superficial mesenteric incisions along the SMA; mobilize mesentery up to and anterior to the duodenum
- orient the mesentery straight along the retroperitoneum, proximal loops NOT tethered caudal to it

Double-stapled:

- air-test the rectal staple line FIRST
- anvil into the pouch apex on a running purse-string
- trocar through the transverse staple line – abd. operator clears adjacent tissue before firing

Mucosectomy + hand-sewn:

- Lone Star retractor, dilute epinephrine submucosally
- strip to a short denuded muscular cuff above the dentate line
- full-thickness pouch to IAS and anal mucosa AT the dentate line

Presacral drains + diverting loop ileostomy

Post-op as for LAR. High ileostomy output – hydration, antidiarrheals. ERAS pathways are safe

Ileostomy closure at 6-8 wks: DRE for tone & stricture, pouchoscopy, contrast study FIRST

CONTROVERSIES

ONE-, TWO-, OR THREE-STAGE

Staged is far more common and prudent – diversion prevents catastrophe from a leak

Single-stage argument: ileostomy complications 43% – obstruction 23%, dehydration 25%, closure complications 29%

317 diverted vs 670 undiverted: leak rate identical, 13.6% vs 13.7%

- unplanned trip to OR after leak 12% diverted vs 45% undiverted
- unplanned ostomy after leak 27% vs 60%
- 4031-pt series: reop for diversion 12% vs 48%. Pouch survival same at 5 & 10 yrs
- biased – omission is only offered to the healthiest
- AUTHORS: routine diverting ileostomy

Indications for 3-stage – TAC + end ileostomy first:

- hospitalized pt having urgent colectomy
- malnutrition, albumin < 3 g/dL
- steroids > 20 mg/day
- obesity, cancer, women desiring pregnancy, IBD-unclassified

Biologics:

- PUCCINI, prospective, 955 pts: any infection 19% vs 20%, SSI 12% vs 13% w/ vs w/o pre-op anti-TNF
- vedolizumab & ustekinumab – no robust signal
- pre-op biologics ALONE are NOT an indication to stage

Obesity:

- independent predictor of pouch abandonment – visceral fat in the ileal mesentery limits reach
- ↑ overall complications and leak. Target BMI < 30 before IPAA

Modified Two-Stage:

- TAC + end ileostomy, then completion proctectomy + IPAA WITHOUT diversion
- avoids ileostomy morbidity and a third operation
- 223 traditional 2-stage vs 237 modified: leak 15.7% vs 4.7%
- selection and generalizability unsettled

POUCH DESIGN

Parks & Nicholls S (3 limbs, hand-sewn) → Utsunomiya J (2 limbs) took over with the stapler

Meta-analysis of 23 studies: NO difference in leak, pelvic sepsis, or pouch failure b/n designs

J-pouch = one more BM per 24 hrs. All other function equivalent

S-pouch gives ~2 cm more length – reserve for the short fat mesentery in a long narrow pelvis

STAPLED VS HAND-SEWN

Stapled = default for 3 decades

1990s RCTs: neither superior – small, single-institution, type II error

Long-term function EQUAL. Short-term morbidity consistently LOWER with stapled

No ↑ neoplasia in the ATZ or pouch after stapling

- dysplasia or CA at the TIME of IPAA is the independent risk factor

Rectal HGD or adenocarcinoma at surgery → mucosectomy + hand-sewn

Everything else → stapled

TRANSANAL POUCH (TA-J-POUCH)

Fixes the oblique laparoscopic distal transection – fewer firings, ↓ leak
Avoids a long retained cuff → less cuffitis and fewer evacuation problems
Single stapled anastomosis, no “dog ears,” reinforceable transanally
Short- and long-term function supportive. Robust data still awaited

ALTERNATIVES TO IPAA

ILEORECTAL ANASTOMOSIS

Advantages: technically easier, single stage, NO pelvic dissection

- avoids pelvic sepsis, poor function, pouchitis, sexual/urinary dysfunction, infertility

Preserve the ileocolic AND superior rectal aa. Transect where the taeniae splay

- 29 mm EEA for a wide lumen, anvil placed extracorporeally
- underwater flex sig test, ICG perfusion
- diversion NOT needed if healthy, tension-free, and intact

Complications 24-28%, leak 3-4%

Long-term FAILURE 18-49%

- pre-op immunosuppressant AND anti-TNF → predicts failure
- colectomy for acute severe colitis → LOWER failure

CA in the retained rectum:

- 4.8% overall; 0% under 10 yrs of dz, 13% at 25 yrs
- meta-analysis subsequent CRC: rectal stump 2.1% / IRA 2.4% / IPAA 0.5%
- IRA or stump vs IPAA – OR 6.4
- prior CRC is the dominant risk factor – OR 12.8 after IRA, OR 15.0 after IPAA

Candidates: biologic-naïve ASUC, indeterminate colitis w/ rectal sparing, young women maximizing fertility

Rectal compliance must be adequate – chronic UC impairs it

TOTAL PROCTOCOLECTOMY + END ILEOSTOMY

Elderly, distal rectal CA, severely compromised anal function, or pt preference

Loop-end ileostomy if the mesentery will not reach

CONTINENT ILEOSTOMY (KOCK)

NOT first choice for permanent diversion

For a failed Brooke, or an IPAA candidate blocked by rectal CA, perianal fistula, poor sphincter, or occupation

Crohn's = CONTRAINDICATED – pouch recurrence costs ~45 cm of small bowel

Relative contraindications: obesity, age > 40

Only highly motivated, emotionally stable pts

Technique:

- terminal 45-50 cm of ileum – 3-5 cm outlet, 18 cm nipple valve, 30 cm pouch

- peritonectomy over the valve mesentery on both sides → adhesion, prevents slippage
- S configuration, valve made with 3 firings of the GIA WITHOUT the knife
- pouch anchored to the posterior rectus sheath, stoma flush with skin
- drainage tube occluded progressively from day 10 until 8 hrs without distress
- then intubate TID

Complications:

- nipple valve slippage 30% – MOST COMMON, surgical correction near inevitable
- pouchitis 25%, fistula 10%, obstruction 5%
- prosthetic wrap stops slippage but causes parastomal abscess & fistula
- Barnett modification: no controlled data that it is any better

90% eventually fully continent after one or more procedures – satisfaction high despite complications

FUNCTION & QUALITY OF LIFE

~6 BMs/day at 1 yr. Pouch capacity 2-4x initial by 1 yr

Continence for stool & flatus ~80% by day, ~70% at night.

Major incontinence 5%

IRA gives 2-4 BMs/day – fewer than IPAA

Pelvic dissection ↓ female fertility – laparoscopic and staged approaches mitigate it

Discuss bowel, sexual, urinary function and fertility

PREOP, and follow them after

50 BENIGN COLORECTAL DISEASE: COMPLICATIONS OF

THE PELVIC POUCH

RISK FACTORS FOR POUCH DYSFUNCTION

Pelvic sepsis in up to 25% of IPAA pts – MOST COMMON cause of pouch loss

- from disruption of the pouch-anal anastomosis, less often the tip-of-J staple line
- 30% of these go on to pouch failure

High-dose steroids and biologics impair pouch healing → STAGE the operation

Anastomotic complications leave occult sinuses or chronic leaks that MIMIC pouchitis

Technical missteps at the index operation:

- 180° or 360° twist of the SB mesentery → obstruction or ischemia
- elongated rectal cuff or S-pouch outlet → outlet obstruction, poor emptying

Pelvic floor or sphincter compromise predicts poor function – be honest preop

Other reported rates:

- SBO 20%, leak 5%, IPAA stricture 5-38%, pouch-vaginal fistula 3-16%
- infertility ~26% after pelvic dissection

APPROACH TO THE DYSFUNCTIONAL POUCH

The prior diagnosis is OFTEN WRONG – “chronic pouchitis” and “Crohn’s” are over-applied

- up to 3/4 of pts labeled CD of the pouch were reclassified as anastomotic leak at a specialty center, and salvaged

Full Hx + ALL operative reports – technical difficulty at the index op is the clue

Establish the etiology when dysfunction BEGINS, even if Sx are longstanding – treatments diverge completely

Pouchoscopy – standardized every time:

- rectal cuff, pouch body, afferent limb
- elongated cuff, strictured or twisted afferent limb, prolapsing anterior wall

Best done WITH an anoperineal EUA – fistulae and abscesses are missed in the endoscopy suite

Contrast enema + pelvic MRI for sinus, chronic leak, fistula

Multidisciplinary – CRS + GI in the OR together, biopsies, plan given to the pt the same day

The “thoughtful” ileostomy:

- diversion palliates while the workup proceeds, pouch left in situ
- lets the pt experience a stoma without burning the bridge
- laparoscopic if possible – fewer adhesions for a future redo
- explore at the same time: mesenteric twist, AL adhesions, mesh adherent to the pouch, ovarian cyst
- site it at the most dependent SB, ≥ 15 cm proximal to the pouch – that enterotomy can serve a future pouch-anal anastomosis

STRUCTURAL COMPLICATIONS

AFFERENT LIMB SYNDROME – POUCH INLET

Obstructive Sx, pain, cramping, dyschezia with a NORMAL pouch body and outlet

Causes: fibrotic stricture, adhesive angulation, mesenteric malrotation, volvulus or trapping under the mesentery, accordioning of a floppy pouch

- pre-pouch ileal inflammation is NOT necessarily Crohn’s – NSAIDs and CD-like conditions look identical
- mesenteric compression is intermittent – insufflation at endoscopy overcomes it and looks normal
- angulation is often only found at laparotomy

Rx: balloon dilation first, many persist

- resection + primary anastomosis, strictureplasty, or bypass to the pouch inlet
- pouch-pecty w/ temporary ileostomy “on stretch” for the floppy or rotated pouch
- if CD confirmed, treat as fibrostenotic CD elsewhere – post-op medical Rx

POUCH-ANAL ANASTOMOTIC LEAK

Acute: fever, leukocytosis, pelvic pain, night sweats → CT
- indolent presentation = prolonged ileus or urinary retention

Water-soluble contrast enema, pelvic MRI, EUA

Upper pelvic or abdominal abscess → percutaneous drainage

LOW pelvic abscess or an obvious anastomotic defect → EUA + transanal mushroom drain THROUGH the defect

- transanal PREFERRED over percutaneous – percutaneous routes create complex fistulae

Peritonitis or instability → OR washout and wide drainage

- pouch excision then exceeds 40%, ileostomy reversal unlikely

Speed matters – early control preserves function; delay → peri-pouch fibrosis, non-compliant pouch

Leak vs Crohn’s:

- leak: sepsis originates AT the anastomosis, fibrosis is at the anastomosis, distal pouch & anal canal soft and supple
- CD: waxy skin tags, bluish hue, fixed fibrosis of the distal pelvis

ANASTOMOTIC SINUS

Blind-ending tract from anastomotic dehiscence – 2.8-8%
Presents months to years later, often after a leak that was never documented

MOST COMMON site: posterior pouch-anal anastomosis, w/ presacral inflammation

Independent predictor of pouch failure

- asymptomatic and undiverted → leave alone
- found before ileostomy closure → delay closure 3-6 mo, repeat pouchogram

- persistent → serial I&D, heal by secondary intention over 6-9 months
- endoscopic sinusotomy (needle-knife) ± diversion
- refractory → transabdominal/transanal redo

TIP OF J LEAK

The anatomic end of the small bowel – ischemia or staple line dehiscence

Upper pelvic abscess adjacent to the tip, or a draining midline wound w/ low-output ECF

Will NOT heal alone – surgical revision, rarely full pouch recreation

Anterior (common channel) staple line failure → mid-pelvic abscess with NO anastomotic defect

- full pouch revision required – be prepared to redo the body

FAILURE OF POUCH “SCAFFOLDING”

Posterior mesentery is the limiting axis → the anterior pouch is floppier

Tethers to presacral fascia → ↓ capacity, internal anterior wall prolapse, outlet obstruction

Can also prolapse full thickness out the anus

Usually misdiagnosed as chronic pouchitis; sometimes only found at exploration

Rx: pouch-pecty ± ileostomy on tension. Mesh/matrix fixation reported, data thin

MESENTERIC ROTATION 180° / 360°

Occurs at anastomotic creation if the pouch is allowed to rotate posteriorly

External compression of the afferent limb, or ↓ pouch volume

Risk of pouch ischemia and undue tension on the POSTERIOR anastomosis – already the vulnerable point

PREVENT it – confirm orientation before firing, especially lap/robotic where the mesentery is out of view

Correction requires complete takedown and recreation of the pouch-anal anastomosis

EFFERENT LIMB – POUCH OUTLET

Difficulty emptying, straining, sense of incomplete evacuation

Anastomotic stricture:

- usually within the first 9 months. Up to 17% of all pouches
- more common after mucosectomy + hand-sewn
- stapler size (28/29 vs 31/33 mm) makes NO difference
- soft weblike stricture after a diverted stapled anastomosis → gentle digital dilation
 - does not recur after restoration of continuity, or responds to daily self-dilation
- long fibrotic stricture = peri-op sepsis or ischemia – dilation FAILS long term
 - pouch advancement (transanal or transabdominal), revision, or excision

Elongated S-pouch outlet / long rectal cuff:

- technical error at the index operation

- transanal intubation palliates, not a long-term answer, and traumatizes tissue
- transabdominal/transanal revision to shorten
- a very long cuff can be restapled with a 30 mm linear stapler preserving the ATZ → redo double-stapled anastomosis, mucosectomy avoided

Retained mesorectum:

- left as a “space filler,” it acts as a COLLAR around the distal pouch → evacuation problems
- correction = completion mesorectal excision + new pouch-anal anastomosis

INFLAMMATORY COMPLICATIONS

POUCHITIS

MOST COMMON long-term complication of IPAA

~50% of UC pts have at least one episode in their lifetime

- 40% single episode – ↑ loose stools, tenesmus, bleeding, cramping, malaise
- resolves with 2-4 wks of oral antibiotics
- 60% relapse. Half of those are REFRACTORY → chronic abx, steroids, or biologics

Classification:

- Classic: dysbiosis or bacterial/viral/fungal infection – homogeneous diffuse inflammation ± ulcers
- Immune-mediated: PSC or other autoimmune dz – pouch body AND afferent limb, sometimes w/ cuffitis
- Ischemia-associated: mesenteric tension – ONE limb only, in a vascular distribution, or ulcers along the common channel staple line
- typical pt = obese male with visceral adiposity

Treatment ladder:

- 2 weeks cipro + Flagyl
- responsive → re-treat as Sx recur
- dependent → maintenance antibiotic / probiotic
- refractory → work up pathogen-induced (CMV, C. diff) or immune-mediated, treat accordingly
- ischemic → visceral weight loss

NO surgical cure. Diversion palliates mucosal inflammation and perianal excoriation

Do NOT offer a redo pouch for refractory pouchitis without a reason to expect a different result

CUFFITIS

Symptomatic inflammation of the retained rectal cuff after a STAPLED IPAA – up to 6%

BLEEDING per rectum points to cuffitis, not pouchitis
Constantly mistaken for refractory pouchitis – the treatments differ

Rx: topical steroid enema or suppository, or 5-ASA

Refractory → transanal mucosectomy + pouch advancement w/ hand-sewn anastomosis, or transabdominal redo

- success higher if the original stapled anastomosis is ≤ 3-4 cm above the dentate, and the redo is tension-free

CROHN’S OF THE POUCH

A dx of CD does NOT mandate pouch removal – phenotype drives retention

57% of de novo CD of the pouch keep a functioning pouch

- fistula at the time of dx, and early dx after the index pouch, both predict pouch FAILURE

Timing separates it: pelvic sepsis within 3-6 mo of ileostomy closure = post-op complication

- CD of the pouch presents > 12 months out

EUA is the FIRST operation – establish the dx, control sepsis, biopsy

- mushroom drains for abscess, NON-CUTTING silastic setons for tracts
- do NOT divide sphincter – incontinence risk is high

Diverting loop ileostomy improves but does NOT resolve anoperineal Sx

- mucus drainage and diversion pouchitis persist

FUNCTIONAL COMPLICATIONS

Irritable pouch syndrome:

- diarrhea, urgency, pelvic pain, cramping with NO endoscopic or histologic inflammation
- visceral hypersensitivity and hypermotility. 43% of 61 pts in one series
- Rx: systemic + topical antidiarrheals, antispasmodics, anticholinergics, CBT

Dyssynergic defecation:

- puborectalis fails to relax → dyschezia and straining
- primary (idiopathic) or secondary to pouchitis / cuffitis
- manometry: paradoxical contraction, failed balloon expulsion
- Rx: biofeedback + treat the underlying inflammation

NEOPLASIA OF THE POUCH

Hard to detect even on surveillance. Poor prognosis

Dysplasia: cumulative incidence 0.8% at 5 yrs, 2.2% at 20 yrs

- located at the ATZ or rectal cuff
- risk highest if the ORIGINAL indication was dysplasia or cancer

Pouch cancer: < 50 cases reported. Adenocarcinoma –

ATZ 64%, pouch body 19%

- cumulative incidence 0.2% / 0.4% / 2.4% at 5 / 10 / 20 yrs

SALVAGE, EXCISION & FAILURE

POUCH EXCISION

Traditional answer to failure – HIGH morbidity

Mayo, 147 pts: short-term complications 57%, long-term 37%, 11% back to the OR

St Mark's: early 25%, late 53.7%. Over half readmitted, > 50% of those reoperated

NONHEALING PERINEAL WOUND is the signature complication – up to 40% at 6 mo, 10% at 12 mo

- another series: perineal wound infection 30%, sinus 28%, perineal hernia 7%

- staged excision after diversion may reduce nonhealing

Pathology confirmed CD in only 7 of 35 pouches removed for “Crohn's”

REDO / REVISION

Remzi, > 500 redo pouches over 3 decades – mostly sepsis-related failure

- complications 53%, pelvic sepsis MOST COMMON
- ileus/SBO 16%, leak 8%, wound infection 8%
- redo IPAA failure 20%, but 83% had a functioning pouch at last f/u
- pouch survival 90% at 5 yrs, 82% at 10 yrs
- > 90% would recommend it and would do it again

81-pt series: pouch survival 85% at 5 yrs, 65% at 10 yrs.

Pouch loss 23%, complications 35.6%

Redo for CD: pouch retention < 60% vs 79% at 5 yrs for index pouches

- complications and function comparable – SELECTION is everything
- best candidates: no active anoperineal dz, limited SB dz, uncompromised sphincter

Many pts denied a redo were simply misdiagnosed as CD when the problem was technical

CONTINENT ILEOSTOMY AS SALVAGE

For the pt who wants control of evacuation but cannot have a pelvic pouch

Long-term pouch survival ~80%

Nipple valve slippage = commonest complication and indication for reoperation

95% would choose it again. QOL better than a conventional ileostomy

BOTTOM LINE

Every pouch failure is different – no RCT, all retrospective, all from high-volume centers

Refer for comprehensive evaluation BEFORE excision

A pt who chooses excision + permanent ileostomy for QOL is supported WITHOUT persuasion

51 BENIGN COLORECTAL DISEASE: INFECTIOUS COLITIS

BACTERIAL COLITIDES

Watery diarrhea: supportive. Dysentery – bloody stool, fever, pain – identify the organism

Multiplex GI PCR stool panel is now the workhorse test

CAMPYLOBACTER

MOST COMMON bacterial colitis – US 19.6/100,000, worldwide 25-30/100,000

C. jejuni. Contaminated food or water, poultry the classic source

Incubation 2-4 days. Multiplies in bile, invades to the lamina propria

Bloody diarrhea, fever, pain. TI & cecum → mimics APPENDICITIS

Resolves in 7 days. Up to 16% colonized for 10 weeks

Complications: LGIB, toxic colitis, pancreatitis, Reiter's, Guillain-Barré

Dx: stool culture or PCR. Endoscopy non-specific

Rx: usually none. Severe → azithromycin x 3 days

- fluoroquinolone resistance > 25% in the US, up to 80% elsewhere
- NO isolation precautions needed

SALMONELLA

Typhoid: S. typhi, S. paratyphi – developing world, can obstruct or perforate

Non-typhoid: S. enteritidis, S. typhimurium – MC in the US, self-limited

The 5 Fs – food, feces, fingers, fomites, flies.

Contaminated meat & poultry

Incubation 8-48 hrs

Dx: stool culture/PCR, or a positive blood culture.

Endoscopy non-specific

Rx: most need none. Treat bacteremia, immunocompromised, frail

- fluoroquinolone. Azithromycin if acquired in an endemic area

SHIGELLA

Young children, but affects all. *S. sonnei* MC in the US

Contaminated water, person to person, fecal-oral

Diarrhea usually NON-bloody first – colitis, tenesmus, bloody stool at 3-5 days

Children recover in 3-4 days. Adults may take 3-4 weeks

Complications: perforation, toxic colitis, HUS, dehydration

Shedding continues up to 6 weeks after Sx resolve

Rx: NOT routine – resistance is rising

- treat systemic signs, complications, immunosuppression
- treat food handlers, child-care providers, nursing home residents – public health
- fluoroquinolone, azithromycin, or 3rd-gen cephalosporin x 3-5 days, by sensitivities

ESCHERICHIA COLI

Five diarrheagenic pathotypes:

- STEC (= VTEC = EHEC) – hemorrhagic colitis
- ETEC – traveler's diarrhea

- EPEC – infants. Non-toxin producer
- EAEC – children, persistent diarrhea; also traveler's
- EIEC – dysentery, endemic South America & Europe. Non-toxin producer

STEC / O157:H7:

- identified 1982. Reservoir = cattle. Undercooked beef, unpasteurized milk
- shiga-like toxin, same as *S. dysenteriae*
- NON-O157 strains account for 75% of STEC infections
- stool culture or multiplex PCR for shiga toxin in ANY pt with bloody diarrhea
- most self-limited; a subgroup → bloody diarrhea → HUS
- AVOID anti-motility agents – ↑ toxin production and HUS risk
- NO antibiotics – several actually ↑ the risk of HUS

YERSINIA

Y. enterocolitica. Undercooked pork, contaminated milk, handling infected animals

Invades Peyer's patches of the TI. Diarrhea, fever, pain up to 21 days

MESENTERIC ADENITIS and ILEITIS – mimics CROHN'S and appendicitis

- right colon erosions on colonoscopy, also Crohn's-like
- migratory arthritis, Reiter's, erythema nodosum confuse it further

Dx: stool culture often FAILS. Hemagglutinin titer 1:128, or PCR for virulence genes

Rx: supportive. Systemic → aminoglycoside, TMP-SMX, doxycycline, or fluoroquinolone

TUBERCULOSIS

Swallowed sputum from the primary pulmonary focus

Abdominal pain, fever, weight loss, RLQ mass

Ileocolic inflammation MISTAKEN for new-onset Crohn's or complicated appendicitis

Endoscopy, biopsy, and CT are all NON-pathognomonic

Rx: medical – INH + rifampin. Some strictures and fistulas resolve on medical Rx alone

Operate only to make the dx, or for perforation / obstruction

OTHER BACTERIA

Vibrio: raw shellfish, Sx within 48 hrs. NOT in routine cultures – must be requested

- *V. cholerae* (O1, O139): up to 1 L/hr → shock and death in hours
- high-volume crystalloid + a SINGLE dose of ciprofloxacin or doxycycline

Aeromonas: travel exposure, diarrhea ≥ 2 wks. Culture must be REQUESTED. Treat only the immunocompromised

Bacteroides fragilis – toxin-secreting subclass, self-limited

Listeria monocytogenes – acute colitis in the immunocompromised. TMP-SMX

Gonorrhea, chlamydia, syphilis – anorectal, separate chapter. *C. difficile* – separate chapter

PARASITIC COLITIDES

AMEBIASIS

Entamoeba histolytica. Tropical Americas, Africa, Asia – TRAVEL is the clue in the US

Fecal-oral. Cysts survive in soil up to a month. Incubation 2-4 weeks

Ingested cyst → excystation → trophozoites invade the colonic wall

Most asymptomatic or mild. Deep ulcers → FULL-THICKNESS PERFORATION

Extraintestinal: liver, brain, lung. Hepatic abscess with NO colonic Sx

- can rupture into abdomen or chest → lung abscess or empyema

Mortality up to 50% with perforation or systemic dz

Diagnosis:

- stool microscopy + PCR antigen (highly sens & spec). Up to 3 samples on different days
- serology positive at 2 wks, higher w/ hepatic abscess, stays positive long after cure
 - a positive titer is NOT proof of active disease
- endoscopy: ulcers mm to cm, RIGHT colon most often
- histology: FLASK-SHAPED ulcers, trophozoites at the ulcer periphery
- Ameboma – granulomatous mass in the colon wall, mimics CANCER and obstructs

Treatment:

- asymptomatic → luminal agent alone: paromomycin, diloxanide furoate, iodoquinol
- active colitis → metronidazole x 10 days FIRST LINE, then a luminal agent
- hepatic abscess → medical Rx; aspirate/drain if large or failing
- surgery only for perforation or toxic colitis

Mexico City, 30 yrs – 112 amebic perforations, 0.6% of emergency laparotomies: right colon 90.5%, MULTIPLE perforations 74%, segmental colectomy 53.3%, subtotal + end ileostomy 35.3%, mortality 40%

NEMATODES

Anisakidosis – Anisakis simplex, raw fish, > 90% from Japan:

- larvae burrow the muscular wall – granulomas and eosinophils
- mimics APPENDICITIS, ileitis, diverticulitis, acute abdomen. Surgery often makes the dx
- albendazole 7-21 days

Strongyloides stercoralis – tropics, and Appalachia:

- larvae penetrate skin. AUTO-INFECTION
- acute: larva currens. Chronic: diarrhea, pain, wasting
- can present as PANCOLITIS mimicking UC
- stool larvae; may need duodenal bx, ELISA, or bronchial washings. Ivermectin x 2 days

Trichuris trichiura – whipworm:

- RECTAL PROLAPSE with the worms visible on the mucosa. Anemia, growth retardation
- barrel-shaped eggs on stool microscopy. Mebendazole or albendazole

Ascaris lumbricoides – up to 1.2 billion infected:

- high worm burden → pain or OBSTRUCTION. Milk the bolus distally, resect if needed

Enterobius vermicularis – pinworm, children:

- female deposits up to 10,000 eggs on perianal skin → intense pruritus, autoinfection
- TAPE TEST diagnostic. Mebendazole. Treat the WHOLE family + environmental cleaning

PROTOZOA & FLATWORMS

Cryptosporidium:

- oocysts resist chlorination and are too small to filter
- immunocompetent self-limited; immunocompromised protracted + acalculous cholecystitis / cholangitis
- stool PCR or ELISA. NO reliably effective drug. HAART in HIV; nitazoxanide otherwise

Giardia lamblia – untreated water. GREASY, foul stool at 1-2 wks. ELISA/PCR. Metronidazole

Balantidium coli – only ciliated protozoan infecting humans, pigs the source. Tetracycline

Schistosomiasis – snail host, cercariae penetrate skin:

- *S. haematobium* → bladder. *S. mansoni* & *S. japonicum* → SB and colon mesentery
- eggs in tissue drive the damage. Eggs on stool exam. Praziquantel

Tapeworms:

- *T. solium* (pork) – CYSTICERCOSIS. *T. saginata* (beef)
- *D. latum* (raw fish) – attaches to TI, MEGALOBlastic ANEMIA
- praziquantel for all

Trypanosoma cruzi – Chagas, Central & South America:

- acute febrile illness → latent period. 30% develop chronic disease
- destroys GI neurons → cardiomyopathy, megaesophagus, MEGACOLON, VOLVULUS
- benznidazole or nifurtimox. Surgery for complications is palliative, NOT curative

FUNGAL COLITIDES

Histoplasma – Ohio & Mississippi valleys. Disseminated dz → deep ulcers, pseudopolyps, mimics IBD or CANCER

- tissue culture. IV amphotericin B; itraconazole second line

Candida – only with severe systemic illness. White plaques, budding yeast w/ hyphae. Fluconazole

Aspergillus – disseminated, GI bleeding. Voriconazole

Cryptococcus – colitis with SPONTANEOUS

PERFORATION, nodules mimic IBD. IV amphotericin B

VIRAL COLITIDES

CMV

dsDNA herpesvirus. Seroprevalence 31-70% of US children, up to 83% globally

Usually REACTIVATION of latent infection from altered T-cell competence

Highest risk: HIV, IBD, transplant. Also the elderly and critically ill immunocompetent

Sx: fever, malaise, pain, diarrhea, rectal bleeding, weight loss

Endoscopy: friability and ulceration. Can look ISCHEMIC, or be a polypoid mass mimicking CANCER

Dx is BIOPSY, not serology – seropositivity is near universal

- biopsy multiple sites INCLUDING the ulcer edges
- H&E: "owl's eye" inclusion with a clear halo, surrounded by INFLAMED cells = active
- inclusions surrounded by normal-appearing cells = colonization only
- H&E is insensitive → PCR or CMV immunohistochemistry if not confirmed

Rx: ganciclovir or foscarnet – IV first in the severely ill, then oral

Surgery for medical failure, persistent bleeding, or perforation

- most perforations b/n ileum and splenic flexure, often MULTIPLE
- resection WITHOUT anastomosis + fecal diversion
- mortality 31% in immunocompetent pts, higher if immunosuppressed

OTHER VIRUSES

Rotavirus, Norwalk, astrovirus, calicivirus, norovirus, sapovirus, adenovirus – mostly gastroenteritis

HSV-2 → STD proctitis. Oral acyclovir

THE IMMUNOCOMPROMISED HOST

IBD

Presentation and endoscopy are INDISTINGUISHABLE from an IBD flare

The trap: escalating immunosuppression for a presumed flare → sepsis and perforation

Stool studies and biopsies may MISS the superimposed infection – repeat them

CMV in IBD: incidence 10-33%, more common in UC than CD

- treat if severe Sx or steroid-refractory, even if CMV may not be the cause

Steroids allow progression and perforation, with or without underlying IBD

HIV

CD4 < 200/mm³ → significant risk of opportunistic infection

Diarrhea is the most common GI symptom. CMV the MOST COMMON opportunistic colitis

Stool studies + endoscopy with biopsies. Multiplex PCR

HAART raises the CD4 count and is part of the treatment

TRANSPLANT

Diarrhea in 22-52% of solid organ recipients

Distinguish infection from IMMUNOSUPPRESSANT side effects

422 admissions: most had NO identifiable etiology and were self-limited

- commonest identified: C. difficile, norovirus, CMV

Workup: CBC, renal & liver function, blood cultures if warranted, multiplex stool PCR

- colonoscopy if Sx persist and the preliminary workup is negative

52 BENIGN COLORECTAL DISEASE: CLOSTRIDIUM

DIFFICILE INFECTION

EPIDEMIOLOGY & MICROBIOLOGY

Clostridioides (formerly *Clostridium*) *difficile* – anaerobic, spore-forming, Gram (+) rod

- renamed 2016 – “*C. difficile*” kept for name recognition

Causes ≥ 20% of all antibiotic-associated diarrhea

Now the MC hospital-acquired infection – has surpassed MRSA

CDC: 4-fold ↑ 1993 → 2009; > 200% ↑ in pts > 65

Cost \$8,911-30,049 per admission, ~\$3 billion/yr to the US system

TOXINS

PaLoc – 19.6 kb pathogenicity locus, carries *tcdA* & *tcdB*

- toxin genes are of bacteriophage (viral) origin

Toxin A (308 kDa) & Toxin B (269 kDa) – large clostridial toxins

- monoglucosylate Rho GTPases → cytoskeletal depolymerization → colonocyte cytopathy
- A & B present in virtually ALL symptomatic human infection

Binary toxin (CDT) – encoded OUTSIDE the PaLoc

- ADP-ribosyltransferase; drives microtubular protrusions, ↑ membrane surface area 5x → ↑ adherence
- present in up to 6% of isolates

RIBOTYPE 027 (NAP-1)

Ribotyping = restriction enzymes on the ribosomal intergenic spacer region

> 15x the toxin A & B production of other strains, and usually makes binary toxin

- ↑ toxic colitis, ↑ mortality; > 200 genes not found in other strains

MC strain in pts admitted from long-term care (OR 4.87 vs. admitted from home)

Falcone: in 027(+), *metronidazole* monotherapy a/w recurrence (HR 2.38) → use *VANCOMYCIN*

Ribotype 078 – virulent, zoonotic link. Soil, animal & human reservoirs

CLINICAL RISK FACTORS

Antibiotics:

- the most consistent MODIFIABLE risk factor
- clindamycin (OR 2.8) & carbapenems strongest
- 2nd-4th gen cephalosporins OR 3.2, fluoroquinolones OR 1.6
- ANY antibiotic, ANY duration – stop the inciting agent as soon as CDI is diagnosed
- Balch: peri-op abx continued > 24 h → 6.7-fold ↑ CDI

Age:

- older pts – more severe colitis, more failure of 1st-line Rx
- comorbidity likely matters more than age itself
- Louie: cure ↓ and recurrence ↑ with each successive decade

Immunocompromise:

- HIV: CD4 ≤ 50/mm³ is the strongest risk – aOR 5.2-27.6
- transplant & oncology pts – mostly via the usual risk factors

IBD:

- presents during an acute flare; ↑ morbidity, mortality, need for surgery
- risk: low albumin, Hgb < 9, active colitis, biologics, antibiotics
- 6-fold ↑ need for colectomy. TNFRSF14 locus a/w 6-fold ↑ CDI
- TEST every IBD pt with severe colitis, and treat with *VANCOMYCIN*

Proton Pump Inhibitors:

- OR 1.6-1.7. MC risk factor in community-acquired CDI (31% of cases)
- omeprazole stimulates toxin production in vitro
- evidence is poor – use judiciously, do NOT globally discontinue

Healthcare Contact:

- fecal-oral transmission; ↑ bed occupancy a/w risk
- hand washing with SOAP, not alcohol gel – spores
- *Aquina*, 150,878 NY colectomy/proctectomy pts: CDI 0-11.3% by surgeon, 0-6.8% by hospital. Patient factors explained only 24% of the variation, ~70% unexplained hospital factors

Bowel Prep:

- MBP + oral abx does NOT ↑ CDI – incidence actually LOWER, and ↓ SSI & leak

Probiotics:

- NOT established for primary prevention – heterogeneous data, baseline incidence < 3%

DIAGNOSIS

Diarrhea is the hallmark, and is typically NON-bloody

Keep INFECTION distinct from the CARRIER state (stool positive, no symptoms)

- 16.4% of geriatric pts w/o diarrhea are PCR (+); 16% of those go on to symptomatic CDI
- pts who form antibody to toxin A tend to remain asymptomatic carriers

Severe: distention, colicky pain, tachycardia, hypotension

- exaggerated leukocytosis, often > 30,000

Peritonitis, localized or generalized = INDICATION FOR SURGERY

CT: wall thickening + pericolic stranding; transudative ascites w/ severe dz

- pneumoperitoneum & portal venous gas – fulminant, on pressors, non-viable colon

DIAGNOSTIC TESTS

Culture, then CCCNA (cell culture cytotoxicity

neutralization) – historic, sens as low as 65%

Toxigenic culture – outdated in practice, but still the reference standard for new tests

GDH Immunoassay:

- glutamate dehydrogenase – ubiquitous, present in toxigenic AND non-toxigenic strains
- sens 80-100%, excellent NPV, cheap, fast – a SCREEN. NOT specific

Toxin EIA:

- the confirmatory test – detects the TOXIN, so it means disease
- GDH + toxin EIA costs less than PCR, results < 24 h

NAAT (PCR):

- amplifies tcdA / tcdB – more sensitive than EIA, but 2-3x the cost
- detects the GENE, not the toxin → will be positive in CARRIERS

Algorithm: GDH (-) → stop. GDH (+) → toxin EIA.

GDH(+)/EIA(+) with symptoms = CDI. GDH(+)/EIA(-) → NAAT

Do NOT retest to prove cure – DNA lingers up to 30 days

SEVERITY CLASSIFICATION

IDSA 2017, initial episode:

- Non-severe: WBC < 15,000 AND Cr < 1.5
- Severe: WBC > 15,000 AND Cr > 1.5
- Fulminant: shock, ileus, or megacolon

ACG:

- Severe: WBC ≥ 15,000 OR albumin < 3
- Fulminant: ICU, T ≥ 38.5, ileus, distention, AMS, WBC > 35,000 or < 2,000, lactate > 2.2, or any organ dysfunction

ANTIBIOTIC THERAPY

Metronidazole is NO LONGER first line

- Zar RCT: severe dz – vanco cure 97% vs. metronidazole 76% (p = 0.02); no difference in mild dz
- Cochrane, 22 studies / 3,215 pts: vanco > metronidazole, fidaxomicin > vanco
- IDSA: restrict metronidazole to an initial NON-severe episode when nothing else is available
- ONE course only – neurotoxicity with prolonged or repeated use

First episode, non-severe – either, x 10 days:

- Vancomycin 125 mg PO q6h
- Fidaxomicin 200 mg PO q12h

Fulminant:

- Vancomycin 500 mg PO q6h
- + metronidazole 500 mg IV q8h
- ileus → vancomycin retention enema, 500 mg in 100 cc NS q6h (weak data)

Fidaxomicin:

- macrocyclic, narrow spectrum – inhibits sigma subunit of RNA polymerase
- non-inferior to vanco for cure, LOWER recurrence (in non-NAP-1 strains)
- EXTEND: extended-pulsed fidaxomicin – 11% better cure at 30 days vs. vanco

- cost is the only real barrier

De-escalate or stop every non-CDI antibiotic – outcomes worse if the inciting agent continues

Resuscitate to euvolemia before grading severity. TREND organ function, don't chase absolute cutoffs

RECURRENCE & FMT

Fecal Microbiota Transplant:

- cure > 80% for RECURRENT CDI – superior to conventional antibiotics
- commercially screened & prepared stool – endoscopic, NG, or capsule
- no longer experimental, inpatient or outpatient
- NOT first line for a primary episode
- INSUFFICIENT data for fulminant CDI – recrudescence is the problem; add vancomycin if used there

Bezlotoxumab: monoclonal ab to toxin B, single infusion alongside standard Rx – ↓ recurrence. Adjunct only, never monotherapy

SURGERY

Up to 10% of CDI pts come to surgery

Surgical consult AT RECOGNITION of severe/fulminant dz – not after medical failure

Indications: peritonitis, perforation, shock, clinical deterioration, failure of maximal medical Rx

Total Abdominal Colectomy + End Ileostomy:

- the traditional procedure of choice, largest body of evidence
- Bhangu, 31 studies: survival benefit vs. continued medical Rx
- pre-op septic shock predicts post-op mortality
- 2nd systematic review, 510 pts: pooled OR for mortality 0.70 with total colectomy
- PARTIAL colectomy – AVOID. 16% reoperation to resect more colon

Diverting Loop Ileostomy + Antegrade Lavage (Neal, 2011):

- loop ileostomy, intra-op colonic lavage w/ 8 L polyethylene glycol, then post-op vancomycin per stoma
- 83% completed laparoscopically, shorter operation
- mortality 19% vs. 50% for colectomy (historical controls, matched APACHE)
- leaves the colon → far higher likelihood of restored continuity
- NIS 2011-2015: use rose from 11% to 25% of CDI operations
- EAST multicenter: unadjusted mortality 23.8% diversion vs. 33.8% colectomy (p = 0.44)
- propensity-adjusted 17.2% vs. 39.7% (p = 0.002)
- but NO advantage in sepsis, renal failure, ALI, reoperation, or overall mortality
- an acceptable ALTERNATIVE, not yet a replacement – an infected colon is left in situ

53 BENIGN COLORECTAL DISEASE: RADIATION, MICROSCOPIC, AND ISCHEMIC COLITIS

RADIATION COLITIS

Biphasic injury – acute and delayed

Acute: during therapy or within 6 months – cell loss of the rapidly turning over superficial mucosa

- 50-75% of pts receiving pelvic XRT
- diarrhea (MC), mucous d/c, cramping, tenesmus, urgency, incontinence, pain
- RESOLVES as the epithelium turns over

Chronic: acute symptoms persisting > 6 months, or new symptoms after a latent period

- progressive obliterative endarteritis & submucosal fibrosis → chronic ischemia
- MC presentation 8-12 months after treatment; reported as late as 30 yrs
- hemorrhagic proctitis is the MC manifestation – mild bleeding to exsanguination
- also stricture, and rarely perforation or fistula
- does NOT improve over time

DOSE & RISK

Incidence of chronic radiation colitis: historically 30%, now 1-5% with modern delivery

< 45 Gy rarely causes chronic colitis; > 70 Gy causes significant injury

- rectal CA long course: 45-54 Gy
- cervical CA: 45-50 Gy EBRT + brachytherapy focally escalating to 90 Gy

Risk: pre-existing IBD, DM, HTN, nicotine, PVD, adjuvant chemo, prior surgery

RVF – MC after cervical CA XRT. Rectourethral fistula – after prostate CA XRT

- both unusual after standard rectal CA treatment

RTOG / EORTC GRADING

Acute:

- 1: ↑ frequency or rectal discomfort, no medication
- 2: diarrhea needing parasympholytics, mucus not needing pads, pain needing analgesics
- 3: diarrhea needing parenteral support, severe bloody/mucous d/c needing pads, distention
- 4: obstruction, fistula, perforation, bleeding requiring transfusion

Late:

- 1: mild diarrhea/cramping, 5 BMs daily, slight discharge or bleeding
- 2: moderate diarrhea or colic, > 5 BMs daily, excess mucus, intermittent bleeding
- 3: obstruction or bleeding REQUIRING SURGERY
- 4: necrosis, perforation, or fistula

PREVENTION

Limit exposure – full bladder, position change, CT-based planning, IMRT, VMAT

Sucralfate, SCFA, probiotics, glutamine – NO clear benefit

Amifostine (free radical scavenger) – most promising, but not routine

Mesh or omentum to exclude small bowel – has NOT been shown to ↓ colitis or enteritis

- do NOT do a laparotomy for this purpose alone

DIAGNOSIS

No lab or imaging finding is specific

Hx + ENDOSCOPY = mainstay

- classic: pallor with telangiectasias and easy friability
- biopsy only if malignancy is in question – biopsy ↑ the risk of FISTULA

Path: acute – eosinophilic infiltrate, crypt atrophy, crypt abscesses. Chronic – small vessel vasculopathy, obliterative endarteritis, intimal fibrosis WITHOUT inflammation

MEDICAL

Sucralfate has the best data – 20 mL enema BID; 77% improved at 1 mo, 92% at 4 mo

- long-term self-administration of enemas is the practical limit

Metronidazole – ↓ symptoms in 2 studies, no defined regimen or durability

Vitamins A, E, C – small studies, little harm, not standard
Mesalamine, steroids, SCFA – insufficient data. SCFA may help the ACUTE phase only

Hyperbaric O₂ – effective. Oscarsson: 89% success in 39 pts

- 36-40 treatments – cost and time make it impractical. Endoscopic Rx equals or beats it

ENDOSCOPIC

Formalin – the authors' choice for hemorrhagic radiation proctitis:

- chemical necrosis, sclerosing the telangiectatic neovasculture
- Sharma: 50 mL of 4% sprayed via flex sig, 3 min x 3 = 9 min – 62% complete, 34% reduced
- Raman: 100 cc of 2% as an enema held 2 min – 48% complete, 30% reduced
- Haas: 10% on a 16" cotton applicator held 60 sec – 93% success in 100 pts
- 1-3 sessions. No bowel prep or sedation required
- PROTECT the perianal skin – lidocaine jelly

Argon Beam Coagulation:

- 1-2 L/min, 40-65 W, 1-2 sec pulses; depth of penetration 1-3 mm
- Sebastian 81% complete cessation; Zinicola 86% – failures were the most severe, salvaged w/ formalin
- Yeoh: ABC ≈ formalin
- 20% get asymptomatic rectal ulcers, which resolve

Others:

- bipolar electrocoagulation = ABC
- Nd:YAG – depth ~5 mm and variable → ↑ perforation
- RFA, cryotherapy – very limited data

Strictures:

- firm, stretch poorly. Endoscopic balloon dilation, or Hegar if distal & transanally reachable

SURGICAL

LAST RESORT – for refractory bleeding, obstructing stricture, fistula, or disabling symptoms
 Altered planes, loss of tissue compliance, severe adhesions – difficulty tracks the degree of injury
 Counsel for small bowel injury, anastomotic leak, and fistula

- Lasser 1985, 85 pts: mortality 33%, enterocutaneous fistula 30%
- Mayo 1950-83: complication rate 80%, mortality 14%
- Zelga, radiation RVF: resection possible in only 4%
- Zhong, 26 radiation RVF: resection in 10/26, and only 3 were ever reversed

Diversion alone improves QOL, incontinence, obstruction and fistula symptoms

- but bleeding, pain, mucus and tenesmus PERSIST

Resect if health status allows – it removes all active disease

A PERMANENT ostomy is often required

MICROSCOPIC COLITIS

Chronic, relapsing, NON-bloody watery diarrhea with a grossly NORMAL colon

10-15% of pts scoped for consistent symptoms will have it

Incidence rising: 3.1 (collagenous) & 5.5 (lymphocytic) per 100,000 in the late 1980s → 19.6 by 2001

- some recent series as high as 219/100,000

Mean age at Dx 61. Female predominance, OR 2-8

40% have another autoimmune disorder

- polyarteritis: OR 20.8 for collagenous, 8 for lymphocytic
- 40% of celiac pts show MC histology

ASSOCIATIONS

Smoking – ↑ incidence & earlier Dx (OR 2.4 collagenous, 3.8 lymphocytic)

Drugs: NSAIDs (OR 3.8 / 4.7), PPI (OR 6.4 / 2.7), SSRIs, statins

- causation NOT established – these drugs cause diarrhea, which prompts the scope
- still, STOP the suspect drug as the first step

Prior Yersinia enterocolitica and prior C. diff proposed as triggers

DIAGNOSIS – SPANISH MICROSCOPIC COLITIS GROUP

- chronic or intermittent NON-bloody diarrhea, AND
- macroscopically normal or near-normal mucosa, AND
- characteristic histopathology

FULL colonoscopy is MANDATORY – flex sig is not enough

- 2 biopsies from EVERY segment; sampling rectum or sigmoid alone ↓ sensitivity

No lab test is diagnostic. Calprotectin ↑ (175 vs. 28 mcg/g) but nonspecific

Ddx: IBD, IBS, bile acid malabsorption, celiac, SIBO, lactose intolerance

HISTOLOGY

Lamina propria inflammation – lymphocytes & plasma cells. Crypt architecture PRESERVED

Collagenous: subepithelial collagen band $\geq 10 \mu\text{m}$

- band thins with treatment, but symptoms track the lamina propria inflammation, NOT the band

Lymphocytic: ↑ surface intraepithelial lymphocytes; collagen layer $< 10 \mu\text{m}$

- CD3 stain if H&E is equivocal

Same presentation, same management – minimal difference between the two

COURSE & TREATMENT

75% achieve remission – no symptoms, no medication, x 1 yr. Spontaneous remissions last longer

NO ↑ colorectal CA risk – actually a ↓ adenoma incidence

Stop the offending drug. Stop smoking

Loperamide / diphenoxylate:

- symptomatic only – no remission, no histologic change.

For mild disease

Budesonide:

- MAINSTAY, and the only evidence-based treatment
- 9 mg/day induction, 4.5 mg/day maintenance. Extensive first-pass → few systemic effects
- Miehke RCT: budesonide 68% vs. mesalamine 26% vs. placebo 21%
- Munch: 84% induction; 61% still in remission at 1 yr on maintenance vs. 16% placebo
- recurrence is common once stopped – restart it

Bismuth subsalicylate:

- 262 mg, 3 tabs tid x 8 weeks; response at 2 weeks. Meta-analysis 81% response

Not supported:

- mesalamine – failed vs. placebo in the large RCT. Probiotics, antibiotics – no
- prednisolone works but is NOT superior to budesonide and has more side effects

Immunomodulators:

- 6-MP / azathioprine, anti-TNF – small series only, for refractory disabling disease

Surgery:

- LAST resort. Diversion ± resection; both improve symptoms
- inflammation and the collagen band regress in the diverted segment
- SYMPTOMS RECUR when the ostomy is reversed

ISCHEMIC COLITIS

NOT the same disease as mesenteric ischemia

Mesenteric ischemia: sudden onset, named-vessel accident, TOTAL loss to the segment, pain out of proportion → EMERGENT SURGERY

Ischemic colitis: onset over hours, multifactorial, TRANSIENT low flow in the small vessels near the bowel wall, moderate localized tenderness + hematochezia → mostly MEDICAL

ANATOMY & EPIDEMIOLOGY

Splanchnic vessels are the most reactive in the body – take 10-35% of cardiac output

Marginal artery of Drummond parallels the colon and gives off the vasa recta

Watershed: Griffith's area (splenic flexure), Sudeck's point (rectosigmoid)

- if Drummond is not intact, a low-flow state → symptomatic ischemia

Colon is the MC site of GI ischemia. Incidence 4-9 per 100,000

- COPD: up to 100 per 100,000, RR 2.6-4.3

After AAA repair: 0-4.6% elective, up to 21% after RUPTURED AAA

Younger pts – collagen vascular disease, hematologic disorders, long distance running, cocaine

PRESENTATION

Abdominal pain + rectal bleeding are the earliest symptoms

- 15-20% of all lower GI bleeds – 2nd only to diverticulosis
- also nausea, vomiting, distention, dizziness, syncope

Right-sided colitis – LESS likely to bleed. Keep it in mind for isolated right-sided pain

Exam usually only mild-moderate tenderness over the involved segment

- peritonitis only once full-thickness necrosis and perforation occur

Many instances are self-limited. PAIN is a worse prognostic factor than melena

DIAGNOSIS

No reliable marker. ↑ WBC & acidosis suggest full-thickness ischemia

- rule out infectious bloody diarrhea – Klebsiella, Salmonella

Plain film: thumbprinting or pneumatosis in only 20-30%

CT: accuracy 74-79%; nearly 100% of acute IC has SOME finding

- pericolonic & free fluid, change in bowel wall density, wall thickening
- pneumatosis in < 5%. Pneumatosis + wall necrosis → mortality > 70%

NO contrast enema in the acute phase – distention worsens the ischemia

- useful later to assess a stricture

Colonoscopy confirms and excludes other colitides – NOT if peritonitis or free perforation

- erythema 84%, edema 70%, superficial ulcer 57%, friability 43%, deep ulcer 22%, stenosis 8%, intraluminal blood 8%
- submucosal bluish-black blebs/nodules = the thumbprinting

Path: GHOST CELLS are pathognomonic. Superficial crypt necrosis with the DEEP crypts SPARED, preserved architecture, mucin depletion

MANAGEMENT

Conservative unless perforation or toxicity – bowel rest, IV fluids, broad-spectrum antibiotics

- abx do not alter the disease; cover gut flora incl. anaerobic coliforms

- ischemia ↑ mucosal permeability and translocation

NO anticoagulation

Stop splanchnic vasoconstrictors, optimize cardiac output, NG if ileus

- no drug ↑ colonic mucosal blood flow

Improvement expected in HOURS TO DAYS – failure to improve = progression to transmural

Indications for Operation:

- fever, hypotension, tachycardia, ↑ tenderness, ↑ WBC = progression
- suspected infarction → explore
- resect NORMAL bowel to NORMAL bowel; extent driven by involvement
- AVOID primary anastomosis – do the safe operation, reverse the stoma electively
- mortality up to 40% for anyone requiring an operation

PROGNOSIS

Resolves in 24-48 h; full clinical & radiographic resolution in 1-2 weeks

Scope electively after recovery to confirm healing and look for stricture

~10% develop an obstructing stricture months later

- non-significant strictures may resolve on their own over 12-24 months
- symptomatic → ELECTIVE resection, plan on a primary anastomosis

20-30% develop chronic colitis from irreversible injury – diarrhea, bleeding, weight loss

- also an indication for elective surgery

54 BENIGN COLORECTAL DISEASE: INTESTINAL

STOMAS

PHYSIOLOGY & OUTPUT

~120,000 stomas created annually in North America;
450,000-800,000 ostomates
NSQIP: 37% unadjusted complication rate for ELECTIVE cases with a stoma, 55% for EMERGENCY
Colon gets 1500-2000 mL of liquid stool/day, reabsorbs ~90% (1350 mL), excretes 100-150 mL
- colonic transit 24-150 h → distal colostomies function PERIODICALLY; gas passes continuously
Sigmoid/left colostomy – solid. Transverse/ascending – liquid, behaves like a small bowel stoma
Small bowel 275-850 cm, mean 500 cm; receives 9-10 L/day
- jejunum reabsorbs ~6 L, ileum 2.5 L. Most nutrient absorption in the first 150 cm
Normal end ileostomy output 200-1200 mL/day
Diversion > 100 cm proximal to the ICV → steatorrhea and B12 deficiency
Even a new TERMINAL ileostomy runs high transiently – the ICV and colon are bypassed

PREOPERATIVE

STOMA SITE MARKING

ASCRS & WOCN recommend it. ↓ complications, ↑ stoma-specific and overall QOL, ↓ cost
- a properly chosen location is the single most important predictor of QOL after a stoma
Mark STANDING, SITTING, and LYING – folds and creases only appear when the pt sits
“Stoma triangle”: ASIS, pubic tubercle, umbilicus – site at the geometric center, within the rectus
Alternative: summit of the infraumbilical fat pad where it meets the rectus sheath
Within the rectus sheath – may ↓ parastomal hernia, though this has been challenged
Needs a 2-inch perimeter of clear, intact skin
AVOID scars, folds, creases, umbilicus, costal margin, ASIS, pubic symphysis, beltline
Pt MUST see the stoma sitting – large pannus → move supraumbilical (the wall is thinner there too)
Two stomas (GU + fecal) – opposite sides, DIFFERENT levels, so a belt still works

PREOPERATIVE EDUCATION

By a WOCN, BEFORE surgery – guideline recommended
Chaudhri: two 45-min pre-op visits – proficiency 9 → 5.5 days, LOS 10 → 8 days, saved \$2,104/pt

CONSTRUCTION

END SMALL BOWEL STOMA

Excise a 2-cm skin disk. Do NOT core out the subcutaneous fat
3-cm VERTICAL anterior sheath incision, split the muscle, 3-cm vertical posterior sheath

Pull 5-6 cm of bowel + mesentery above skin. Pull more than you think you need

Brooke eversion to protrude 2-3 cm

- height is INVERSELY proportional to complications; < 2 cm is a problem

RCT, full-thickness (dermal + epidermal) vs. intradermal Brooke sutures: excoriation 52% → 41%, but no difference in QOL, cost, or complications

LOOP SMALL BOWEL STOMA

Incise the DISTAL limb near-circumferentially at skin level.

Do NOT divide the mesenteric side

Evert the distal “hood” over the proximal limb; the PROXIMAL limb protrudes 2-3 cm

Stoma Rod – out at 3-5 days, never under posterior tension:

- 2019 meta-analysis: NO change in retraction, and ↑ necrosis & peristomal dermatitis
- conditional use only

END COLOSTOMY

Treat the conduit LIKE AN ANASTOMOSIS – no tension, proven perfusion

- sigmoid may need little; a descending colostomy may need full splenic flexure takedown + high ligation

3-4 cm above skin on the table; mature to protrude 1-2 cm – solid effluent is not caustic

Perfusion Assessment – do SOME form of it, every time:

- ICG near-infrared fluorescence – fast and highly accurate
- dusky but ICG-perfused = VENOUS congestion from a tight trephine → enlarge the trephine
- an error here → sloughing, mucocutaneous separation, stenosis, retraction, necrosis

LOOP COLOSTOMY

Mature by incising along the LONG AXIS and sewing the cut edge to skin circumferentially

Dual lumen → proximal diversion WITH retrograde venting of the distal segment

- ideal for palliative diversion of an obstructing distal lesion

LOOP ILEOSTOMY VS. TRANSVERSE LOOP COLOSTOMY

ASCRS CPG: LOOP ILEOSTOMY PREFERRED for temporary diversion in most cases

Similar complication RATES, different complication PROFILES

- ileostomy: ↑ dehydration, readmission, post-reversal obstruction
- ileostomy: ↓ post-closure sepsis, ↓ pre- and post-op hernia, better QOL
- transverse colostomy: large, cephalad, prolapse-prone, malodorous
- but favor T-colostomy for a distal obstruction needing retrograde venting, and in the obese

POSTOPERATIVE CARE

Stoma swells to 2-3x its size, shrinks to final size by 4-6 weeks

- the flange aperture must be re-trimmed SMALLER over those weeks

Colostomy: gas → liquid → solid. Small bowel stomas function SOONER

> 30% of new ileostomy pts become dehydrated; readmission > 15%

Half of stoma care is done by a spouse, a quarter by an offspring – TEACH THE CAREGIVER

ACS: only 53% of new ostomates could apply a pouch at discharge, only 28% could fix a leak

APPLIANCES

Trim the aperture to the EXACT size of the stoma – no skin exposed to effluent

A flange should last 3-5 days. More frequent = wrong appliance, wrong technique, or a bad stoma

- convex wafer for a flush or retracted stoma; belt for a leak-prone one
- paste or a barrier ring to “caulk” folds, creases, and mucocutaneous separation

PERISTOMAL SKIN

THE MC complication of an ostomy. Up to 70% of new ostomates

- > 60% had irritation at the first WOCN visit; nearly HALF did not know it
- almost all of it is a poorly fitted or improperly sized appliance

Fungal: red shiny patch with SATELLITE papules under the flange – Candida. Nystatin powder rubbed in, brush off excess, seal over it. Clears in 1-2 weeks

PERISTOMAL PYODERMA GANGRENOSUM

0.5-5% of IBD pts, ~0.6% of all ostomates. 20% of PPG pts have NO IBD

Painful ulcer, violaceous UNDERMINED border, epidermal bridges. Dx is CLINICAL

Rx the underlying IBD. Topical / intralesional / systemic steroids, immunomodulators, biologics

- intralesional steroid + gentle debridement: 40% complete, 40% partial healing
- > 50% of treated pts ultimately need RESITING, and resiting does not prevent recurrence

PERISTOMAL VARICES

Portosystemic shunt at the mucocutaneous junction. 27-50% of ostomates with portal HTN

40% bleed and require transfusion; mean 70 months from stoma creation to first hemorrhage

- local Rx – pressure, epinephrine gauze, suture ligation – 85% REBLEED
- TIPS is 5x more effective than local measures and cures bleeding in ~80%

PSC + UC → do the IPAA and avoid the stoma

- 53% variceal bleed within 4 yrs of TPC with end ileostomy

STOMA COMPLICATIONS

Retraction 0-22%. Parastomal hernia 0-40%. Prolapse 0-10%. Necrosis 0-7%. Peristomal skin 10-42%. TOTAL 12-72%

Early – leakage, dermatitis, dehydration – fixed with stoma care and education

Late – prolapse, stenosis, parastomal hernia – may need an operation

NECROSIS, RETRACTION, STENOSIS

Arterial insufficiency is the MC cause; venous ischemia from a tight trephine is rare

LOOP stomas resist ischemia better than END stomas – collateral flow from both sides

TEST TUBE exam at the bedside – lubricated glass tube + flashlight to find the proximal extent

- ischemia BELOW the fascia → REVISE. It will progress to intraperitoneal necrosis & perforation
- colostomy viable above the fascia → OBSERVE; solid stool pouches even if it ends up flush
- permanent end ILEOSTOMY with muscularis ischemia → revise early; a flush ileostomy can't be pouched

Chronic low-grade ischemia → late stenosis and retraction

- asymptomatic stenosis or retraction with a good seal → OBSERVE
- skin-level colostomy stenosis → local revision. SUBFASCIAL → open, bring out new bowel

PROLAPSE

Proximal bowel intussuscepts through the matured stoma.

Fixed vs. SLIDING (the problem variant)

- up to 8% of end stomas, 47% of loop stomas

Minimally symptomatic → reassurance, appliance change, external fixation device

- prolapsing TEMPORARY loop → just REVERSE IT
- intra-abdominal pexy of mesentery/conduit – for recurrence or a concurrent parastomal hernia
- a prolapsing loop colostomy is usually the DISTAL limb → convert to end or loop-end

Incarcerated → ice and TABLE SUGAR to pull the edema out. STRANGULATED = immediate surgery

PERISTOMAL ABSCESS – EARLY FROM CONTAMINATION, LATE FROM PENETRATING CROHN'S

Drain REMOTE from the flange, or straight through the mucocutaneous junction

NEPHROLITHIASIS

Na, water & bicarb loss → ↓ urinary pH. 8% vs. 4% in the general population

- 60% are uric acid (10% in gen pop); also ↑ calcium oxalate – avoid high-oxalate foods

HIGH OUTPUT STOMA

Normal ileostomy 800-1200 mL/day. HIGH is > 1200-2000 mL/day

- < 1200 mL usually tolerated; 1200-1500 is borderline

~HALF of post-op high output stomas resolve spontaneously within 2 weeks

Behavioral & Dietary:

- small frequent meals, separate solids from liquids, SIP don't gulp
- thickeners: bread, crackers, peanut butter, bananas; psyllium 1 tbsp BID
- restrict hypotonic fluid to 500-1000 mL/day and give ORAL REHYDRATION SOLUTION
 - WHO ORS per L: 3/8 tsp salt, 1/4 tsp salt substitute, 1/2 tsp baking soda, 2 tbsp + 2 tsp sugar
- most high-output pts avoid IV fluid on ORS alone

Medical, in order:

- loperamide 2-4 mg 30 min BEFORE meals & at bedtime (max 16 mg/day)
- + diphenoxylate/atropine 2.5-5 mg QID
- + antisecretory – H2 blocker or PPI
- + paregoric or codeine – costly, sedating, LAST

Work up persistently high output:

- partial SBO on CT or SBFT – paradoxically ↑ output
- small bowel Crohn's – enteroscopy through the stoma
- C. difficile ENTERITIS – stool testing
- steatorrhea after ileal resection → cholestyramine. Pancreatic insufficiency → enzymes
- short gut → TPN; octreotide, teduglutide, growth hormone

Bolus obstruction – nuts, seeds, shellfish, sausage casings, raw produce:

- irrigate via red rubber just past the fascia (< 6 in), 30-50 mL aliquots by Toomey. Takes 1-2 h

Early fascial-level obstruction – peristomal pain, thin non-bilious effluent or none:

- 14-18 Fr Foley/red rubber at the bedside past the fascia. Do NOT inflate the balloon

REVERSAL

PREOPERATIVE

- diversion colitis grossly and histologically MIMICS IBD

End colostomy for diverticulitis: confirm the stump is RECTUM, not sigmoid

- completion sigmoidectomy at the time of reversal if a sigmoid stump was left

TIMING

Loop stomas – typically 2-3 months

- Scandinavian RCT: closure at 8-13 DAYS as safe as 12 weeks after LAR in selected pts, with FEWER complications and no QOL difference at 3, 6, 12 mo – but 70% of screened pts were ineligible
- authors default to 8-12 weeks; early closure only in highly selected pts with leak excluded

Adjuvant chemo: dogma keeps the stoma through treatment

- but chemo compounds dehydration – readmission in up to 11%

End stomas – delay 3-6 months; adhesions become less tenacious and less vascular

- early (< 3 mo) Hartmann reversal a/w ↑ leak, sepsis, and death

TECHNIQUE

- protect the mesentery – marginal artery injury on a loop COLOSTOMY → distal colonic ischemia
- ASCRS CPG: stapled and hand-sutured closure are BOTH acceptable
- skin: PURSE-STRING closure – significantly lower wound infection, better satisfaction

End stoma reversal is often a BIGGER operation than the index case

- mark a new stoma site pre-op in case a protecting loop is needed

REVERSAL COMPLICATION RATES – LOOP ILEO / LOOP COLO / END COLOSTOMY W/ COLORECTAL ANAST

Superficial SSI: 3-13.5% / 5-20% / 14-43.8%

Deep space or leak: 2-4% / 2-4% / 1.5-21%

Obstruction or ileus: 5-16% / 4% / 23%

Clinically diagnosed hernia: 0-50% / 2-38% / 3-31%

THE DIFFICULT STOMA

Obesity ↑ complications – the pts needing the best stoma get the hardest one to make

The supraumbilical wall is THINNER in the obese.

Subxiphoid T-loop colostomy in the super obese AVOID subcutaneous lipectomy – it leaves a recessed, unpouchable stoma

LOOP-END STOMA – preserves the mesentery to the entire conduit and buys reach

Prasad end-loop: both limbs exteriorized when anastomosis is inadvisable – later reanastomosis is LOCAL, no laparotomy

“Pie crusting” – score the mesenteric peritoneum PERPENDICULAR to the vessels; 2-3 cm of reach

Ligate the ileocolic proximally for reach on a terminal end ileostomy

A poor stoma in a good LOCATION beats a good stoma in a poor location

TURNBULL BLOWHOLE COLOSTOMY

For toxic megacolon when the colon is tissue-paper thin and resection means contamination and death

Transverse colon loop out the midline – close fascia around it, then a WATERTIGHT seromuscular-to-peritoneal suture line BEFORE opening the colon

Expect prolapse and pouching problems – it is a BRIDGE, not a definitive operation

55 BENIGN COLORECTAL DISEASE: ABDOMINAL WALL

RECONSTRUCTION AND PARASTOMAL HERNIA

REPAIR

INCIDENCE & RISK FACTORS

120,000 ostomies created/yr in the US; ~800,000

Americans living with a stoma

PSH = an incisional hernia at the ostomy site – the MC complication of a stoma

Rates ~30% at 1 yr, ~40% at 2 yrs, > 50% with longer f/u

- occurrence plateaus after the first 2 post-op yrs
- median time to presentation 18 mo, range out to 27 yrs
- with long enough f/u some argue a PSH is inevitable

By stoma type: end colostomy HIGHEST → end ileostomy
≈ loop colostomy → loop ileostomy lowest

Enlarged trephine at creation a/w PSH – minimize the defect w/o strangling the bowel

Israelsson definition: "any palpable defect or bulge adjacent to the stoma detected when the pt is supine with legs elevated, or while coughing or straining when erect"

- f/u stoma pts ≥ 1 yr to capture occurrence
- inter-observer reliability among staff surgeons is poor

Risk factors: age, DM, SSI, malnutrition,

immunosuppression, malignancy, emergent case

- OBESITY and waist circumference carry the strongest association
- Swedish registry, > 6300 permanent colostomies: BMI > 30 the ONLY risk factor
- waist circumference > 100 cm → 75% probability of a PSH

SYMPTOMS & DIAGNOSIS

Pain 35%, appliance difficulty 28%, leakage 27%, irrigation difficulty 24%, skin irritation 22%

Complications: prolapse 18%, obstruction 15%, hemorrhage 2%

Self-report is a good screen – 76% of self-identified PSH were symptomatic, 56% went on to repair

CT for operative planning: concomitant incisional hernia, obstruction, defect size → need to re-site

- supine CT OVERestimates by calling redundant subcutaneous bowel a hernia
- supine CT also UNDERestimates the bulge seen standing/prone or w/ Valsalva
- pt symptoms track the need for operation better than the scan does

CLASSIFICATION

None validated, none widely adopted

Rubin & Bailey: I true PSH (sac through a dilated stomal canal) / II intrastomal / III subcutaneous prolapse / IV pseudohernia (denervation of the lateral abd wall)

Devlin: I interstitial / II subcutaneous / III intrastomal / IV peristomal (w/ prolapse)

Moreno-Matias: Ia sac < 5 cm / Ib sac > 5 cm / II sac contains omentum / III loop other than the stoma bowel

EHS (2014) – the one that drives decisions:

- I: < 5 cm, NO concomitant incisional hernia
- II: < 5 cm, WITH concomitant incisional hernia
- III: > 5 cm, NO concomitant incisional hernia
- IV: > 5 cm, WITH concomitant incisional hernia
- each also graded primary vs. recurrent
- the only 2 variables that matter: defect size, and a concomitant incisional hernia

OPERATIVE INDICATIONS

SELECTIVE repair – up to 70% of pts living with a PSH never pursue an operation

Repair for a PSH COMPLICATION (obstruction, prolapse) or QOL-limiting symptoms

- pouching failure and intermittent obstruction are what reliably drive pts to surgery
- pain alone is too variable to be an indication

ACHQC: only 22% of stomas were reversible at the time of the hernia repair

NO repair is very successful – recurrence > 50% with long f/u even WITH mesh

Each redo with prosthetic is harder – complication rates > 50%, re-operation 12%

PROPER (ESCP): standardized PSH QOL measure in development

PREOPERATIVE OPTIMIZATION

- BMI < 35-40 – offer surgery < 30, > 50 prohibitive, 30-50 ambiguous
- smoking cessation ≥ 4 weeks
- HbA1c < 7-8
- stoma marking by ET nurse in ALL pts, even when not planning to re-site
- NO bowel prep – no benefit, more watery spillage, may worsen wound morbidity in contaminated cases (ACHQC)

Counsel and reassess at 3-month intervals – hold the line, non-adherence is the pt's risk too

REPAIR TECHNIQUES

No RCT compares mesh repair techniques – all data is series

Primary Suture Repair:

- recurrence up to 80% – ABANDONED except in extremis

Relocation w/o Mesh:

- recurrence up to 86%, plus a new midline incisional hernia and hernia at the old stoma site (up to 52%)
- ipsilateral and repeat re-siting make it worse
- reserve for pts in extremis or urgent and sub-optimized

Onlay:

- incision ≥ 10 cm from the stoma to stay off the appliance
- extraperitoneal dissection over anterior rectus sheath / EO aponeurosis, 5-10 cm overlap

- close fascia to a 2-fingerbreadth aperture, then UNCOATED polypropylene
- keyhole slit if the stoma stays in situ, cruciate if it is mobilized off the skin
- AVOIDS laparotomy – small-medium defects, high-risk pt, hostile abdomen
- does NOT address a concomitant incisional hernia; skin flaps → ischemia & seroma (closed suction drains)
- Hansson pooled 149 pts: recurrence 19%, SSI 2%, mesh infection 2.6%
- Ho keyhole PP 7% / DeRuiter cruciate PP + PP ring 15% (but 26% needed the prosthetic removed) / Steele “stove pipe hat” 26%
- Warwick, keyhole PORCINE DERMIS: 90% recurrence at 3 yrs, median 10 mo to failure – do NOT use biologic here

Open Intraperitoneal:

- close the defect, ≥ 5 cm overlap, barrier-coated synthetic or ePTFE
- keyhole/cruciate vs. Sugarbaker orientation
- Al Shakarchi pooled 65 pts: recurrence 9% at 38 mo, SSI 3%

Sugarbaker:

- lateralize the bowel as it leaves the peritoneum, mesh over the hernia, bowel drapes b/n wall and prosthetic → flap-valve effect with ↑ intra-abd pressure
- original 7 pts: 0 recurrence at mean 5 yrs; Stelzner ePTFE n=20: 15% at 42 mo
- overcomes the keyhole failure mode – mesh shrinks, the orifice enlarges
- tradeoff is more bowel-prosthetic interface → erosion concern

Laparoscopic Intraperitoneal:

- PROCEDURE OF CHOICE for most – EHS I or III < 10 cm, no large concomitant defect
- 3-4 trocars, reduce contents, optional defect closure, 5 cm overlap in every direction
- DeAsis meta-analysis (15 studies): SSI 4%, mesh infection 1.7%, obstruction needing re-op 1.7%
- recurrence: Sugarbaker 10% (n=191) vs. keyhole 28% (n=231) → SUGARBAKER PREFERRED

Open Retromuscular (Sublay):

- retrorectus dissection to the semilunar line, re-site the stoma contralaterally, then close the posterior sheaths to exclude the viscera
- allows UNCOATED POLYPROPYLENE – cheaper, no visceral contact
- one mesh reinforces the NEW stoma site, the OLD stoma site and the midline
- best when a midline defect coexists or several prior repairs have failed
- early pooled 76 pts: recurrence 8% at 24 mo, infection 4%, NO mesh infection or excision

TAR / Posterior Component Separation:

- when the posterior sheaths will not reapproximate, or a large lateral defect leaves inadequate overlap
- divide transversus abdominis just MEDIAL to the laterally perforating neurovascular bundles
- carries the plane into the lateral preperitoneal space above the arcuate line
- extend to the psoas laterally, central tendon of diaphragm cephalad, space of Retzius inferiorly
- Raigani, TAR + keyhole (n=46): SSI 46%, recurrence 11% at 13 mo
- Beffa, retrorectus ± TAR (n=46): SSI 17%, but 40% if the stoma was re-sited; recurrence 22% at 22 mo
- Pauli, TAR + retromuscular SUGARBAKER (n=44): recurrence 4.5% at 10 mo, no erosion
 - Cleveland Clinic replication: mesh erosion into bowel 3/38 (8%) – modified to no longer fixate mesh adjacent to the stoma

STORM & SMART: 28 mm EEA stapler secures retromuscular mesh to both rectus sheaths – recurrence 17% and 18%

MESH CHOICE

Prophylactic mesh at stoma creation – multiple RCTs, uncoated PP in the sublay position

Synthetic mesh in a contaminated field is no longer a faux pas

- macroporous, monofilament, UNCOATED polypropylene is most resilient to bacterial burden
- AVOID multifilament and barrier-coated mesh where there is contamination
- restrict to OPTIMIZED pts, elective, CDC class II-III
- the retromuscular/TAR pocket puts well-vascularized tissue on both faces of the mesh

Warren 2020, n=402 synthetic in contamination (CDC II 53%, III 42%, IV 6%): SSI 14%, mesh excision 2.4%, recurrence 10% at 21 mo

RICH – porcine dermis, mean width 16 cm: SSI 35%, NO excision, recurrence 28% at 2 yrs

COBRA – Bio-A resorbable, CDC II/III: SSI 18%, NO excision, recurrence 17% at 2 yrs

Phasix (P4HB) – clean cases only: SSI 9%, NO excision, recurrence 9% at 18 mo

Biologic & biosynthetic → essentially no mesh excision but HIGHER recurrence

Synthetic → lower recurrence, durable, at the cost of a ~2% excision risk

ALGORITHM

In extremis or sub-optimized but must operate → primary closure

Not optimized, elective → counsel, reassess in 3 months

Optimized + EHS I or III < 10 cm → LAPAROSCOPIC SUGARBAKER

- open onlay instead if high risk or hostile abdomen

EHS II, III > 10 cm, or IV → open retromuscular ± TAR if
you are comfortable with it
- otherwise open or laparoscopic intraperitoneal mesh

56 PELVIC FLOOR DISORDERS: FUNCTIONAL DISORDERS AFTER COLORECTAL SURGERY AND IBS

IRRITABLE BOWEL SYNDROME

ROME IV CRITERIA

Recurrent abdominal pain ≥ 1 day/week over the last 3 months, PLUS ≥ 2 of:

- related to defecation
- a/w a change in stool FREQUENCY
- a/w a change in stool FORM

Criteria met for 3 months, with onset ≥ 6 months prior

- Rome III "pain or discomfort" and "relief with defecation" have been DROPPED
- IBS remains a Dx of abdominal pain + altered bowel habit
- Dx of EXCLUSION, but specificity and PPV approach 100% in the absence of red flags

Mean 6.6 yrs from symptom onset to a positive diagnosis

SUBTYPES – BY BRISTOL STOOL FORM

Threshold is 25% of bowel movements

- IBS-C: $> 25\%$ Bristol 1-2 (hard, lumpy)
- IBS-D: $> 25\%$ Bristol 6-7 (mushy, watery)
- IBS-M: $> 25\%$ of BOTH
- IBS-U: neither threshold met

Bristol form is a useful surrogate for colonic transit time – use it to pick therapy

PATHOPHYSIOLOGY

No reliable biomarker – heterogeneous, several mechanisms

Visceral hypersensitivity: chronic stimulation sensitizes peripheral gut afferents to normal stimuli

- peripheral sensitization progresses to CENTRAL sensitization, esp. with emotional distress or a trauma hx
- amplified peripheral pain + reduced central pain inhibition
- stress amplifies the maladaptive central response → symptom flares

CAPS (centrally mediated abdominal pain syndrome): near-continuous pain WITHOUT change in bowel habit – distinguishes it from IBS

Psych comorbidity (depression, anxiety) is a risk factor in care-SEEKERS only

- self-managed IBS carries the same odds as the general population

Severity spectrum (Drossman):

- Mild 40%: primarily bowel dysfunction, M = F, older, 0-1 visits/yr
- Moderate 35%: bowel + CNS pain dysregulation, F $>$ M
- Severe 25%: primarily CNS pain dysregulation, F $\gg$ M, younger, ≥ 7 other symptoms, catastrophizing, abuse hx, ≥ 5 visits/yr, work disability $\geq 11\%$

ALARM FEATURES

Abnormal exam, recent antibiotics, documented weight loss, NOCTURNAL symptoms, bloody stool, FHx of colon CA

WORKUP BY SUBTYPE

All subtypes: CBC + age-appropriate CRC screening

IBS-D (broadest differential):

- CRP or fecal calprotectin, IgA TTG $\pm$ quantitative IgA
- random biopsies if scoped – MICROSCOPIC COLITIS
- consider SeHCAT, fecal bile acids or serum C4 for bile acid diarrhea
- consider anti-CdtB / anti-vinculin if post-infectious IBS suspected
- stool studies incl. Giardia

IBS-M: same as IBS-D, plus a stool diary; plain film for fecal loading

IBS-C: refer for physiologic testing if severe or refractory; plain film for fecal loading

Bile acid diarrhea: up to 28.1% of pts labeled IBS-D – ileal resection or malabsorption in an anatomically normal small bowel

- GOLD STANDARD is SeHCAT, but not available in the US
- empiric bile acid sequestrant remains the usual diagnostic AND therapeutic test

Microscopic colitis: watery diarrhea, older, F $>$ M, endoscopically NORMAL mucosa – needs BIOPSY

- yield 2.3% in IBS pts ≥ 45 yrs – scope the older IBS-D woman

IBS AND SURGERY

Surgery has NO role in the treatment of IBS

IBS pts undergo far more cholecystectomy, hysterectomy and appendectomy

- most represent a pre-op misdiagnosis chasing chronic pain

Diverticular disease is the colorectal prototype

- diverticulitis sensitizes visceral afferents – damage, regrowth, altered function
- low-grade inflammation persists → SUDD (symptomatic uncomplicated diverticular disease)
- SUDD may not be separable from IBS; a/w somatization, skews OLDER
- functional complaints BEFORE elective sigmoid resection predict a poor outcome
- persistent symptoms after sigmoidectomy for diverticulitis in up to 25%, mirroring IBS despite inflammation in the specimen

Surgery may also PRECIPITATE IBS – new symptoms after abdominal operations in a meaningful number

TREATMENT

Reassure about the benign natural history. Explain the rationale – the pt-provider relationship is an INDEPENDENT predictor of improvement

Graded: education/reassurance/diet (mild) → +
pharmacotherapy & psych therapy (moderate) →
multidisciplinary + pain center (severe)
Placebo response in functional GI trials is high – active
over placebo is typically only ~10%

CONSERVATIVE & DIET

Exercise – mild improvement in severity, maintains
physical & mental health
Probiotics – mild benefit, LOW quality evidence; best data
for *Bifidobacterium infantis*
Peppermint oil – modest benefit, acts as an antispasmodic
- a rigorous RCT showed NO benefit by FDA/EMA
endpoints
Antispasmodics: use ON-DEMAND or prophylactically, not
scheduled

Fiber:

- SOLUBLE (psyllium, ispaghula, calcium polycarbophil)
> INSOLUBLE (corn, wheat bran)
- psyllium is the best-supported – laxation plus
fermentation, at the cost of gas
- IBS-C with bloating: switch to insoluble, NON-
fermentable cellulose or methylcellulose

Low FODMAP:

- 60-70% of IBS pts are worse with meals, > 70% blame
food, up to 90% avoid foods
- true food allergy is NOT more common; food
INTOLERANCE is, and is subtype-independent
- FODMAPs = poorly absorbed carbohydrates →
osmotic load + colonic fermentation
- symptoms recur off low-FODMAP even when gluten is
excluded – it is the FODMAPs, not the gluten
- elimination then systematic reintroduction – needs a
dietitian, adherence is the limit
- “FODMAP-gentle” = bottom-up, restrict only the
highest-yield foods
- lactase non-persistence in ~30% of Caucasians, ~70%
of Asians

IBS-D DRUGS

- loperamide: helps urgency and stool form, NOT global
symptoms or pain – use as prophylaxis
- bile acid sequestrant: cholestyramine first (titratable,
poorly palatable), then switch to colesevelam or
colestipol long term
- rifaximin: 14-day course, ~10% over placebo, no C. diff
or resistance signal; approved for up to 3 courses
- eluxadoline (mu/kappa agonist + delta antagonist):
pancreatitis and Sphincter of Oddi dysfunction, mostly
in pts WITHOUT a gallbladder
- alosetron (5-HT₃): women with refractory IBS-D only –
ischemic colitis and severe constipation, mitigated by a
low starting dose
- ondansetron (off-label) and ramosetron are the other 5-
HT₃s
- meta-analysis: ramosetron and alosetron > rifaximin
and eluxadoline for IBS-D/M

IBS-C DRUGS

Lies on a spectrum with functional constipation – first line
for both is fiber 20-30 g/day
PEG improves frequency, straining and consistency but
NOT abdominal pain

Secretagogues – all drive net Cl⁻ secretion with water
following:

- lubiprostone 8 µg bid
- linaclotide 290 µg daily
- plecanatide 3 mg daily
- similar efficacy on meta-analysis, no head-to-head;
diarrhea is the MC adverse event (lubiprostone also
nausea)
- choice is usually driven by payer coverage

NEUROMODULATORS

Underused. Downregulate visceral afferent signal
centrally, treat catastrophizing/anxiety, and exploit
their effect on transit

- IBS-D → TCA (most effective agent for pain);
anticholinergic effect is a bonus
- PREFER secondary amines nortriptyline &
desipramine over amitriptyline/imipramine
- “start low, go slow,” doses well below psychiatric
dosing, give it ≥ 2 weeks
- IBS-C / IBS-M → SNRI – duloxetine or milnacipran
over venlafaxine
- SSRI = adjunct for comorbid anxiety/depression,
WEAKER for pain than TCA
- gabapentin / pregabalin in IBS-C and IBS-M

CBT, relaxation therapy, hypnotherapy, psychodynamic-
interpersonal therapy all have evidence – access is the
limiting factor

CHRONIC PELVIC PAIN

Lower abdominal pain ≥ 6 months, unrelated to pregnancy.
Affects up to 11% of the population

IBS symptoms in up to 40% of women with chronic pelvic
pain

- a/w muscular back pain, age > 40, depression, 6-8 pain
sites, high symptom distress, hx of adult physical
abuse
- treat with neuromodulators, chosen to avoid worsening
the bowel habit

Pelvic floor dyssynergia is common in IBS – get
ANORECTAL MANOMETRY if suspected, then
biofeedback

- in non-IBS-D women with anal pain: absent relaxation
or paradoxical contraction 91%, inadequate straining
pressure 53%, prolonged balloon expulsion 47%

FUNCTIONAL ANORECTAL PAIN (ROME IV)

*Levator ani syndrome: rectal pain/aching ≥ 30 min WITH
tenderness on traction of the puborectalis (worse on
the LEFT)*

*Unspecified functional anorectal pain: same, but NO
puborectalis tenderness*

Proctalgia fugax: episodic, unrelated to defecation, seconds to 30 min, NO pain between episodes

All require 3 months of symptoms with onset $\geq$ 6 months prior

Levator Ani – Rx:

- worse sitting; imaging & physiology add nothing but exclusion
- digital massage of the puborectalis sling + hot sitz baths + diazepam: 68%
- RCT n=157, biofeedback vs. EGS vs. digital massage: BIOFEEDBACK SUPERIOR – 94% restored relaxation with strain, 97% expelled the balloon
- biofeedback best in those with defecatory difficulty (65% vs. 35% improved)
- steroid/lidocaine into the arcus tendon beat EGS short term, no difference at 12 mo
- Botox 100 U: RCT showed NO change in frequency, intensity or duration

Proctalgia Fugax – Rx:

- abnormal smooth muscle contraction – thicker IAS and higher resting pressure than chronic proctalgia
- familial form: IAS hypertrophy + constipation → strip myomectomy fixes the constipation, NOT the pain
- 55/68 have pudendal tenderness; nerve block relieved 65% completely
- reassurance, position change, warm bath, sublingual nifedipine or topical NTG, enema
- inhaled albuterol RCT: time to relief 14 min → 5 min
- refractory: biofeedback, local block, Botox 25 U, or LIS

COCCYODYNIA

Pain at/around the coccyx, worse with prolonged sitting on hard surfaces; chronic if > 2 months

Two subtypes: traumatic, and idiopathic (pelvic floor spasm, coccygeal bursitis, DJD, sacrococcygeal hyper- or hypomobility)

Obesity and female sex predispose – pelvic rotation puts the coccyx more posterior

Dx: reproduce the pain by external or transrectal manipulation of the coccyx

- lateral sacral films and dynamic sitting/standing x-rays; MRI only to exclude tumor/mets/DJD

90% improve NONoperatively – rest, donut or wedge cushion, NSAIDs, heat/ice

- coccygeal manipulation RCT: 36% vs. 20% improved at 1 month – neither impressive
- ganglion impar block – ADD corticosteroid, better pain and depression at 1 and 3 months

Coccygectomy: good/excellent relief in up to 85%; beats nonoperative care at 4 yrs

- wound infection in up to 25%

PUDENDAL NEURALGIA

S2-S4, motor and sensory. Passes b/n the sacrospinous and sacrotuberous ligaments at the ischial spine, then into ALCOCK'S CANAL

Entrapment sites: infrapiriformis canal, interligamentary space, Alcock's canal, subpubic dorsal nerve canal
Knifelike pain or foreign-body sensation in rectum, vagina or perineum; worse through the day, often unilateral; defecation triggers it minutes to an hour later

Nantes criteria (all 5 required):

- pain in the pudendal territory (anus to clitoris/penis)
- worse with SITTING
- does NOT wake the pt at night
- NO objective sensory loss (a perineal deficit suggests a sacral root lesion)
- relieved by pudendal nerve block
- exclusions: purely coccygeal/gluteal/hypogastric pain, purely paroxysmal pain, pruritus alone, imaging that explains the symptoms

MR neurography is gaining a role; only operative findings + post-op relief confirm the Dx

Treatment:

- CT-guided block into the sacrospinous ligament AND Alcock's canal: 63% at 1 mo, 50% at 3 mo, 25% at 6 mo
- adding corticosteroid to the block gives NO benefit over local anesthetic alone
- cryoablation 9/11 durable at 6 mo; lipofilling into Alcock's canal 10/15 pain-free at 12 mo
- pudendal neuromodulation > nerve block, and > sacral neuromodulation in 3/4 who had both
- conus medullaris stimulation after failed decompression: 100% of implanted were long-term responders, sitting time tripled
- decompression only if the block WORKED – a negative block predicts operative failure
- approaches: transgluteal, transperineal, transischioirectal, laparoscopic
- RCT n=32: decompression + transposition beat nonoperative care at 3 and 6 months; 8/10 successes held at 4 yrs

57 PELVIC FLOOR DISORDERS: COMMON TESTS FOR THE PELVIC FLOOR

ANORECTAL MANOMETRY

Fecal incontinence affects 8.4-14.4%; evacuation disorders 12-19% of North Americans

Half of parous women have some prolapse, 10-20% of them symptomatic

TEST ONLY after failure of first-line conservative care (stool consistency, lifestyle)

INDICATIONS

- fecal incontinence, constipation & obstructed defecation, anal pain, anal fissure
- Hirschsprung's Dz
- baseline before an operation that threatens continence (fistulotomy, LIS) or evacuation (rectopexy)
- after obstetric injury when the pt wants sphincter function quantified before another delivery
- biofeedback protocols for incontinence and obstructed defecation

EQUIPMENT

Water-perfused:

- 0.2-0.4 mL/min at a 10 psi head, amplifier ≥ 8 Hz
- pump and catheter transducers MUST be level for calibration and during the study
- cheap and simple, but CANNOT be used upright; artifact-prone

Solid-state (strain gauge):

- much higher frequency response $\rightarrow$ captures fast striated-muscle transients
- easy calibration, no leveling required, but FRAGILE and expensive

Air-charged:

- air column from a balloon to an external transducer $\rightarrow$ CIRCUMFERENTIAL, gravity-independent readings
- low cost, disposable, no high-level disinfection – the authors' own choice

HRAM (2008):

- 2 sensors in the rectal balloon, 10 sensors at 6 mm intervals along the canal
- > 6000 mmHg/s, accurate to 1 mmHg, 35 Hz. Susceptible to THERMAL DRIFT – compensated in software

HDAM (3D):

- 10.75 mm probe, 256 sensors – 16 rows x 16 circumferential
- sensed at 4 mm, interpolated to 1 mm, > 20 Hz, resolution 0.1 mmHg
- e Sleeve reduces many sensors to a single max value per time point; color topography replaces linear plots
- HDAM slightly more comfortable than HRAM; no comfort advantage of HRAM over water-perfused
- CPT does not reimburse HRAM/HDAM differently – the authors have NOT found the topography changes management

- Benezech: an anterior high-pressure area on HDAM + excessive perineal descent $\rightarrow$ rectal intussusception, PPV and specificity 100% vs. defecography

TECHNIQUE

Prep optional – enema only if DRE finds a loaded rectum.

No fasting; empty bowels 30 min prior

Left lateral, hips & knees at 90° . DRE first

Distal sensor OUTSIDE the anal verge; rectal balloon 3-5 cm above the top of the canal; recording length ≥ 6 cm

Rest 3-5 min after insertion for reflex contraction to abate

- ultraslow waves 1-1.5 cycles/min, amplitude ≥ 40 mmHg

- low-amplitude slow waves 10-20/min, distal canal – may move contents cephalad and aid continence

STATIONARY PULL-THROUGH is the method of choice – 1 cm increments from 6 cm to the verge

- CONTINUOUS pull-through (1 cm/sec) causes a reflex contraction $\rightarrow$ falsely HIGH pressures
- HRAM/HDAM need no pull-through at all – shorter study

RESTING PRESSURE

IAS contributes up to 80% of resting tone; also EAS and the hemorrhoidal cushions

Record ~ 3 min after insertion, over 20-30 seconds

- Women: 52 mm Hg (39-65). Men: 59 mm Hg (47-71)

High-pressure zone: the length of canal where pressure exceeds HALF the maximum resting pressure

Lower in women, and falls with age in women

LOW $\rightarrow$ passive fecal incontinence. HIGH $\rightarrow$ anal fissure or constipation

SQUEEZE PRESSURE

Voluntary EAS contraction – maximal squeeze held as long as possible, measured over 30 sec

- Women: 128 mm Hg (83-173). Men: 228 mm Hg (190-266)

- squeeze increment = mean squeeze – mean resting pressure

- endurance: hold > 30 sec at $> 50\%$ of max

- lower in women and in older women

- HDAM can separate the puborectalis contribution, but is NOT better at identifying puborectalis or EAS injury

COUGH REFLEX

Sudden $\uparrow$ intra-abdominal pressure $\rightarrow$ reflex EAS

contraction. Tests the SACRAL REFLEX ARC

- PRESERVED with cord lesions above the sacrum
- ABSENT in cauda equina lesions and pudendal neuropathy

PUSH / SIMULATED EVACUATION

Normal = $\uparrow$ rectal pressure WITH simultaneous $\downarrow$ anal pressure. 3 pushes x 15 sec, 30 sec apart, balloon deflated

Rao classification of dyssynergic defecation:

- I: adequate push + PARADOXICAL anal contraction
- II: inadequate push + PARADOXICAL anal contraction

- III: adequate push + absent or incomplete (< 20%) anal relaxation
- IV: inadequate push + absent or deficient anal relaxation

Manometry ALONE cannot diagnose dyssynergia – up to 90% of asymptomatic controls show the pattern

- 2/2 the non-physiologic left lateral position and an empty rectum
- a negative rectoanal gradient is present in most asymptomatic women

Defecation index = max intrarectal pressure / anal residual pressure. NORMAL ≥ 1.2

RAIR

Rectal distension → transient EAS contraction, then IAS RELAXATION. Mediated by the MYENTERIC PLEXUS

Inflate the rectal balloon with ≥ 30 mL air; repeat with larger volumes if megarectum suspected

- also hard to detect when resting pressure is very low

ABSENT in Hirschsprung's, acquired myenteric neuropathy, and after rectal reconstruction

RECTAL SENSATION & COMPLIANCE

Balloon ≥ 3 -5 cm above the top of the canal. Ramp (1-2 cc/s) or phasic insufflation

- 3 thresholds: FIRST SENSATION, first urge to defecate, MAXIMUM TOLERATED VOLUME
- compliance from the derived pressure–volume curve

HYPERsensitivity: urge incontinence, radiation proctitis, UC, IBS-D, LARS

HYPOsensitivity: 18-66% of chronic constipation – predicts poor response to biofeedback AND to colectomy

Sensory thresholds improve with successful behavioral, drug, or surgical therapy (incl. neuromodulation)

BALLOON EXPULSION TEST

4 cm balloon, 50 mL warm water, pt sits on a commode and passes it PRIVATELY

Abnormal = cannot pass, or takes > 1 minute

Highly SPECIFIC for obstructed defecation, sensitivity only 50%. Does NOT define the mechanism

LONDON CLASSIFICATION (IAPWG 2014-2018)

Four parts: (a) RAIR disorders, (b) anal tone & contractility, (c) rectoanal coordination, (d) rectal sensation

- MAJOR findings, never seen in health: rectoanal areflexia, anal hypotension & hypocontractility, rectal hypo- and hypersensitivity
- MINOR / inconclusive, seen in health too: anal hypertension, dyssynergia

Aim is a common language, akin to the Chicago classification for esophageal motility

NEUROPHYSIOLOGIC TESTING

EMG

Maps EAS defects, striated muscle function, denervation-reinnervation potentials

NEEDLE EMG largely obsolete – endoanal US and MRI replaced it; painful, poorly tolerated, low yield

- normal MUP amplitude < 600 μ V, duration < 6 ms

SURFACE EMG easy and global; the anal plug intraluminal electrode is more accurate than pads (no gluteal artifact)

- recruitment tracks anal pressure on squeeze
- BEST USE: detecting paradoxical contraction or failure to relax with push, and biofeedback retraining

PNTML

Pudendal nerve stretch injury: 3rd trimester, 2nd stage of labor, forceps delivery

Bipolar disposable electrode stimulates the nerve; time to the EAS compound action potential = PNTML

Prolonged = pudendal neuropathy or loss of fast-firing fibers

LARGELY DISCREDITED – operator dependent, poor sensitivity & specificity, normal despite partial injury, variable normal limits, does NOT predict sphincteroplasty outcome

SPINAL MOTOR LATENCY & MEPS

Transcutaneous cauda equina stimulation over the lumbar spine – needles in puborectalis, EAS, urethral sphincter

- comparing latencies across levels localizes the LEVEL of injury
- cauda equina delay in up to 23% of idiopathic fecal incontinence

Translumbar & transsacral MAGNETIC stimulation → rectal and anal MEPS

- prolonged in 87% of fecal incontinence vs. prolonged PNTML in 63%, and BETTER TOLERATED
- clinical significance still unproven

ENDOANAL ULTRASOUND

GOLD STANDARD for sphincter anatomy in fecal incontinence – replaced EMG mapping for surgical planning

Also for fistulas (cryptoglandular, rectovaginal, Crohn's) – muscle involved, and post-repair assessment

EQUIPMENT & TECHNIQUE

3D endocavitary rotating transducer, 360°. BK 20R3 probe, 3-20 MHz

- 6 cm axial acquisition WITHOUT moving the probe, then reformat in any plane

Two enemas before. Awake, left lateral decubitus. Tip placed at the puborectalis

- H2O2 injection makes a fistula tract HYPERechoic – sensitivity to 90%

INTERPRETATION

Upper canal: hyperechoic horseshoe of the PUBORECTALIS

Mid canal: complete rings – IAS HYPOechoic, EAS HYPERechoic

Distal canal: IAS gone, EAS only

Perineal body: press the posterior vaginal wall with a finger and measure to the IAS

- normal nulliparous 17.4 ± 2.7 mm; < 10 mm a/w an anterior sphincter defect

Anal canal length in females ~ 3 cm

IAS thickness: nulliparous < 30 yrs 4.5 ± 0.7 mm vs. > 50 yrs 5.9 ± 1 mm – 33% thicker with age

EAS in nulliparous women: posterior 24.7 ± 4.6 mm, anterior 6.6 ± 1.7 mm

MRI

Static endoanal-coil MRI has FALLEN OUT OF FAVOR – expensive, no better than US for repairable anterior EAS defects

- MR DEFECOGRAPHY has taken its place

DEFECOGRAPHY

Fluoroscopy of defecation with thickened barium paste, in the PHYSIOLOGIC SITTING position

- oral contrast for enterocele; vaginal and bladder contrast to add compartments
- still and cine images. Mean dose 0.5-5.0 mSv

Indications: obstructed defecation, and prolapse (rectal prolapse, enterocele, cystocele)

Five criteria for a normal evacuation:

- $\uparrow$ anorectal angulation
- obliteration of the puborectalis impression
- wide opening of the anal canal
- TOTAL evacuation of rectal contents
- normal pelvic floor resistance

Anorectal angle: between the anal canal axis and the posterior rectal wall

- rest 90 - 110° , evacuation 110 - 180° (mean $92^\circ \rightarrow 137^\circ$, an increase of $\sim 45^\circ$)
- structures are not fixed $\rightarrow$ measurement is unreliable

Pubococcygeal line (pubis to tip of coccyx) defines the pelvic floor – prolapse graded by descent below it

Pelvic floor descent: normal < 3 cm; > 3.5 cm is abnormal $\rightarrow$ hypermobility, stretch of muscle and nerve

Rectocele: < 2 - 3 cm is normal, seen in up to 93% of asymptomatic women

- measure from the anterior anal canal border to the most anterior point of the rectocele
- MUST be a/w outlet symptoms to be pathologic; assess POST-evacuation for barium trapping
- digitalization of the posterior vaginal wall or perineum is the telling maneuver

Enterocele: small bowel into the pouch of Douglas – MC after hysterectomy. Sigmoidocele: sigmoid into the same space

- best seen on STRAINING with oral contrast; bowel should never descend below the upper rectum

Anismus: 90% of pts unable to evacuate 2/3 of a 120 cc enema in 30 sec had anismus confirmed physiologically

Oxford grading of intussusception:

- I & II: does NOT reach the anal canal
- III & IV: into the anal canal, not through the anus
- V: protrudes from the anus (overt prolapse)
- higher grades a/w increasing FECAL INCONTINENCE, not constipation

MR DEFECOGRAPHY

Vagina and rectum filled with ultrasound gel, SUPINE with knees bent, imaged through maneuvers

Comprehensive structural + functional assessment, NO radiation

Weakness: the supine position – pts cannot defecate normally, and gravity-dependent findings are missed

2016 study, $n=50$, MR vs. fluoroscopic defecography for posterior compartment (peritoneocele, rectocele, prolapse, anismus): NO statistical difference

- choose by local availability and expertise; use BOTH in difficult cases

DYNAMIC ULTRASOUND / ECHODEFECOGRAPHY

Cheap, safe, no radiation, no need to defecate contrast.

Also shows tape and mesh position

Three probes: transperineal, transvaginal, endoanal.

Dorsal lithotomy, hips flexed and abducted, enema first

- transperineal convex probe from mons pubis to anal margin, minimal pressure
- imaged at rest, maximal Valsalva and pelvic floor contraction, saved as a cine loop
- shows the whole puborectalis and its pubic rami attachment

Prospective multicenter trial, 86 women with obstructed defecation: echodefecography detects the SAME anorectal dysfunctions as defecography, minimally invasive and well tolerated

TRANSIT STUDIES

Colonic transit – radiopaque markers:

- normal total colonic transit ~ 35 hrs (right 11.3, left 11.3, rectosigmoid 12.4)
- one capsule of 24 markers on day 0, supine AXR on day 5
- ≤ 5 markers remaining = NORMAL
- diffuse scatter = colonic inertia. Pooled in the rectosigmoid = outlet obstruction

Radionuclide scintigraphy:

- gamma scintigraphy – 24-48 hrs of scanning instead of 5-7 days

Small bowel transit:

- lactulose breath hydrogen – lactulose ferments only in the colon
- time from ingestion to a 20 ppm rise in breath hydrogen

58 PELVIC FLOOR DISORDERS: EVALUATION OF

CONSTIPATION AND TREATMENT OF THE ABDOMINAL COMPONENT

DEFINITION & EPIDEMIOLOGY

Chronic constipation: 15-20% of North America

- F > M, ↑ with age, non-whites more often
- ~8 million physician visits/yr, ~\$19 million/yr in direct cost

Not just infrequency – straining, hard/lumpy stool, incomplete evacuation, anorectal blockage, digitation are equally or more common

Acute: < 1 week

Chronic: symptoms ≥ 3 months, onset ≥ 6 months prior

Mean transit times:

- males 31 hrs, females 39 hrs
- small bowel 90-120 min

SECONDARY CAUSES

Meds: opioids, anticholinergics, TCAs, CCB, diuretics, bile acid sequestrants, calcium, iron

Mechanical: CA, ischemic or diverticular stricture, endometriosis, IBD, Hirschsprung's, pseudo-obstruction

Metabolic: DM, hypothyroid, ↑Ca, ↓K, ↓Mg, uremia, heavy metals

Myopathy / neuropathy: amyloid, scleroderma, Parkinson's, MS, cord injury or tumor

Psych: depression, eating disorder, sexual abuse

Pelvic floor: fissure, anal stenosis, non-relaxing puborectalis, POP

ROME IV SUBTYPES

Need 3 months of symptoms, onset ≥ 6 months prior, and NO organic pathology

Functional Constipation (FC):

- does not meet IBS criteria – pain absent or < 1 day/wk
- ≥ 2 of the following:
 - straining > 25% of defecations
 - lumpy or hard stool (Bristol 1-2) > 25%
 - incomplete evacuation > 25%
 - anorectal obstruction / blockage > 25%
 - manual maneuvers (digitation, pelvic floor support) > 25%
- < 3 spontaneous BMs per week

IBS-C:

- ≥ 25% of stools Bristol 1-2 and < 25% Bristol 6-7
- abd pain ≥ 1 day/wk w/ 2 of: ↓ frequency, harder stool, related to defecation

Opioid-Induced (OIC):

- FC criteria, but new or worsening on starting / ↑ opioid

Functional Defecation Disorders:

- FC or IBS-C criteria PLUS 2 of 3 – abnormal balloon expulsion, abnormal evacuation pattern on manometry or EMG, impaired evacuation on defecography w/o structural lesion

EVALUATION

HISTORY

What the pt means by “constipated” – stool size,

frequency, consistency, ease & efficacy of evacuation

- onset, diet & exercise, meds incl. OTC and herbals, PSHx
- hx of sexual abuse, psych illness
- urinary or vaginal symptoms

Bristol Stool Chart. Patient diary

ALARM: sudden change in habit, weight loss, family hx of CA, bleeding w/o hemorrhoid or fissure

PHYSICAL

Abdomen: distention, bloating, mass

Prone, left lateral, AND on the commode – to exclude POP

DRE: mass, stricture, impaction, rectocele, canal length, resting & squeeze tone, ability to RELAX with straining

Anoscopy / procto: inflammation, solitary rectal ulcer

LABS & SCOPE

CBC. TSH and Ca only if clinically indicated

Colonoscopy for alarm symptoms or age-appropriate screening

WHICH TEST FIRST

Do NOT test everyone – only those who fail empiric therapy

- abdominal predominance (bloating, high-volume laxatives) → START with transit study
- outlet symptoms (straining, digitation) → START with manometry + defecography

TRANSIT & PELVIC FLOOR TESTING

Radiopaque Markers:

- simplest & cheapest. No standard protocol
- one capsule (24 markers), AXR day 5. Off laxatives, 30 g fiber daily
- > 5 markers (20%) retained = SLOW TRANSIT
- scattered throughout → colonic inertia; clustered at rectosigmoid → suggests dyssynergia, but CHALLENGED
- number retained does NOT correlate with symptom severity or QOL
- reproducibility poor – 31% had disparate findings on a repeat study

Scintigraphy:

- 24-48 hrs vs. 5-7 days for markers – better if pt can't stop laxatives that long

Wireless pH-Pressure Capsule (SmartPill):

- pH marks small bowel then colonic entry. Also measures colonic motor activity
- gives gastric emptying and small bowel transit as well
- > 80% agreement w/ scintigraphy for normal vs. slow

Colonic Manometry:

- catheter to the ileocecal junction. Invasive, not widely available
- standard in peds. Distinguishes colonic NEUROPATHY from MYOPATHY

Anorectal Manometry:

- pressures, RAIR, rectal sensation & compliance
- absent RAIR → Hirschsprung's
- pair w/ balloon expulsion to detect dyssynergia

Defecography:

- barium / cine – physiologic seated position, but not standardized and the anorectal angle is poorly reproducible
- MR – supine, but better for POP and bony landmarks

MEDICAL MANAGEMENT

Educate, eliminate secondary causes, clean up the med list

Behavioral first: fiber, fluid, exercise, adequate time on the toilet

Fiber:

- 20-30 g/day
- insoluble = bulk → direct stimulation of secretion & motility; soluble = hydrophilic + osmotic ferment
- bloating, distention, flatulence limit compliance
- slow transit and pelvic floor dysfunction are LESS likely to respond – fiber is not for everyone
- extra fluid alone: no difference in stool output.
Sedentary pts 3x more likely constipated, but 9-RCT meta-analysis could not confirm an exercise effect

Softeners:

- docusate 100 mg BID LESS effective than psyllium (RCT, n = 170)

Osmotic:

- PEG, mag citrate, sodium phosphate, lactulose / sorbitol
- Cochrane: PEG SUPERIOR to lactulose for frequency, consistency, and pain
- lactulose & sorbitol equal effect, lactulose more nausea
- these yield H₂ and CO₂ – caution explosion risk

Stimulants:

- bisacodyl, sodium picosulfate, senna, cascara, aloe
- ↓ water absorption, ↑ motility & prostaglandin release
- melanosis coli = lipofuscin in mucosal macrophages – REVERSIBLE, functionally insignificant
- NO evidence stimulants permanently damage the colon

Secretagogues:

- lubiprostone – Cl channel activator. Nausea the commonest SE
- linaclotide & plecanatide – guanylate cyclase-C agonists, equal efficacy & tolerability
 - diarrhea forces discontinuation in < 5%
- tenapanor – NHE-3 inhibitor, FDA 2019, superior to placebo in phase 2 & 3

Prokinetics (5-HT₄):

- tegaserod – pulled 2007 for ischemic CV events. Back in 2019 for women < 65 w/ no CV disease and ≤ 1 risk factor
- prucalopride – more selective. 6 RCTs, n = 2484: superior to placebo for ≥ 3 SBM/wk. Also works in OIC

- diarrhea & headache, gone within the first week

Opioid-Induced:

- 51-87% of pts on opioids for CA, 41-57% for chronic non-CA pain
- early recognition. Standard laxatives first
- PAMORAs 2nd line – naloxegol, naldemedine, alvimopan, SQ methylnaltrexone
- do NOT cross the BBB – central analgesia preserved, no withdrawal
- meta-analysis 27 RCTs: naldemedine & naloxone most efficacious, naloxone SAFEST

Elobixibat: ileal bile acid transporter inhibitor – approved in Japan only

SURGERY FOR SLOW TRANSIT

PATIENT SELECTION

No standard definition of “medically refractory” – no consensus on drug, dose, or duration

The transit study identifies NORMAL transit. It is NOT a marker of who will succeed after colectomy

- two abnormal studies → significantly more likely to succeed

Exclude concomitant pelvic floor dysfunction

- outlet obstruction on EMG or defecography → much LESS likely to be satisfied
- rectal hyposensitivity on manometry → less satisfied
- but an abnormal test alone does not preclude surgery (Reshef, n = 41)

Rule out gastric & small bowel dysmotility – delayed gastric emptying does WORSE

- small bowel transit findings alone did not change success (Zmora, n = 17)

Psych: ↑ anxiety & depression vs. matched controls

- sexual abuse hx does not guarantee failure, but predicts post-op abdominal complaints
- CBT before surgery

Predominant PAIN & BLOATING → proceed cautiously, but not abandoned

TOTAL ABDOMINAL COLECTOMY WITH IRA

PROCEDURE OF CHOICE – 2016 ASCRS guideline (strong rec, low-quality evidence)

Rationale: ↓ colonic transit → liquid content delivered to the rectum → easier evacuation, ↑ frequency

Outcomes:

- Zutshi, median 11 yrs: 77% still pleased. SBO 20%, 90% done open
- Patton, median 16 yrs: SBO requiring admission 21%
 - 41% have 1-3 BM/day, 18% have 7-10/day, 7% > 10/day
- moderate incontinence 29%, SEVERE 21% (St. Mark's score)
- 41% w/ ongoing abdominal pain, yet 81% “at least somewhat pleased” with frequency

Variants:

- antiperistaltic cecorectal (saves the ileocecal valve), cecorectal w/ bypass, J-inling (side-to-side ascending-to-rectum)
- Xie: antiperistaltic cecorectal (n = 20) vs. standard IRA (n = 35) – NO difference in any score

SEGMENTAL RESECTION

Small series, based on regional transit

Hampered by inaccurate isolation of the “diseased” segment, small data sets, and reported failures

NOT recommended

MEGARECTUM / RECTAL INERTIA

Megarectum = dilation > 6.5 cm at the pelvic brim on contrast enema

Colectomy w/ IRA does WORSE – recurrent constipation

IPAA: small series, QOL improved, but 13% pouch excision in a series of 15

- confirm the anus and pelvic floor RELAX before offering a pouch
- ASCRS: available studies do NOT justify IPAA – only after all else exhausted

LESS INVASIVE OPTIONS

Transanal Irrigation (TAI):

- 500-1000 mL ~3x/week
- 102 idiopathic constipation: 63% moderately or much improved at 12 mo, 53% still irrigating
- other series only 34-45% in chronic constipation. Discontinuation high

Antegrade Continence Enema (ACE):

- appendicostomy or other conduit. Avoids a stoma
- 2016 systematic review, 209 pts: 68% success at median 39 mo – but success is rarely defined beyond ongoing use
- Dutch cohort: only 37% successful, long-term QOL no different from controls
- revision for stenosis & leakage HIGH – reoperation approaches 90% even at specialized centers
- ASCRS: highly selected, motivated pts ONLY

Sacral Neuromodulation:

- S3 / S4 stimulation ↑ distal colonic transit & contractility
- NOT FDA approved for constipation
- longer f/u disappointing – success 5-30%
- no predictors of response identified, likely because all constipation subtypes are lumped together

Ileostomy:

- for significant incontinence, high operative risk, or refusal of colectomy
- also salvage after diarrhea / incontinence complicating a colectomy
- St. Mark's, n = 24: 64% satisfied despite below-average HRQOL, 20% reoperation
- elsewhere 29% reoperation for retraction and hernia

Abdominal symptoms → transit study first. Outlet

symptoms → manometry + defecography first

Slow transit + normal pelvic floor + no upper gut

dysmotility → TAC with IRA

Outlet obstruction, or predominant pain & bloating → fix that first and counsel hard

Segmental resection NOT justified. IPAA is a last resort

BOTTOM LINE

Test only the medically refractory

59 PELVIC FLOOR DISORDERS: TREATMENT OF DIFFICULT AND OBSTRUCTIVE DEFECACTION

OBSTRUCTED DEFECACTION SYNDROME

Straining; incomplete, unsuccessful, or painful evacuation;
infrequency; bloating; need for digitation

Constipation divides into colonic inertia (abdominal),
PELVIC (ODS), and normal-transit / IBS

- Rome IV functional constipation criteria apply – see ch 58

Two mechanisms, and they coexist:

- FUNCTIONAL – dyssynergic defecation
- STRUCTURAL – rectocele, enterocele, sigmoidocele, internal intussusception, overt prolapse

EVALUATION

Number of evacuations, consistency, mucus or blood,
pain, incomplete evacuation, need for digitation

- dyssynergic pts have MORE trouble with SOFT stool

Laxative and diet habits. LLQ pain → think IBS, CA, celiac,
diverticular

Inspection: rectal tumor, anal stenosis, internal prolapse,
thrombosed hemorrhoids, fissure

Proctoscopy: melanosis coli (chronic laxative abuse),
solitary rectal ulcer, prolapse, descending perineum,
rectocele

Impaction in the elderly or a child → investigate further
DRE is the BEST test for dyssynergia – non-relaxation or
paradoxical puborectalis contraction on Valsalva

TESTING

Anorectal manometry / HRAM:

- resting pressure = IAS, squeeze = EAS
- RAIR, rectal sensation, compliance. Loss of RAIR → Hirschsprung's
- 4 types of dyssynergia, by push force and sphincter response

Cinedefecography:

- rectocele, enterocele, internal prolapse, perineal descent, anorectal angle

Echodefecography:

- sphincter defect, IAS hypertrophy, structural abnormality

Radiopaque marker transit:

- diffuse spread = slow transit; markers collected in the pelvis = ODS

MR defecography:

- separates a structural substrate from a purely functional disorder

EMG:

- motor unit potentials, denervation / reinnervation, anismus

PNTML:

- prognostic only, when surgery is planned

Scores: CSI (78 items), ODS score (prospectively validated), CRQOL, PFDI + CRADI for combined compartments

Testing is COMPLEMENTARY – mild radiologic findings without symptoms do not predict a good operation

DYSSYNERGIC DEFECACTION

Paradoxical contraction or inadequate relaxation of the pelvic floor, ± inadequate propulsion

Up to 40% of all constipated pts. Incidence 4.6% in pts without IBS

Aka anismus, paradoxical puborectalis contraction

CLINICAL diagnosis. Confirm w/ manometry ± cinedefecography

TREATMENT

Tell the pt this contraction can be UNLEARNED. Treat coexisting constipation at the same time

Pelvic Floor PT / Biofeedback:

- FIRST LINE
- sensory biofeedback, electrical stim, visual manometry, simulated defecation
- diaphragmatic breathing and RELAXATION
- ask what they actually did – often given KEGELS, the exact opposite of the intended treatment
- multiple visits before calling it a failure. Home-based program if no trained therapist

Electrogalvanic Stimulation (EGS):

- transanal probe, galvanic current at increasing amplitude to FATIGUE the muscle
- 3x/wk first week, 2x the second, then weekly
- NOT better than biofeedback in most studies – method is not standardized. Few centers offer it
- SE: pain, rectal irritation, bleeding

Botulinum Toxin A:

- for refractory symptoms. No consistent dose or technique across centers
- 200 units in 6-7 cc saline, loaded into six tuberculin syringes
- under anesthesia, from the perineum w/ a finger in the rectum, into the levator – posterior, posterolateral, lateral, anterolateral
- include the IAS only if tight – warn about temporary incontinence
- continue PT while it is working. Repeat at 3-month intervals if it worked

Puborectalis Myomectomy:

- described, never popularized

Failed everything → DIVERSION

Slow transit + dyssynergia → colectomy (± ileostomy) first, then treat the dyssynergia

RECTOCELE

Herniation of the rectal wall into the vagina

R.F.: age, obesity, obstetric injury, multiple vaginal deliveries

Size on defecography:

- small < 2 cm, moderate 2-4 cm, large > 4 cm

OPERATE only if symptomatic – vaginal or perineal support needed to defecate, and the rectocele does NOT empty, or empties only with digitation
Address the urologic / gynecologic problems at the same operation

TRANSANAL

Incise the anterior rectal wall, excise the mucosa over the rectocele, plicate the rectovaginal septum, close the mucosal defect

30-90% symptom improvement, but recurrence by ~48 months

Complications: dyspareunia, fecal incontinence, rectovaginal fistula

STARR

Full-thickness excision of the rectal wall w/ two PPH01 staplers – one anterior, one posterior

Do NOT draw the vaginal wall into the purse string or the staple line

Good short-term results, but ~40% recurrence of symptoms long term

Complications: bleeding, anastomotic leak, rectovaginal fistula, rectal pain

Declining in Europe, few US centers still offer it

TRANSPERINEAL

Advantage: does NOT breach the vaginal mucosa

Perineal incision, dissect to the vaginal apex, simple fascial repair apex to perineum

May be augmented w/ mesh – controversial

TRANSVAGINAL

Vaginal incision, separate mucosa from fascia, fascial stitches close the defect, trim excess mucosa

Recurrence ~7.1%. Complications: dyspareunia, bleeding, wound infection

Meta-analysis: NATIVE TISSUE transvaginal repair PREFERRED over the other repairs

Technique, graft-augmented:

- full prep. Prep vagina and anus widely. Lithotomy, foley, local block
- double-glove one hand, palpate the defect from the rectum – new outer glove every time the hand comes out
- mark the perimeter of the defect transvaginally with the bovie
- incise cephalad to the sphincters, find the rectovaginal septum plane. Allis clamps to elevate and show the plane
- excise ~1 cm strip of vaginal mucosa, dissect toward the cervix. STAY OFF the rectal serosa
- expose fascia over the levator plate, raise 1-2 cm flaps circumferentially
- graft tacked by 2 cephalad sutures below the cervix, then interrupted vicryls around – ≥ 1 cm overlap, ALL to levator fascia
- close vaginal mucosa, full-thickness interrupted 2-0 vicryl

- pack w/ clindamycin cream, NOT tight. Packing and foley out the next morning. No baths x 6 weeks
- KEY: stay in the right plane, and get complete hemostasis – a hematoma breaks the wound down

ENTEROCELE & SIGMOIDOCELE

Pelvic floor herniation of bowel into the pouch of Douglas

Enterocoele = small bowel between rectum and vagina, reaching below the upper third of the vagina on straining

Degree:

- 1st: above the pubococcygeal line
- 2nd: below pubococcygeal, above ischiococcygeal
- 3rd: below the ischiococcygeal line

Size – PCL to the most inferior point of the sac:

- small < 3 cm, moderate 3-6 cm, large > 6 cm

Same hernia w/ different contents = peritoneocoele, omentocoele, sigmoidocoele

Do NOT operate on radiology alone – need a demonstrated OUTLET OBSTRUCTION plus a substantial hernia

PREFERRED repair: sacrocolpopexy w/ monofilament nylon mesh, $\pm$ ventral rectopexy. Plan it with urogynecology

INTERNAL PROLAPSE / INTUSSUSCEPTION

Funnel-shaped infolding of the rectal wall on defecation that does NOT protrude from the anus

Suspect clinically, demonstrate on cinedefecography – MUST correlate with the symptoms

Asymptomatic intussusception is NOT an indication

Ventral Rectopexy:

- the current operation for symptomatic internal intussusception – technique in ch 60
- nonabsorbable mesh complications published; absorbable / biodegradable mesh gives similar outcomes

Delorme:

- an option for short-segment internal prolapse

STARR:

- popularized a decade ago, now largely abandoned in the USA
- benefit likely OVERESTIMATED – heterogeneous patient-reported outcome measures
- variants described, incl. Transtar

Overt full-thickness prolapse → ch 60

SOLITARY RECTAL ULCER SYNDROME

Disorder of defecation, usually a/w rectal prolapse or internal intussusception

Sx: bleeding (occasionally transfusion-requiring), difficult defecation, tenesmus, mucus discharge, anal or pelvic pain

Mucosa traumatized as the rectum invaginates → transient ischemia

Thickened mucosa on the ANTERIOR rectal wall

- ulcers in only 23%, polyps or masses in 74%
- when present: single or multiple, shallow, hyperemic margins, pale base
- misdiagnosed as a polyp or a CA

Biopsy: fibrotic obliteration of the lamina propria, thickened muscularis mucosae

Colitis Cystica Profunda: mucous cysts lined by columnar epithelium DEEP to the muscularis mucosae – same symptoms, same gross appearance

EUS: thickened IAS is the typical finding

Treatment:

- FIRST normalize defecation – diet, bowel retraining, pelvic floor PT
- argon plasma coagulation for bleeding
- deep intussusception (Oxford 3-4) from WEAKNESS w/ multicompartiment POP → rectopexy
- SRUS from STRAINING and dyssynergia → do NOT operate. Pelvic floor PT

THE MESH QUESTION

Synthetic mesh in the rectovaginal septum remains controversial

Native tissue transvaginal repair PREFERRED over mesh augmentation on meta-analysis

Nonabsorbable mesh complications drove the shift to biologic / absorbable for ventral rectopexy – similar outcomes

Erosion after lap ventral mesh rectopexy 2% overall

- synthetic 2.4% vs. biologic 0.7%
- polyester 6.5% vs. polypropylene 1.6%
- nonabsorbable (Ethibond) suture 4% erosion vs. 0% with PDS

BOTTOM LINE

Dyssynergia is a CLINICAL diagnosis on DRE. PT first, then Botox or EGS. Never operate on it

Rectocele: only if symptomatic, non-emptying, and digitation-dependent. Native-tissue transvaginal is the default

Enterocoele / sigmoidocoele: sacrocolpopexy, with urogynecology

Internal intussusception: ventral rectopexy has replaced STARR

Radiology without correlating symptoms is NOT an indication to operate

60 PELVIC FLOOR DISORDERS: RECTAL PROLAPSE

OVERVIEW

Full-thickness protrusion of the concentric rings of the rectum through the anal canal

Parous women, 7th-8th decade. Men affected younger and 2/2 other dz

> 100 described operations, NONE is a gold standard

Mechanism poorly understood

- Broden & Snellman: begins as a mid-rectal intussusception that progresses down through the anal canal
- connective tissue disorders, esp. in the young
- straining, solitary rectal ulcer, slow transit, malabsorptive diarrhea
- multicompartiment POP – global pelvic tissue weakness

Mucosal prolapse: mucosa only – RADIAL grooves

Full-thickness: CONCENTRIC rings

Internal intussusception: full rectal wall telescopes but does NOT protrude

MULTICOMPARTMENT DISEASE

A defect in one compartment usually carries symptoms or defects in another

- ↑ with age, ↑ BMI, prior hysterectomy

20-35% of prolapse pts report urinary incontinence

15-30% have significant vaginal vault prolapse

POP → earlier and higher recurrence after rectopexy ALONE

Repairing ONE compartment often worsens the others → multidisciplinary plan, avoid sequential operations

OXFORD GRADING (ON DEFECOGRAPHY)

- I: descends no lower than the proximal limit of the rectocele
- II: into the level of the rectocele, but not into the anal canal
- III: into the PROXIMAL anal canal
- IV: into the DISTAL anal canal
- V: protrudes from the anus – overt prolapse

I & II low grade, III & IV high grade

EVALUATION

Sx: rectal / pelvic pressure, irregular habits, incomplete evacuation, mucous seepage, blood on the paper, urgency, outlet obstruction

Prior prolapse or POP repair changes the plan – ask

Ask about urinary incontinence & hesitancy, vaginal pressure, dyspareunia, vaginal bulging

DRE: resting tone, squeeze strength, pelvic floor coordination on squeeze and bear-down

Have the pt bear down SITTING upright on a commode, with a mirror

Colonoscopy for the change in bowel habit

Manometry:

- repeated stretch of the sphincter complex → low resting pressure and diminished squeeze recruitment

- use it to counsel expected post-op continence and the need for PT

EMG:

- unreliable in POP – prolapsing tissue itself causes a paradoxical ↑ that MIMICS dyssynergia

Defecography:

- fluoroscopic, seated, is the most helpful test – exam cannot show internal collapse
- also shows rectocele, internal intussusception, enterocele, sigmoidocele, uterovaginal prolapse, cystocele
- dynamic MR defecography is SUPINE and LESS sensitive – misses intussusception, prolapse, small rectoceles, vault descent

IMPACT (Pelvic Floor Disorders Consortium, 2019) – the combined validated questionnaire

TREATMENT

Nearly always surgical. Bowel regimen and pelvic floor PT optimize but do NOT resolve prolapse

Choice driven by general health, symptoms, concomitant POP, prior surgery, urgency

- INCARCERATED → perineal, mandatory
- perineal carries higher recurrence – reserve it for very poor surgical candidates

The optimal operation remains unclear

PROSPER (Senapati, n = 293): abdominal vs. perineal, suture vs. resection rectopexy, Delorme vs. Altemeier – NO significant difference in recurrence in any comparison, and recurrence high across the board

PERINEAL REPAIRS

Light anesthesia, spinal, or local, no paralysis. No abdominal incision – early ambulation

DELORME – MUCOSAL SLEEVE RESECTION

Best for a SHORT segment, or prolapse limited to part of the circumference

Morbidity and mortality very low, but recurrence 16-30%

Technique:

- general, spinal, or MAC. Extend the prolapse with Babcocks, or a dry sponge and traction
- Lone Star retractor
- 1:400,000 adrenaline submucosally to elevate ALL the exposed mucosa – opens the plane, ↓ bleeding
- circumferential incision 10-15 mm from the dentate line, MUCOSA ONLY – bovie
- strip the mucosal sleeve off the muscularis to ≥ 2.5 cm beyond the apex of the prolapse
 - in the right plane there is no bleeding – penetrating vessels tied or coagulated
- 8-12 plicating absorbable sutures b/n the muscular wall and the cut edges
 - in and out along the longitudinal axis – accordion / concertina effect
 - SAME number of bites (3-4) in every suture

- tie – the muscle forms a doughnut
- divide the mucosal sleeve, sew the cut ends together, 3-0 chromic

ALTEMEIER – PERINEAL RECTOSIGMOIDECTOMY

Prone jackknife preferred. Inject the rectal wall for hemostasis

Full-thickness circumferential incision 2 cm above the dentate line

Divide the mesorectum at the point of maximal tension, working circumferentially, pulling redundancy out

Anteriorly the peritoneal reflection / hernia sac is entered

Divide when no redundancy remains – MUST have mesentery and NO tension

Hand-sewn coloanal, or low stapled colorectal, anastomosis

LEVATORPLASTY anteriorly if incontinent – improves post-op continence

Risks: anastomotic bleeding, pelvic abscess, leak.

Recurrence up to 20%, and 50% in registry data

CANNOT be done after a prior sigmoid resection – vascular disruption to the coloanal anastomosis

ANAL ENCIRCLEMENT – THIERSCH

Largely abandoned – high failure and complication rates

Permanent suture, wire, biologic or synthetic mesh in the subcutaneous space to narrow the canal

Does not fix the prolapse, only traps it above. Recurrence 33-44%

Erosion of the device, severe outlet dysfunction

Only for extreme comorbidity, or prolapse w/ a permanent colostomy

INCARCERATED PROLAPSE

Reduce manually if pink and well perfused – granulated SUGAR ↓ the edema

Irreducible or gangrenous → ALTEMEIER

COMBINING

Colpocleisis can be added to a Delorme or Altemeier for vaginal prolapse

A prior abdominal prolapse repair does NOT preclude a perineal operation

ABDOMINAL REPAIRS

Lower recurrence, better function, low morbidity vs. perineal

Age alone is NOT a contraindication

- NSQIP (Fang): in ASA 3-4, mortality RR 4x HIGHER with perineal than abdominal. Laparoscopic mortality 0%
- Danish registry, 41% octogenarians – overall 30-day mortality 2.1%
 - open rectopexy 3.3%, lap rectopexy 1.4%, Altemeier 4.4%, Delorme 2.3%

SUTURE RECTOPEXY

Cutait, 1959. Mobilize anterior and posterior to the levators, PRESERVE the hypogastric nerves

Pull cephalad, suture the mesorectum to the anterior longitudinal ligament at the promontory, non-absorbable

Recurrence up to 20%

Continence improves in 34-42%. NEW incontinence 7%

NEW constipation up to 50% – denervation via the lateral ligaments

- preserve one lateral stalk and fix unilaterally to mitigate it

Hidaka, 6-yr double-blind RCT, lap suture vs. lap ventral mesh rectopexy: recurrence 23.3% vs. 8.2%. ODS, incontinence, constipation and QOL ALL favored ventral mesh

RESECTION RECTOPEXY

Frykman & Goldberg, 1969. Adding sigmoid resection ↓ post-op constipation, esp. if constipated pre-op

Feared leak did NOT materialize – 0-2.3%

Laubert: incontinence improved in 64%, constipation in 77%

Recurrence 10-13% – LOWER than rectopexy alone

Equal to lap ventral rectopexy for constipation and ODS

POSTERIOR MESH RECTOPEXY – WELLS

1959, Ivalon polyvinyl alcohol sponge. Pelvic sepsis up to 16% forced the change to mesh

Mesh wraps the rectum leaving the ANTERIOR wall open, fixed to the lateral stalks and the promontory

Constipation significantly WORSE post-op

Recurrence 6% full-thickness, 12% mucosal – rises with longer f/u

Pelvic sepsis morbidity still high even with mesh

RIPSTEIN – ANTERIOR SLING

1952. Sling from the anterior rectum to the anterior longitudinal ligament

Recurrence up to 13%, 3x higher in men (24%)

Continence improves, constipation does NOT. De novo constipation 14%, impaction needing admission 5%

Complication rate 52%. Ripstein himself left a gap anteriorly to ↓ obstruction

Performed much less often now

VENTRAL MESH RECTOPEXY (VMR)

D'Hoore. Now considered the GOLD STANDARD for rectal prolapse in Europe

PURELY ANTERIOR dissection – avoids the autonomic injury of a posterior dissection

Corrects the middle and anterior compartments too – same plane as sacrocolpopexy, so it teams easily with urogyn

Technique:

- expose the sacral promontory, mobilize anteriorly, open the rectovaginal septum ALL THE WAY to the pelvic floor
- mesh, biologic or synthetic, cut to size
- 8-12 stitches to the distal and mid ANTERIOR rectum, partial thickness

- add stitches to levator ani and pubococcygeus if the floor is lax
- then to the anterior longitudinal ligament at the promontory
- D'Hoore: stitch mesh to the posterior vaginal wall first, obliterating the rectovaginal septum – fixes enterocele / sigmoidocele
- re-peritonealize OVER the mesh
- uterus absent, or supracervical hysterectomy at the same setting → sacrocolpopexy

Outcomes:

- recurrence 0-15%. POP recurrence 8.5%
- fecal incontinence improves in 60-80.2%, constipation in 58-85%, ODS in 78.6%
- NEW or worse incontinence 1-3.9%. New or worse constipation 1.4-12.9%
- stress urinary incontinence improves in 53%, gyn prolapse symptoms up to 93.75%

Mesh & fixation complications:

- erosion 2% of 2203 – synthetic 2.4% vs. biologic 0.7%
- polyester 6.5% vs. polypropylene 1.6% – USE POLYPROPYLENE
- nonabsorbable (Ethibond) suture 4% erosion vs. 0% with PDS
- DISCITIS 0.1-2% – a risk of ANY sacral fixation. Back pain radiating to the legs → image and treat aggressively

Learning curve:

- proficiency for clinical and QOL outcomes at 82-105 cases, for operative time at 54
- poor technique loses the functional benefit and raises complications

Robotic:

- ideally suited – deep pelvis, suturing the distal rectum and the pelvic floor
- meta-analysis vs. lap: same recurrence, conversion, reoperation
- longer operative time, but LESS blood loss, fewer complications, shorter stay

RECURRENT PROLAPSE

No algorithm – evidence insufficient

Prior prolapse surgery → recurrence after VMR 25% at 5 yrs, vs. 9.7% when VMR was the primary repair

- highest when the index operation was a DELORME

Recurrence treated PERINEALLY → re-recurrence up to 50%

- manage a recurrence ABDOMINALLY whenever you can

Warn the pt of the much higher re-recurrence risk regardless of approach

Vascular traps:

- resection rectopexy after a prior Altemeier → devascularized rectal segment b/n the two anastomoses – mucosal sloughing, stricture

- Altemeier after a prior resection rectopexy → AVOID, unless the old anastomosis comes out with the specimen

After VMR:

- early recurrence → suspect DETACHMENT from the promontory. Reattach
- may need a more extensive, even posterior, dissection

INCONTINENCE VS. CONSTIPATION

Incontinent → abdominal rectopexy, or Altemeier + levatorplasty

Constipated → RESECTION rectopexy, or ventral rectopexy

Suture rectopexy alone creates new constipation in up to 50% – do NOT use it in a constipated pt

VMR improves BOTH, which is why it displaced the posterior operations

Best functional results and lowest recurrence: resection rectopexy and ventral mesh rectopexy

Fix ALL compartments – correcting one alone often worsens the others

61 PELVIC FLOOR DISORDERS: FECAL INCONTINENCE

— EVALUATION AND TREATMENT

DEFINITIONS & EPIDEMIOLOGY

Fecal Incontinence: uncontrolled passage of feces or gas (anal incontinence) ≥ 1 mo, in a pt who previously had control

Soilage: continuous or intermittent liquid anal discharge

Pseudoincontinence: prolapse, incomplete evac, poor hygiene, fistula, STD – NOT true FI

Encopresis: involuntary loss of stool w/ functional constipation as a child

Urgency: must go immediately or leak – impaired rectal compliance

Prevalence 1-19% – varies by definition, method, population

- a/w depression, social stigma, nursing home placement

Continence = stool consistency + sphincter fxn + rectal compliance + neuro fxn + pelvic floor coordination

- FI is usually MULTIFACTORIAL – disruption of ≥ 1 component

ETIOLOGY

Anatomic:

- sphincter trauma – obstetric, accidental, anorectal surgery
- rectal prolapse / intussusception, imperforate anus

Neurologic:

- pudendal neuropathy – stretch, DM, XRT, chemo
- CNS – stroke, dementia, cord injury, trauma, tumor, MS, cauda equina
- irreversible nerve injury w/ as little as 12% stretch

Stool Characteristics:

- diarrhea – infectious, IBS, IBD, malabsorption, laxative abuse, radiation enteritis
- constipation w/ overflow

Rectum:

- radiation proctitis, LAR / coloanal anastomosis, ↓ compliance

Obstetric Injury:

- clinical injury 0.6-9%
- OCCULT defects on ERUS in > 30% of primiparous, > 40% of multiparous
- risk: forceps (highest), mediolateral episiotomy, primiparity
- 60% w/ concomitant pudendal injury – compression + traction

Iatrogenic:

- Park's retractor MC – IAS damage from excessive dilatation
- sphincter division, rectal resection, neoadjuvant XRT

Radiation:

- 75% acute phase reaction, 20% late radiation proctitis

SEVERITY SCORING & QOL

CCF-FIS (Wexner):

- 5 items – solid, liquid, gas, wears pad, lifestyle alteration
- each 0-4: never 0 / rarely < 1 mo / sometimes < 1 wk ≥ 1 mo / usually < 1 day ≥ 1 wk / always ≥ 1 day
- 0 = perfect continence, 20 = complete incontinence
- validated, simple – the workhorse

Others:

- St. Mark's (Vaizey) – adds urgency & antidiarrheal use
 - FIS – ASCRS severity index, type-weighted
 - FIQOL – lifestyle, coping, depression, embarrassment
- CCF-FIS ≥ 10 predicts FAILURE of biofeedback

EVALUATION

History:

- OB hx, prior anorectal / colorectal sx, episiotomy, BM frequency, stool form, recent change
- meds, DM, neuro dz, IBS, IBD, XRT, prolapse
- concomitant urinary sx or anterior compartment prolapse
- *Active (urge): tries to hold & can't – intact sensation, EAS dysfxn*
- *Passive (unaware): IAS dysfxn or neurologic*

Physical:

- inspect – scars, deformity, fistula, excoriation, patulous anus, prolapsing hemorrhoids
- bear down for prolapse & perineal descent
- puborectalis sling palpated posteriorly – anorectal angle normally ~90°
- perineal body thickness, rectocele
- anal wink = intact sacral reflex arc
- DRE: resting tone, squeeze increase, is the gluteus cheating?

Anorectal Manometry:

- may not correlate w/ exam or predict response – still guides therapy
- *Resting pressure (40-70 mmHg): IAS = 55-60% of tone. Low → passive leakage*
- *Squeeze pressure (2-3x resting): EAS. Low → urge incontinence*
- *HPZ: 2-3 cm women, 2.5-3.5 cm men*
- *RAIR (10-30 mL): absent in Hirschsprung's, post anorectal sx, connective tissue dz. Hard to read if resting pressure low*
- *Rectal sensation (~40 mL): hyposensitive → constipation w/ incontinence; hypersensitive → urgency, IBS, LARS, XRT*
- *Compliance: ↓ in IBD, post-XRT, scleroderma*
- pressures may be NORMAL or ↑ w/ anismus or incomplete evac – esp. men

Ultrasound:

- objective sphincter integrity – cheap, in office, BEST imaging of the IAS
- MRI better for the anterior EAS and for concomitant prolapse

- full pelvic floor US (transperineal + transvaginal) images all compartments

Defecography (fluoro or MR):

- confirms inability to retain, identifies impaired evac & pelvic organ prolapse
- rectal intussusception = NEGATIVE predictor for SNM
→ consider ventral rectopexy

Neurophysiology:

- PNTML (2.0 ± 0.2 ms): prolonged latency does NOT independently predict treatment success – has faded away
- EMG sphincter mapping: needle, painful, now rarely used

Colonoscopy:

- per screening guidelines, and for any bowel sx
- diarrhea in an older woman → scope WITH biopsies to r/o microscopic colitis

CONSERVATIVE TREATMENT

Goal = ↓ frequency & severity, improve QOL – complete resolution is rare w/ ANY modality

Diet & Bulking:

- avoid sweeteners, dairy, caffeine; review aggravating meds
- psyllium / calcium polycarbophil – 50% reduction in FI episodes
- goal 1-2 well-formed stools per day

Antidiarrheals:

- loperamide – Mu receptor, ↑ resting pressure, improves rectal sensation
- diphenoxylate-atropine, codeine, amitriptyline
- cholestyramine for bile salt diarrhea

Bowel Management:

- enemas or suppositories to keep the rectum empty
- transanal irrigation (Peristeen) – up to 1.5 L a few times/week, best data in LARS

Biofeedback / Pelvic Floor Exercise:

- FIRST LINE. Needs rectal sensation, ability to contract, and MOTIVATION
- ≥ 6 sessions. Improves sensory threshold, coordinates EAS contraction to rectal distention
- Regadas et al: 50% reduction in FI score in ~50% of pts
- predicts FAILURE: CCF-FIS ≥ 10, prior vaginal delivery, prior anorectal / colorectal sx, can't sustain a squeeze
- Cochrane: biofeedback + e-stim better than either alone

Insertion Devices:

- anal plug (Renew) – hard to tolerate; 77% of completers ≥ 50% reduction over 12 wks
- vaginal bowel control system (Eclipse) – balloon occludes rectum from the vagina, deflate to defecate
 - only 62% could be fitted; of those fitted, 94% success at 12 mo

MINIMALLY INVASIVE TREATMENT

Sacral Neuromodulation (SNM):

- most common MIS option, for moderate-severe FI
- works w/ OR w/o a sphincter defect – SAME results in both groups
- mechanism unclear – somatovisceral reflex, direct sphincter effect, afferent neuromodulation
- Stage 1: lead at S3, 1-2 wk trial w/ diary. ≥ 50% ↓ in episodes → stage 2
 - motor: plantar flexion of great toe, levator bellows.
 - Sensory: vaginal or rectal pulling
 - office PNE = slightly higher failure; quadripolar lead in OR = slightly higher infection
- US pivotal trial: 83% w/ ≥ 50% reduction at 1 yr, 41% fully continent
- 5 yr: 89% w/ ≥ 50% reduction, 36% fully continent
- European group at 7 yr: 71% sustained in retained devices, ~50% on intention-to-treat
- reoperation up to 42%; generator change q 3-5 yrs
- rechargeable device – up to 15 yrs, full-body MRI conditional
- infection usually MUST be explanted; everything else reprogrammed or managed medically

Injectable Bulking Agents:

- NASHA Dx (hyaluronic acid / dextranomer), FDA approved 2011
- inject submucosa ABOVE the dentate line
- RCT vs sham: 52% vs 31% success; sustained 52% at 36 mo
- no better than biofeedback head-to-head
- best use: mild-moderate FI, men w/ soiling, residual FI after SNM, keyhole deformity

Percutaneous Tibial Nerve Stimulation (PTNS):

- shares sacral segmental innervation. Early series 63-82%
- UK RCT (227 pts): NO difference vs sham at 12 wks
- SNM superior to PTNS on function & QOL

Radiofrequency (SECCA):

- energy into the IAS → collagen deposition & thickening
→ ↑ outlet resistance
- mild-moderate FI w/ intact or limited defect. 4 quadrants, 4 levels
- 55-80% responders at 12 mo, durability poor – 6-22% at 3 yrs
- ASCRS guideline: may be used, but pursue alternatives FIRST

Stem Cell & Anal Sling:

- myoblast injection – CCF-FIS ↓ 13.7 pts at 12 mo, sustained at 5 yrs. NOT FDA approved
- TOPAS transobturator posterior sling – 70% w/ ≥ 50% reduction, 19% fully continent at 1 yr. Not available in US

OPERATIVE TREATMENT

Overlapping Sphincteroplasty:

- for the YOUNGER pt w/ an isolated defect – obstetric injury
- also corrects a thinned perineal body or RVF – RVF is NOT a contraindication
- prone jackknife, curvilinear perineal incision, dissect laterally to find the sphincter
- preserve the scar to hold suture, overlap w/ mattress sutures
- do NOT dissect > 180° laterally – pudendal nerve
- separate IAS imbrication adds NOTHING to durability
- levatorplasty when appropriate
- < 5 yrs: 70-90% improved – DECAYS with time, few are ever fully continent
- satisfaction stays high despite symptom deterioration

Ventral Mesh Rectopexy:

- D'Hoore 2004. External prolapse or significant intussusception w/ FI
- dissect the rectovaginal septum to the pelvic floor, mesh fixed to the promontory
- FI score ↓ 45%, constipation also improves. Treatment of choice for prolapse in Europe
- expanded to Oxford grade 3-4 internal prolapse w/ FI + obstructed defecation
- risks: mesh erosion (vagina, bladder, rectum), mid-rectal stricture, RVF, chronic pelvic pain

Gracilis Transposition:

- single proximal pedicle – easy harvest, no donor deficit
- native muscle fast-twitch fatigable; electrode + generator retrains it to slow-twitch fatigue-resistant
- infection, pain, device failure, erosion, outlet obstruction all common
- DYNAMIC graciloplasty no longer available

Magnetic Anal Sphincter (Fenix):

- magnetic beads on a titanium string around the sphincter → ↑ outlet resistance
- rectal pressure separates the beads to defecate
- HDE approval after failed medical & surgical Rx. Success in ~half at 5 yrs
- infection, pain, gluteal swelling, explantation. Currently NOT available

Artificial Bowel Sphincter (ABS):

- cuff around the anus, pump in labia majora or scrotum, reservoir in the space of Retzius
- 60-70% success in end-stage FI
- HIGH infection & erosion → revision then explantation. No longer available in the US

Antegrade Continence Enema (MACE):

- reverse appendicostomy or cecostomy, flush antegrade
- for the selected MOTIVATED adult
- 47-100% still using the stoma despite it being time-consuming
- stomal stenosis 8-50%, often needs reintervention

Stoma:

- end colostomy or ileostomy when all else fails – it IMPROVES QOL, offer it
- resect the rectosigmoid leaving a 3-4 cm cuff to prevent mucous discharge
- anal encirclement (Thiersch) is obsolete – colostomy is better

BOTTOM LINE

Start conservative in everyone – diet, bulking, loperamide, biofeedback

Defect or no defect → SNM is the workhorse second step

Young pt w/ an isolated obstetric defect → sphincteroplasty, but counsel on decay

Prolapse or high-grade intussusception → fix the prolapse, NOT SNM

All else fails → MACE or a stoma

62 PELVIC FLOOR DISORDERS: LOW ANTERIOR

RESECTION SYNDROME

DEFINITION & SCOPE

LARS: disordered bowel function after rectal resection, leading to a detriment in quality of life

Delphi consensus (325 experts, incl. 156 pts w/ LARS): 8 symptoms + 5 consequences

- Dx = at least ONE symptom AND at least ONE consequence

8 Symptoms:

- variable & unpredictable bowel function, altered stool consistency, ↑ frequency, repeated painful stools
- emptying difficulty, urgency, incontinence, soiling

5 Consequences:

- toilet dependence, preoccupation w/ bowel function, dissatisfaction w/ bowels, strategies & compromises
- impact on mental & emotional wellbeing, social & daily activity, relationships & intimacy, roles & responsibilities

Sx in the first year: FI 20-80%, urgency 30-90%, fragmentation 30-70%, painful defecation 20-50%, clustering

Must have had part of the rectum REMOVED – otherwise it is not LARS

- definitive chemoXRT for cervical or anal SCC gives the same picture – major bowel sx in 50%

MECHANISM

Poorly understood, none of it validated

- loss of the rectal reservoir
- autonomic denervation from the pelvic dissection
- ↑ colonic motility
- XRT damage to rectal mucosa
- ↓ rectoanal sensitivity
- ↓ anal resting pressure

LARS SCORE

5 questions, total 0-42. Validated in many countries – the mainstay of clinical assessment

- incontinence to flatus: never 0, < 1x/wk 4, ≥ 1x/wk 7
- leakage of liquid stool: never 0, < 1x/wk 3, ≥ 1x/wk 3
- frequency: 1-3 per day 0, 4-7 per day 2, > 7 per day 4, < 1 per day 5
- clustering (BM again w/in 1 hr): never 0, < 1x/wk 9, ≥ 1x/wk 11
- urgency: never 0, < 1x/wk 11, ≥ 1x/wk 16

0-20 No LARS – 21-29 Minor LARS – 30-42 Major LARS

No existing tool covers all 8 symptoms – severity still needs conversation and a bowel diary

INCIDENCE & NATURAL HISTORY

Major sx in 58% at 3 mo, 46% at 12 mo, 41% at 54 mo

- for some it is a LIFETIME condition

Treat it as a CHRONIC dz – like DM or HTN, needing repeated adjustment

RISK FACTORS

Age, female > male, tumor height from the anal verge

Extent of the mesorectal dissection

Neoadjuvant or adjuvant pelvic XRT

Fecal diversion – loop ileostomy, even 3 months of it

Anastomotic leak – inflammation causes irreparable pelvic floor muscle damage

Continence does NOT protect – 85% stay continent after most/all of the rectum is out, 42% still get LARS

POLARS nomogram: preoperative online tool predicting LARS – use it in counseling

PREVENTION & COUNSELING

No intervention has been PROVEN to prevent LARS

- avoid pelvic XRT where the oncology permits
- close the stoma early if feasible
- colonic J pouch / colectomy: better function short term, NO difference vs end-to-end by 18 mo
- mechanical bowel prep and duration of diversion both under study

Discuss an END COLOSTOMY as a real option

preoperatively w/ EVERY pt – not just the wheelchair-bound or those w/ baseline dysfunction

Informed consent MUST cover projected bowel dysfunction, not just peri-op complications

DIAGNOSIS

Start asking about bowel function ~3 mo after restoration of continuity

Exclude the correctable LARS MIMICS first

- pancreatic malabsorption, small bowel bacterial overgrowth, 5-FU induced lactose intolerance
- radiation enteritis, fat & bile salt malabsorption → trial of cholestyramine
- anastomotic stricture, ischemic conduit, tumor

RECURRENCE

Flex sig for LARS-like sx in the first 5 yrs – esp. sudden worsening or inadequate surveillance

Reassess bowel sx after treating any mimic

TREATMENT

Sequential and ADDITIVE – never a replacement for what already works. Add ONE intervention at a time

Set expectations, educate, give WRITTEN instructions, keep a bowel diary

- a systematic protocol dropped mean LARS score 31 → 18, major LARS 51.9% → 26.3%
- RCT: algorithmic care beats ad hoc care

MEDICAL

Fiber + water for consistency, then bulking agents (psyllium) – ↓ fragmentation & clustering

Loperamide next – acts on intestinal muscle only

- opiates (diphenoxylate-atropine, tincture of opium) are CNS-active and controlled

- the CLUSTERING phenotype may get worse on opioids
Amitriptyline 10-25 mg qhs when the above fails – ↓ FI score in 85% (open label)
Cholestyramine for bile salt diarrhea
Reassure the pt that LONG TERM use of these agents is not harmful
Probiotics: VSL#3 RCT after ileostomy closure showed NO effect

SKIN CARE

Zinc barrier cream (Calmoseptine) or lanolin, copiously, up to the dentate line
Antifungal powder dusted on w/ a cotton ball if superinfected; a dry cotton ball to wick moisture
AVOID chemical wipes, abrasive paper, drying soaps – use a washcloth, wet paper, or a bidet
2% lidocaine over the barrier cream for severe pain, until the anoderm re-epithelializes

PELVIC FLOOR RETRAINING (BIOFEEDBACK)

Run it in PARALLEL w/ medical therapy
Systematic review, 5 trials / 321 pts: 4 of 5 showed improved bowel function – all low quality
Open-label RCT: benefit is in the FI component, overall LARS score unchanged
Best in the MOTIVATED pt whose dominant symptom is FI
If emptying, clustering, or fragmentation dominates and no gain after 6-8 sessions → STOP

RETROGRADE COLONIC IRRIGATION (RCI)

NOT an enema – 500-1000 mL tepid water to fill the entire LEFT colon, then evacuate
28 Fr Foley + tube-feeding bag hung for hydrostatic pressure, or a kit (Peristeen)
~30 min, same time daily – conditions the bowel, buys a clean rest of the day
Works regardless of the predominant symptom
- RCT vs PTNS: ↓ stools day & night, better LARS and Wexner scores
- open-label RCT: LARS score 35 (major) → 12 (mild)
- Martelucci et al: sx RETURN on discontinuation, 85% asked to resume

Minimum 6 mo trial before escalating to surgery

SACRAL NEUROMODULATION

Test phase then implant – see Ch 61
Systematic review, 10 studies: 94 tested, 75 (79.8%) went on to permanent implant
- median improvement in LARS sx 67%
- LOWER than SNM success in benign FI, but still meaningful

Reasonable in ANY LARS pt who has failed the above, before a stoma

PTNS: office or home, intriguing data – NOT FDA approved for bowel dysfunction (sham-negative FI RCT)

ANTEGRADE CONTINENCE ENEMA (ACE)

Appendicostomy, Chait or MACE tube, cecostomy, or percutaneous endoscopic cecostomy (PEC)
Meta-analysis, 17 studies / 426 pts: 83.6% success in FI at median 39 mo

PEC series: 73% success in FI, 90% after restorative proctectomy

- CCIS 14.3 → 2.7 at 6 mo, drifts back to 10.4 by 2 yrs
Consider it in the motivated pt BEFORE a permanent colostomy

MACE tube data in LARS is thin – still investigational

FECAL DIVERSION

LAST RESORT, after everything else has failed

Prefer a COLOSTOMY – but respect the blood supply after sigmoid & rectal resection

Make the very best stoma you can – the goal is to improve QOL, not trade one problem for another

BOTTOM LINE

Ask at 3 months. Score it. Exclude the mimics – stricture and recurrence FIRST

Fiber → loperamide → amitriptyline → RCI & biofeedback → SNM → ACE → stoma

Nothing comes off as you climb – every step is ADDITIVE

63 PELVIC FLOOR DISORDERS: SEXUAL FUNCTION

AFTER COLORECTAL SURGERY IN WOMEN

SCOPE OF THE PROBLEM

Sexual dysfunction = one of the MOST common long-term effects of CRC treatment

- prevalence 19-62% after treatment
- rarely asked about, rarely treated
- > 80% of women say discussing it is important; only 29% are ever asked, < half get any information

Female sex is INDEPENDENTLY a/w lower sexual function & enjoyment after CRC

30-40% of women sexually active before treatment become INACTIVE after

- > half report a change in body image

Occurs after BENIGN dz as well as CA – and after abdominal as well as pelvic operations

Anxiety, depression, and fatigue amplify it

Unlike most treatment effects, it does NOT resolve within the first 1-2 yrs

QoLiRECT: pre-treatment sexual activity 29% women vs 41% men → 25% vs 34% at 1 yr

NORMAL RESPONSE & CATEGORIES OF DYSFUNCTION

Four phases: desire → excitement (arousal) → orgasm → resolution

Dysfunction categories: desire, arousal, orgasmic, and sexual PAIN disorders

Most common complaints: loss of desire, numbness / loss of sensation, genital pain, delayed orgasm

NEUROANATOMY – WHAT PELVIC SURGERY INJURES

Sympathetic:

- T12-L2 → anterior to the aorta → superior hypogastric plexus at the origin of the IMA
- divides into R & L hypogastric nerves at the SACRAL PROMONTORY
- run posterolateral to the mesorectum
- injury → urinary urgency & incontinence

Parasympathetic:

- pelvic splanchnics run medially along the mesorectal fascia

Inferior Hypogastric (Pelvic) Plexus:

- hypogastrics + pelvic splanchnics, on the PELVIC SIDEWALL
- NVB runs anterolateral to the rectum, lateral to the distal ureter, cervix, vaginal fornix, bladder base, lower lateral vaginal wall
- controls detrusor contractility, VAGINAL LUBRICATION, and genital swelling with arousal
- injury → ↓ vaginal & vulvar blood flow → ↓ lubrication

Danger Points:

- sacral promontory – hypogastrics splayed on the mesorectal fascia
- pelvic sidewall / lateral stalks – inferior hypogastric plexus

- anterolateral dissection at Denonvilliers – the NVB
- Colonic resection generally spares all of it – it is the PELVIC dissection that does the damage

EFFECT OF EACH MODALITY

Surgery:

- TME by definition = a NERVE-PRESERVING sharp dissection b/n mesorectal and parietal pelvic fascia
- bladder denervation fell from 50-60% to < 20% w/ TME; effect on female sexual fxn less well described
- lap vs open TME: NO clear difference in bladder or sexual function
- robotic reported to have less urinary & sexual impairment – studies are almost all in men
- APR vs AR: less sexually active, less frequent intercourse, less arousal & orgasm
- HIGH AR better QOL and function than low AR
- adhesions → dyspareunia. Loss of rectal sexual pleasuring after proctectomy

Radiation:

- damages the pelvic autonomic nerves, microvasculature, and soft tissue
- vulva & vagina turn over rapidly – very radiosensitive
- acute (resolves by ~3 mo): erythema, desquamation, mucositis, congestion
- late & PROGRESSIVE, after a latent period: sloughing → ulceration → necrosis → fibrosis
- endarteritis obliterans → ischemia → friable, hemorrhagic vagina and introitus
- thinning, atrophy, adhesions, ↓ elasticity, shortening, narrowing → vaginal STENOSIS
- premature OVARIAN FAILURE, permanent menopause
- severity tracks the diagnosis, the field, and total dose to vagina & pelvis

Chemotherapy:

- ↓ desire, ↓ frequency of intercourse
- ovarian estrogen loss → thinning, dryness, ↓ elasticity, dyspareunia, orgasmic dysfunction
- vaginal mucositis, tears → infection
- peripheral neuropathy alters touch; alopecia & nausea hit self-image

Stoma:

- worse QOL and MORE sexual dysfunction than non-stoma pts
- kills spontaneity – discomfort and embarrassment during intercourse
- QOL improves GREATLY after reversal

RADIATION – THE DATA

Dutch TME trial: short course 5 x 5 Gy w/ a significant negative impact on sexual activity & function

- Marijnen et al: ↓ sexual interest, pleasure, and satisfaction at 24 mo

- Lange et al: activity fell from > 50% pre-op to 18.4% at 2 yrs. 2/3 of the sexually active developed NEW dysfunction – neoadjuvant XRT the ONLY significant risk factor
- at 14 yrs irradiated women still had more vaginal dryness & dyspareunia than the general population

Bregendahl et al: XRT a/w ↓ vaginal dimensions, dyspareunia, lack of desire, sexual inactivity

- bowel dysfunction itself a/w lack of desire and dissatisfaction

Tekkis et al: FOURFOLD ↑ in dyspareunia w/ XRT vs surgery alone

Bruheim et al (SVQ, 172 pts, 4.5 yrs): more dryness, dyspareunia, ↓ vaginal dimension – interest preserved

Thyo et al (SVQ): XRT → OR 1.80 overall dysfunction, OR 1.72 dyspareunia

Adams et al: sx as frequent at 6-11 yrs as at 1-5 yrs – late effects DO NOT fade

Sexual pain in 1 in 5 women after pelvic XRT – and they still WANT intercourse

BENIGN DISEASE & IPAA

Sexual dysfunction in 40-60% of women w/ IBD

Nonoperative UC pts have NORMAL fertility – neither the dz nor the medical Rx is the problem

- it is the PELVIC POUCH. Fertility reduced 44-82% after IPAA
- LAPAROSCOPIC IPAA improves fertility vs open – less tubal & adnexal scarring

IPAA a/w ↑ miscarriage, need for ART, C-section, stillbirth, preterm birth – evidence quality poor

Controversy persists on routine C-section post-IPAA to protect the sphincter

Pelvic floor surgery can be “curative” for incontinence and still injure sensation, blood flow, and anatomy

FERTILITY

Colonic resection: likely NO effect on fertility

Resection BELOW the peritoneal reflection: autonomic injury + adhesions → ↓ fecundity

5-FU: little effect. Oxaliplatin & irinotecan: gonadal effect unclear

XRT at current dosing may cause premature ovarian failure

Preservation:

- established: ovarian transposition, embryo cryopreservation
- investigational: ovarian tissue cryopreservation, ovarian suppression, apoptotic inhibitors

Guidelines: DISCUSS infertility risk before treatment, even though the CRC-specific data are thin

MEASUREMENT

PFDC Working Group (2020) selected TWO instruments as best practice

FSFI-9:

- 9 items – desire, arousal, lubrication (x2), orgasm (x2), satisfaction (x2), pain
- validated in peri- and postmenopausal women
- use when sexual function is a SECONDARY endpoint and brevity matters

PISQ-IR:

- 20 items, condition-specific (12 items in 4 domains if not sexually active)
- use in women w/ KNOWN pelvic floor disorders

Combine exam + clinical interview + a validated questionnaire

TREATMENT

Multidisciplinary: CRS, psychologist / psychiatrist, sex therapist, pelvic floor PT, med & rad onc, nutrition, geriatrics

Desire:

- HRT ± testosterone for treatment-induced menopause or hypogonadism, if no oncologic contraindication
- psychosexual therapy, CBT, mindfulness, couple therapy

Arousal:

- topical estrogen, psychosexual therapy (sensate focus), guided fantasy / masturbation

Orgasm:

- sensate focus, mindfulness, couple therapy, VIBRATOR therapy

Sexual Pain:

- superficial dyspareunia from treatment-induced menopause → HRT, vaginal estrogen where feasible
- estrogen contraindicated → nonhormonal vaginal MOISTURIZERS to restore pH and moisture content
- intimate lubricant to ↓ friction
- graduated vaginal DILATION within a sensate-focus framework – breaks the pain / anticipation / avoidance cycle
- deep dyspareunia w/ adhesions, fibrosis, stenosis, shortening → dilation, though evidence & compliance are POOR

Myofascial Pelvic Pain:

- increasingly recognized after colorectal surgery and adjuvant Rx, worse w/ intercourse
- myofascial trigger point release, biofeedback, electrical stimulation

Proactive dilation after pelvic XRT is NOT routine care – and surgery interrupts it. Coordinate the two

COUNSELING – BEFORE THE OPERATION

The pt will almost never raise it – embarrassed, ashamed, assumes it is trivial next to the CA

The PROVIDER must bring it up. Pts are consistently more comfortable when we do

Our barriers: no time, no privacy, surveillance takes priority, no referral pathway, no knowledge of the treatments

Cover BEFORE surgery: stoma & spontaneity, dyspareunia, dryness, fertility, and that dysfunction may be PERMANENT

Ask again at every f/u – the late effects surface after a latent period

64 PELVIC FLOOR DISORDERS: MALE GENITOURINARY DYSFUNCTION AS A CONSEQUENCE OF COLORECTAL SURGERY

OVERVIEW

Autonomic nerve dysfunction complicates many colorectal procedures – benign disease included. Prevalence 20-80%

Magnitude depends on which nerves, extent of damage, and PRE-OP function

Manifests as erectile dysfunction, orgasmic/ejaculatory dysfunction, urinary retention ± overflow incontinence

Beyond the knife: XRT, systemic chemo, long-term fecal diversion

AUTONOMIC ANATOMY

SYMPATHETIC

- T12-L2 → superior hypogastric plexus, fibers around the distal aorta to the bifurcation
- coalesce into the bilateral hypogastric nn. at the SACRAL PROMONTORY, course laterally around the rectum
 - join the nervi erigentes at the ANTERIOR PERITONEAL REFLECTION → inferior hypogastric plexus (IHP)
 - IHP courses anterior to Denonvilliers' fascia to supply the GU tract

Function: ejaculation primarily, erection to a lesser degree

- expulsion (1st): SYMPATHETIC – seminal fluid into the prostatic urethra
- emission (2nd): SOMATIC – contraction of penile musculature, fluid out of the urethra

Injury: retrograde ejaculation

- aberrant bladder innervation → bladder atonia, retention & overflow incontinence

PARASYMPATHETIC

S2-S4, predominantly S3 → pelvic splanchnics (nervi erigentes), ascend bilaterally to the IHP

IHP runs the PELVIC SIDEWALL, then at the prostate dives deep to Denonvilliers' – seminal vesicles, prostate, posterior bladder

- IHP carries BOTH parasympathetic and sympathetic fibers

Function: erection – vasodilation & engorgement of the penile vascular sinuses

- “micturition center” S2-S4, heavily regulated centrally

Injury: nervi erigentes or IHP → IMPOTENCE

- IHP also gives retrograde ejaculation (sympathetic fibers within it)
- parasympathetic bladder pathway → detrusor-sphincter dyssynergia & functional outlet obstruction

WHERE THE NERVES ARE INJURED

Risk ↑ as the operation goes LOWER into the confined pelvis

Oncologic > non-oncologic resection, though the literature conflicts

Sigmoid & upper mesorectum, IMA origin:

- superior hypogastric plexus, R & L hypogastric nn. – readily apparent, easy to preserve here
- GU dysfunction after anterior resection is quite LIMITED

Mid & upper rectum:

- IHP and nervi erigentes – anatomy less apparent, narrow workspace impairs exposure
- stay SUPERFICIAL to Denonvilliers' mobilizing the anterolateral rectum – minimizes but does NOT eliminate it

APR: HIGHEST risk – autonomic injury + disruption of pelvic floor anatomy + body image

MIS vs. open:

- magnification exposes the lateral, anterior & presacral planes – all nerve territory
- despite that, impact is VARIABLE. All small, single center, retrospective. Robotic vs. lap equally unsettled

RADIATION & CHEMOTHERAPY

XRT: erectile dysfunction & infertility the common long-term complications – toxicity manifests for YEARS to DECADES

Urinary: urgency, incontinence, radiation cystitis, ureteral & urethral stricture, fistula

Systemic chemo also detrimental, but LESS so than XRT or surgery

FUNCTIONAL METRICS

Pelvic Floor Disorders Consortium – multispecialty group standardizing pelvic metrics so all specialties speak with one voice

SEXUAL

IIEF-15: RECOMMENDED by the consortium. Most cited instrument for male sexual function

- 5 validated domains: erectile fxn (6 q), orgasmic/ejaculatory (2), desire (2), intercourse satisfaction (3), overall satisfaction (2). 10 languages

IIEF-5 (SHIM): brief & practical – erectile fxn items 2, 4, 5, 15 + intercourse satisfaction item 7

- drops orgasmic/ejaculatory, desire, overall satisfaction

EPIC: 50 q – urinary (12), bowel (14), sexual (13), hormonal (11). Thorough but impractical, validated ONLY in prostate CA

Gay Sexual Functioning Inventory: Rosser et al. 2019 (Restore Study), 37 items, insertive AND receptive anal sex

URINARY

Naughton review – metrics weighted by strength of evidence. Highest ranked:

- UDI: 19 LUTS, validated only in women. UDI-6: both sexes, English only

- King's Health Questionnaire: 10 factors, both sexes, multiple languages
- Danish Prostatic Symptom Score: 12 symptoms – frequency and distress
- ICS-male: 20 symptoms. Short form = voiding subscore (5 q) + incontinence subscore (6 q)

TREATMENT – ERECTILE DYSFUNCTION

PDE5 Inhibitors:

- ↑ systemic nitric oxide, but require INTACT neurologic input
- therefore LESS successful in the neurogenic impotence that follows pelvic surgery

Vacuum Constriction Device:

- oldest, most widely used, LEAST EXPENSIVE. Minimal risk – bruising at the base. Satisfaction 50-80%
- complaints: interrupts intimacy, unnatural erection, blocked ejaculation, anorgasmia

Intracorporeal Injection:

- still effective for NEUROGENIC ED after pelvic surgery – erection in 90%, satisfaction ~80%
- alprostadil single agent. Bimix (papaverine/phentolamine), Trimix (alprostadil/phentolamine/papaverine) – more effective, more complications
- pain at site, hematoma, penile fibrosis, priapism. 30-70% stop using it over time

Intraurethral Alprostadil:

- success 60% – lower than injection, but wider acceptance, fewer complications. Penile pain, mild urethral bleeding

Penile Prosthesis: after medical options fail

- inflatable: most natural, deflatable – more malfunction & revision, needs manual and mental dexterity
- semirigid rod: easy, but permanently semirigid and hard to conceal
- both: infection and erosion → explantation. Fibrosis impairs penile length

RETROGRADE EJACULATION / ANEJACULATION

“Dry orgasm” – urethral sphincter fails to close, ejaculate passes into the bladder

NOT harmful, orgasm PRESERVED. Causes male infertility

Post-surgical cases do NOT respond to pseudoephedrine

Most seek no treatment unless trying to father children – semen harvested from urine and washed → assisted fertilization

URINARY DYSFUNCTION

Long-term urinary dysfunction after pelvic surgery is

UNUSUAL – retention ± overflow incontinence

Short term: indwelling catheter + alpha blockade usually relieves it

Persists → chronic intermittent catheterization, taught in the office

Non-recoverable bladder, or a pt who cannot self-cath → long-term indwelling or suprapubic

COUNSELING & SPECIAL POPULATIONS

Build rapport PREOPERATIVELY – baseline function

predicts post-op function, and consent must name the specific dysfunctions that may follow

Post-op, ASK – pts will not volunteer it, and both sides are embarrassed. Reassure that function typically improves with time

Cultivate a urologist with an interest in male GU dysfunction

Gay & Bisexual Men:

- > 30% of the general population sees the anus and rectum as sexual organs
- more mood & anxiety disorders, depression, suicidal ideation vs. heterosexual men post-treatment; less familial, social & partner support
- Anodyspareunia – pain w/ receptive anal intercourse – a recognized consequence of anorectal & pelvic surgery
 - 82% avoided anal sex, 49% restricted to insertive only, 31% relationship disrupted, 15% unable to take new partners
- switching from receptive to insertive role is NOT trivial – it changes initiation, affection & identity

Fecal Diversion:

- near-universal alteration in body image, lifestyle change 80%, impaired sexuality 40%
- QOL better in non-ostomates INDEPENDENT of bowel function, and ↑ risk of suicide in ostomates

65 PELVIC FLOOR DISORDERS: MIDDLE AND ANTERIOR

PELVIC COMPARTMENT

THE THREE COMPARTMENTS

Anterior: bladder & urethra. Middle: uterus & vagina.

Posterior: rectum & anus

POP = downward descent of pelvic organs through the muscular pelvic floor – best explained to the pt as a PELVIC FLOOR HERNIA

Anterior & middle compartments are the most susceptible
Prolapse of uterus & vagina is 2x as common as the other organs – hence the focus there

Sx: vaginal or perineal bulge / pressure w/ standing, exercise, straining – resolves when the activity stops

More than one compartment is usually involved

EPIDEMIOLOGY & RISK

US prevalence 3-50%, depending on the definition used.

Disorder of the older adult, ages 70-79 (peak 71-73)

Prevalence ↑ 40% with each decade of life

Lifetime risk of POP surgery by age 80: 12.6%

> 300,000 POP procedures/yr in the US. Ambulatory costs
↑ 40% from 1996 to 2005

Wu: symptomatic POP will ↑ by ≥ 46% by 2050 – others call this an overestimate (↓ parity, ↑ elective C-section)

Primary POP:

- VAGINAL PARITY – the strongest predictor in women < 60
- 2 vaginal deliveries RR 8.4, ≥ 4 vaginal deliveries RR 10.9 vs. nulliparous
- advancing age
- obesity (BMI > 25)
- hysterectomy for a non-POP indication ↑ cumulative risk 5% – not settled

Recurrent POP:

- age < 60 at the time of the primary VAGINAL repair
- obesity
- Stage III or IV at initial presentation

Recurrence needing a 2nd repair: 30-50% in pre-2000 data, 6-30% in modern series

Less well studied: chronic constipation, connective tissue disorder, menopause, smoking, HRT, COPD, DM, ethnicity, family Hx

EVALUATION

Symptoms:

- sexual: bulging, heaviness, pressure, pain, ↓ pleasure, ↓ body image
- urinary: leaking, urgency, frequency, weak stream, incomplete emptying, splinting, positional voiding, pain
- defecatory: stool or flatus leakage, incomplete evacuation, straining, urgency, splinting or digitating, pain
- ASK about bowel function and dyspareunia – they will not volunteer it

Instruments:

- PFDI – 20 q, prolapse + anal + urinary distress
- PFIQ – 7 q, life impact
- PISQ – 12 q, sexual function in women w/ pelvic floor disorders

Exam:

- left lateral, standing, or lithotomy. Bladder & rectum EMPTY. Remove any pessary or tampon
- pelvic AND rectal exam, at rest and with strain. Palpate the levators through rectum or vagina
- if the full extent is not seen in lithotomy, repeat STANDING with Valsalva – give the prolapse time to protrude

POP-Q

Measures the genital hiatus (GH), perineal body (PB), total vaginal length (TVL), and each compartment

All measurements taken during VALSALVA except the TVL, and all referenced to the HYMENAL RING

- Aa: 3 cm proximal to the hymen on the anterior wall = the BLADDER NECK
- Ap: 3 cm proximal to the hymen on the posterior wall
- Ba / Bp: most distal point of the anterior / posterior wall w/ Valsalva, b/n the A point and the apex
- C: cervix, or the vaginal cuff if prior hysterectomy
- D: posterior vaginal fornix – present ONLY if a cervix is present
- A & B points negative if inside the hymen, positive if outside

Overall stage = the worst compartment:

- Stage 0: Aa, Ba, Ap, Bp all = -3, and C or D ≤ (TVL-2)
- Stage 1: leading edge < -1
- Stage 2: leading edge ≥ -1 but ≤ +1
- Stage 3: leading edge > +1 but < (TVL-2)
- Stage 4: leading edge ≥ +(TVL-2)

ADJUNCTIVE TESTING

Hx & physical alone is SUFFICIENT to plan the operation in most

Test further only when the exam does NOT explain the symptoms – MC indication is urinary Sx

- urodynamics for leakage, frequency, or difficulty voiding – done with a comfortably full bladder
- office US at the time of urodynamics – confirms PVR, evaluates levator ani
- dynamic MR or cinedefecography – intussusception, rectocele, enterocele; MR also images ligament & muscle

Urodynamic findings:

- incomplete emptying: large PVR
- stress incontinence: leakage w/ ↑ intra-abdominal pressure and NO detrusor contraction
- ↑ bladder sensation: early desire to void at low volume, no abnormal rise in detrusor pressure
- detrusor overactivity: involuntary contraction during FILLING
- detrusor underactivity: poor contraction during VOIDING

NONOPERATIVE MANAGEMENT

Asymptomatic → educate and reassure. Nothing to correct
Pelvic floor muscle training may improve the bulge

sensation – POPPY: does NOT prevent the need for corrective surgery

Local or systemic estrogen – limited evidence for slowing progression

Pessary:

- effective, short-term, low-risk option in up to 90% of women
- for Stage II-IV, but LESS successful in Stage III & IV
- pt should manage it herself – remove, clean, replace q 3 months
- up to 9% get mild vaginal wall ulceration → resolves with local estrogen

OPERATIVE MANAGEMENT

For Stage II-IV SYMPTOMATIC pts who failed conservative therapy. Symptoms MUST match the exam findings

Goal = restore normal anatomy

- (1) obliterative vs. reconstructive
- (2) transvaginal vs. transabdominal
- (3) which compartments to repair concurrently

Age ≥ 80 → most urogynecologists go TRANSVAGINAL – fewer serious post-op adverse events. MC complication is UTI

Anterior wall prolapse is often a/w apical prolapse and SUI – consider concomitant repair, especially in severe prolapse

OBLITERATIVE

For the older woman NO LONGER SEXUALLY ACTIVE.

High satisfaction, low recurrence

Le Fort Partial Colpocleisis:

- uterus present. Denude a strip of anterior & posterior vaginal epithelium and suture them together
- leaves a channel to drain cervical secretions. NORMAL PAP required first

Colpectomy / Tight Colporrhaphy:

- no uterus. Excise vaginal mucosa from the urethrovesical junction to the apex
- reduce the prolapse w/ sequential interrupted sutures until it sits above the levator plate
- wide perineorrhaphy incorporating the levators, reducing the GH to 1-2 cm
- address urinary Sx at the same sitting – a common cause of poor post-op QOL

TRANSVAGINAL RECONSTRUCTION

Apical suspensions require ADEQUATE vaginal length

Sacrospinous Ligament Fixation (SSLF):

- ligament palpated through the vaginal wall as a thick cord running from the ISCHIAL SPINE posteriorly to the sacrum

- evert & open the vagina, mobilize any enterocele, the bladder and the rectum away first
- attach the ligament to the FULL THICKNESS of the fibromuscular layer of the vaginal apex
- peritoneum is NOT entered – PREFERRED when pelvic adhesions are suspected
- 36% recurrence at 2 yrs

Uterosacral Ligament Suspension (USLS):

- INTRAPERITONEAL. Ligaments lie posterior and medial to the ischial spine
- can be joined in the midline to strengthen the repair
- usually done with a vaginal hysterectomy. 35% recurrence at 2 yrs

Anterior Colporrhaphy (cystocele):

- transverse incision from the urethrovesical junction to the apex, mobilize the bladder, plicate the muscular wall & paravaginal tissue
- 40-60% success in RCT. Mesh more durable but 10% erosion

Posterior Colporrhaphy (rectocele):

- midline posterior incision, mobilize the perirectal fascia as far laterally as possible
- small defects: vertical mattress sutures. Larger: plicate the puborectalis
- do NOT over-plicate – it narrows the vaginal lumen
- 80-95% success, and NO improvement with mesh

ABDOMINAL SACROCOLPOPEXY

Best candidates: shortened vaginal length, intra-abdominal pathology, or risk factors for recurrence (age < 60, Stage III-IV, BMI > 26)

Safe and effective laparoscopically or robotically

SUPRACERVICAL hysterectomy PREFERRED – mesh holds more durably on cervical tissue and avoids a vaginal incision

- a vaginal incision ↑ the risk of post-op mesh exposure

Mesh sutured to anterior AND posterior cervix and vaginal wall → longitudinal ligament of the sacrum just below the promontory

Best results with synthetic POLYPROPYLENE mesh
Recurrence 7% (synthetic mesh) vs. 38% (cadaveric fascia) at 5 yrs

Apical suspension ↓ the reoperation rate

Hysteropexy / sacro-hysteropexy if uterine preservation desired – NO benefit demonstrated to preservation

STRESS URINARY INCONTINENCE

Commonly a/w anterior vaginal wall prolapse

FIRST LINE conservative: pelvic floor PT, incontinence pessary

- if overactive bladder coexists: PT + behavior modification first, then antimuscarinics

Midurethral Sling:

- MOST EFFECTIVE surgical option. Introduced in the 1990s. Autologous tissue or polypropylene
- retropubic: mesh through a small vaginal incision, hugging the pubic bone, exiting suprapubically

- trans-obturator: through the obturator foramen, exiting lateral to the labia majora
- both ~80% long-term subjective & objective success. Mesh infection risk only 2%
- sacrocolpopexy + retropubic sling: 50% ↓ in post-op SUI

THE MESH STORY

FDA warnings 2008 and 2011 → mesh is NO LONGER placed vaginally for an anterior or posterior repair

Mesh IS still used routinely for APICAL support – sacrocolpopexy and hysteropexy

CARE trial: overall mesh erosion 10.5% with intra-abdominal mesh

- use LARGE-PORE polypropylene for sacrocolpopexy – lowest infection rate in CARE

Chughtai: sacrocolpopexy mesh PLUS midurethral sling mesh had the highest infection rate, OR 2.13 (95% CI 1.76-2.56) – a dose-response to mesh

D'Hoore: ventral mesh rectopexy + mesh posterior colporrhaphy also ↑ infection – blamed on the VAGINAL WOUND exposing the mesh

MULTIDISCIPLINARY MANAGEMENT

No consensus on which approach is superior. Anatomic failure up to 25%

13% need a repeat operation within 5 yrs; 30% will need repeat POP surgery in their lifetime

Failure rates are HIGHER for vaginal and native tissue repairs than for mesh sacrocolpopexy

Most recurrences are attributed to the POSTERIOR compartment

Lap sacrocolpopexy: 82% symptomatic improvement

- of the failures, 2/3 mid-vaginal recurrence, 1/3 urinary & bowel dysfunction; 28% had posterior anatomic failure

RCT of 3 rectocele repairs: 21-51% still had straining, incomplete evacuation, or defecatory dysfunction

Anatomy that matters:

- distal vagina supported by the perineum, lower rectovaginal septum, and levator ani
- mid vagina supported laterally to the levator ani fascia
- upper vagina supported by the CARDINAL-UTEROSACRAL complex
- anterior low rectal wall is FUSED to the mid vaginal wall – sustained pressure from the rectal side bulges it into the mid vagina = rectocele
- lateral ligaments carry the middle rectal a. and nerves, and support the low rectum above the levators

Posterior compartment caveats:

- barium trapping and rectocele SIZE do NOT correlate with symptoms – NOT a criterion for surgery
- no correlation b/n posterior wall prolapse, constipation, and anorectal function testing

- the rectocele may be the RESULT of obstructive defecation rather than the cause
- biofeedback FIRST for rectocele & ODS – 71% response rate
- correcting the bulge does little for constipation or intussusception
- if straining continues post-op, pressure on the repair recurs the prolapse
- occult intussusception can progress after a single-compartment repair – get a dynamic study when Sx and exam disagree

Combined sacrocolpopexy + rectopexy:

- SAFE, low recurrence, improves constipation, FI and QOL. Adds no significant morbidity, mortality, or operative time
- supracervical hysterectomy first if the uterus is present
- urogyn does the hysterectomy, the anterior vesicovaginal dissection, and begins the posterior dissection along cervix & posterior vagina
- sacral dissection: identify the promontory, common iliac a. & v., the RIGHT URETER, and the midline middle sacral a. on the anterior longitudinal ligament
- CR surgeon opens the rectovaginal space down to the pelvic floor
- PRESERVE the lateral stalks – minimizes post-op constipation
- ventral rectopexy mesh to the anterior rectum or perineum, then to the promontory alongside the sacrocolpopexy mesh
- sutured rectopexy may be as effective here – permanent suture through the lateral ligaments, tied below the promontory stitches
- cover the mesh with peritoneum. Cystoscopy at the end for ureteral integrity
- decide on the posterior repair / perineorrhaphy LAST – often unnecessary once the apex and the low rectum are supported
- resection rectopexy + sacrocolpopexy is reported safe, but with a higher complication rate
- AVOID constipation & straining post-op – softeners and laxatives

66 MISCELLANEOUS: PEDIATRIC COLORECTAL

DISORDERS

HIRSCHSPRUNG'S DISEASE

PATHOLOGY & PRESENTATION

Fxnl obstruction from ABSENCE of ganglion cells in the distal bowel – deletion in the long arm of chromosome 10

- absent in (1) Auerbach's: b/n circular & longitudinal muscle, (2) Henle's: submucosa, (3) Meissner's: superficial submucosa

85% have ganglion cells at the rectosigmoid = SHORT SEGMENT

- long segment = ganglion cells only proximal to the middle colic vessels

Three windows of presentation:

- Neonate: no meconium in the first 24 h – vomiting, distention, feeding intolerance
 - DRE may cause explosive passage of stool & gas and transiently relieve
- Enterocolitis: after marginally tolerating the first weeks of feeds – explosive and/or bloody stool, vomiting, fever
 - untreated → sepsis, shock, perforation
- Late: weaning off breast milk or onto solids – distention, markedly dilated colon, chronic laxative use

Encopresis: overflow pseudoincontinence from being overly constipated

DIAGNOSIS

The diagnosis belongs to the PATHOLOGIST – absence of ganglion cells > 1 cm proximal to the dentate line is DIAGNOSTIC

- suction rectal, punch, or full-thickness biopsy all acceptable. Over age 2 needs a general anesthetic
- CALRETININ staining has ended the need for 2nd opinions on H&E and for acetylcholinesterase assay

Contrast enema: dilated colon to the point of aganglionosis – may not show in the early neonatal period

Anorectal manometry: absent RAIR is SUGGESTIVE, NOT diagnostic

- unreliable in the first 12 months (sens 75-90%) – use as a TRIAGE tool for who needs biopsy

INITIAL MANAGEMENT

Individualize: primary pull-through vs. leveling colostomy vs. ileostomy vs. rectal irrigations

Rectal irrigations = high colonic saline enemas. Plain enemas are the WRONG answer – they will not pass

- treats enterocolitis, buys an elective operation, avoids a stoma, and teaches the family a skill they will need post-op

Do NOT default to a primary pull-through in enterocolitis w/ systemic illness, or in long-segment / TOTAL COLONIC disease

- both carry ↑ risk of post-op enterocolitis

Leveling colostomy demands an EXPERIENCED pathologist – calretinin is NOT available on frozen section

- none available, no transfer possible → diverting ILEOSTOMY suffices, even in total colonic disease

THE PULL-THROUGHS

The principle is one line: bring ganglionated bowel to within 1 cm of the dentate line

All three can be done laparoscopically. Most have IAS dysfunction – each operation builds in a way to address it

Swenson: full-thickness circumferential rectal resection, usually transanal, ganglionated bowel to the distal 1 cm of rectum

Soave (first described by Yancy):

- submucosal transanal dissection, then full-thickness, anastomosis 1 cm above the dentate, leaving a small aganglionic cuff
- modified to divide that cuff posteriorly from below the levators = built-in posterior internal sphincterotomy

Duhamel:

- retains aganglionic rectum; ganglionated bowel anastomosed END-TO-BACK just above the dentate = posterior sphincterotomy
- MUST resect aganglionic rectum proximal to that anastomosis or it dilates into a "RECTAL SPUR"
- chosen for LONGER-segment disease, where retained rectum aids continence

Boley modification (Swenson or Soave): elliptical anastomosis – 1 cm from the dentate anteriorly, 0.5 cm posteriorly – lengthens it and builds in a posterior sphincterotomy

With these modifications SUCCESS RATES ARE EQUAL across the three

MIS: shorter LOS, fewer complications, less narcotic. Lap vs. transanal: NO difference

- Swenson & Soave: circumferential anastomotic stricture – expect post-op dilations. Duhamel: neorectal stasis and spur

COMPLICATIONS & OUTCOMES

Recurrent Sx, in order: (1) irrigations, dilations or laxatives, (2) contrast enema, (3) REPEAT BIOPSY of the neorectum

- no ganglion cells → REDO pull-through (coloanal anastomosis)
- ganglion cells within 1 cm of the dentate + dilated rectum → dilations, IAS Botox, or sphincterotomy
- significant Duhamel spur → laparoscopic resection of the spur

HAEC:

- most "outgrow" it by the teenage years, many after age 5. Microbiome theory dominates
- maintenance: antibiotic prophylaxis, prebiotics, probiotics

- life-threatening or refractory → Botox, sphincterotomy, stoma, or ACE

Adult outcomes:

- fecal incontinence ~15%, constipation ~5%. Bowel QOL is NOT compromised despite these
- worse w/ longer-segment disease, and function WORSENS with age – the aging HD pt needs care
- SNS evolving as an adjunct in selected pts

COLONIC & RECTAL DUPLICATIONS

1 in 100,000 live births, 60-80% in the first 2 yrs. Colon & rectum = 17% of enteric duplications, tubular or cystic, on the MESENTERIC side

Long and distal ones a/w GU and SPINAL anomalies incl. fistulae – contrast & endoscopy BEFORE operating

Rectal duplications are PRESACRAL – part of the

CURRARINO TRIAD: anorectal stenosis, presacral mass, sacral anomaly

- mimic perirectal abscess/fistula when infected → drain EXTERNALLY, never internally, to protect the plane

US is the imaging of choice in infants – a DOUBLE-LAYER wall marks enteric wall vs. simple cyst

Complete excision is the GOAL; fistulae to GU tract, spine, or perineum MUST be excised

- long duplication in a small child: partial resection + long stapled fenestrations to create a common lumen

CONSTIPATION

Onset in the first year in 17-40%. Presents neonatally, at toilet training (2-4 yrs), and at school entry

Referred to GI: 50% off laxatives at 6-12 months, 10% on laxatives but well, 40% need escalation

- 50% off laxatives at 5 yrs, 80% at 10 yrs. > 3 months of Sx before referral → delayed recovery

MECONIUM history is the key – documented passage within 24 h makes HD very unlikely

- none in the first 48 h → contrast enema for a transition zone AND rectal biopsy

Workup of medical failure:

- Sitz markers throughout the colon = inertia; stacked at the puborectalis = pelvic floor dysfunction
- colonic manometry (age > 2) predicts who responds to ANTEGRADE FLUSHES

Ladder:

- anismus (withholding Hx, stacked markers, ARM) → anal or pelvic floor BOTOX. Often NO dose adjustment for size
- cecostomy / ACE for antegrade flushes
- SNS: adult data do NOT support it for constipation. Entirely refractory → fecal diversion

Idiopathic constipation: vicious cycle of a colon enlarging and becoming less functional → impaction → encopresis. Resecting all dilated rectosigmoid does not cure, but improves

Volvulus & perforation:

- unusual. Predisposed by constipation and abnormal colonic fixation. Cecal, transverse, sigmoid
- transverse or sigmoid: colonoscopic detorsion FIRST – straightens the mesentery, allows a prep
- definitive = segmental resection, average age 7 yrs. Expect a second volvulus at another segment
- spontaneous perforation from dilation or Ogilvie's – MC the CECUM (Laplace)

RECTAL PROLAPSE

Full thickness by definition, but infants & small children can have MUCOSAL prolapse only. Peak in the FIRST YEAR

A SYMPTOM, not a disease – constipation usually; also cough, diarrhea, parasites, neoplasm, malnutrition, CF, pelvic floor weakness

90% under age 4 resolves with conservative mgmt; rare after 6. Over 4 → look for a neurologic (spinal cord) or muscular defect

SCREEN FOR CYSTIC FIBROSIS before operating – prevalence 11.1% in children with prolapse

Sclerotherapy – MC initial intervention (2018 APSA survey), simplest and most benign:

- 30% saline 83% / 97% cure after 1 / 2 injections; 5% phenol in almond oil 91% / 100%; 70% ethanol 96% / 98%

Local operations:

- Thiersch (1953): absorbable 1-0 PDS circumferentially at the anal opening over a Hegar – weak pelvic floor or CF
- linear cauterization 94%; modified Thiersch (cautery + sclerotherapy + Thiersch) 67% single, 90% combined

Failures:

- Altemeier – temporary FI in some. Posterior sagittal rectopexy up to 25% recurrence
- lap rectopexy ~5% recurrence, only complication is constipation
- FIRST LINE for irreversible conditions and full-thickness prolapse = ABDOMINAL RECTOPEXY
- as in adults, resection rectopexy PREFERRED with intractable constipation and prolonged transit

PERIANAL ABSCESS & FISTULA-IN-ANO

A different disease from the adult version. 0.5-4.3% of all children, MC under age 2

M:F 12:1 under age 2, falling to 2-3:1 after. MC positions RIGHT and LEFT LATERAL – not the adult midline pattern

Fistula after abscess 20-85% – 40% after surgical DRAINAGE vs. 16% without

Step-up management:

- hygiene & diaper care, sitz baths, antibiotics
- needle aspiration (18 g): recurrent abscess 8%, subsequent fistula 11% – a VIABLE definitive treatment

- no resolution in 24 h → I&D. Counsel that 1/3 recur as an abscess or a fistula
- post-op antibiotics do NOT ↓ abscess recurrence, but DO ↓ subsequent fistula formation

Fistula-in-ano:

- spontaneous resolution 17-80% within a year; antibiotics do not affect it
- only 15-25% of children < 2 have a fistula at the primary operation – do NOT hunt for one and risk the sphincter
- THM: do NOT do a fistulotomy in an infant
- age > 8: 50% recurrence, 25% progression to fistula – up-front surgery or fibrin glue once sepsis clears
- recurrent fistula over 12 months old → rule out immunosuppression and IBD. Crohn's diagnosed as young as age 2
- IBD or immunocompromised: drain, DRAINING SETON, biologics or immunosuppressants FIRST

ANAL FISSURE

Complication of constipation, MC at age 2. Posterior midline in infants; females more often ANTERIOR midline

Off-midline or multiple → BIOPSY for Crohn's, malignancy, TB, STD, immunodeficiency

Most heal on bowel management – 10% to > 95%, the high end with a longer, more intensive regimen

2% GTN: Kenny – 84% healing in BOTH arms. Tander & Sönmez – 82-84% vs. 43-50% placebo

Botox: resolution equal to myectomy – 94% vs. 89% – without the long-term cost

Anal dilation: double-blind RCTs show NO benefit. IAS myectomy: relieves 2/3, incontinence cost may take YEARS to surface

Incontinence AND anal stenosis reported in up to 30% at this age – operate on an infant's sphincter with EXTREME caution

CECOSTOMY & ANTEGRADE FLUSHES

Cecal access for a nightly antegrade flush – an alternative to COLOSTOMY, aimed at social continence

MACE (Malone, 1990): appendix to the umbilicus.

Antireflux modification wraps it in cecal wall to stop retrograde leak

ACE / appendicostomy: catheter in nightly, gravity flush, out. No permanent foreign body, hidden in the umbilicus

Percutaneous cecostomy: tract not manipulated daily, but a permanent foreign body – granulation, dislodgement

Run a RETROGRADE enema program FIRST – if that works, antegrade access will work

Flush: escalate to 20 mL/kg normal saline nightly, sit 45 min. Failing that, add irritant, lubricant, promotility agents

TRANSITIONAL CARE

Begin the discussion at AGE 12; complete b/n 16 and 21 – a clearly executed transition, NOT a drift away from pediatric care

MACE flush management passes from the pediatric surgeon to the ADULT GI – communication is everything

ANORECTAL MALFORMATION

1 in 2000-4000 live births. Spectrum from congenital anal stenosis to cloaca. 60% have associated GU, skeletal & SPINAL anomalies

Krickenbeck major defects: perineal (cutaneous) fistula, rectourethral bulbar, rectourethral prostatic, rectovesical (bladder neck), vestibular fistula, cloaca, no fistula, anal stenosis

The HIGHER the fistula, the WORSE the bowel outcome – no embryologic pelvic floor development, plus associated anomalies

- higher: more FI, less constipation, flat perineum.
- Lower: more constipation, less FI

PSARP in infancy – Kraske posterior sagittal approach, separate the rectum from the genital (F) or urologic (M) structures and place it within the identified pelvic floor and external sphincters

Long-term function:

- 30-50% of the more proximal fistulas are CONSTIPATED
- STIMULANT laxatives PREFERRED – osmotics ↑ leakage in a compromised sphincter
- ~20% of adults with ARM report weekly FI. Rx = a fine balance of diet, fiber, antimotility agents
- 30% urinary incontinence. 10% of men ED, 15% ejaculatory dysfunction. Female sexual dysfunction not studied
- SNS reasonable if at least one S3 root is intact – many have sacral anomalies. Failing that → cecostomy or MACE
- rectal prolapse in an ARM pt = long-standing constipation. Treat that FIRST, then limited Altemeier – excise the redundant rectum and sew it to skin, as there is no anus

Cecostomy problems that reach the adult surgeon:

- granulation tissue → topical steroid or silver nitrate.
- ACE stenosis → local VY flap; Chait skin stenosis → dilation

Others arriving in the adult clinic: IBD, FAP, mitochondrial cytopathy, colonic inertia, pelvic floor dysfunction

Juvenile polyps: benign polyps around age 4, long pedicle, MC posterior rectal wall. Blood surrounding the stool. Self-amputate and disappear – biopsy one to confirm benign

67 MISCELLANEOUS: CONSIDERATIONS FOR GERIATRIC PATIENTS UNDERGOING COLORECTAL SURGERY

FRAILTY

SCOPE

56% of new CRC dx in the US are in pts > 65 yo
Life expectancy – healthy 75 yo male: 18 yrs; 82 yo: 10 yrs
- severe comorbidity drops these to 6 and 2 yrs

No consensus on the age cutoff (65 vs. 70 vs. 75) – do NOT treat on chronologic age alone
- two errors: undertreatment for age, overtreatment for degree of frailty

*Frailty: reduced tolerance to surgical stress 2/2
progressive physiologic decline – prevalence 25-46%
by tool*

GOLD STANDARD = Comprehensive Geriatric Assessment (CGA), multidimensional & interdisciplinary
- domains: function, comorbidity, cognition & mental health, nutrition, social support, fatigue, polypharmacy, geriatric syndromes
- only ~7000 geriatricians in the US; < 1/4 of surgeons routinely collaborate w/ one → the surgeon MUST screen

Screen EVERYONE – the tool chosen matters less than the universal nature of screening

SCREENING INSTRUMENTS

- ECOG PS: 0-4, frail ≥ 1 – cancer burden on function
- Katz ADL: 0-6, frail < 5 – functional independence
- Mini-Cog: 0-5, frail ≤ 2 – 3-word recall (3 pts) + clock draw w/ correct time (2 pts), 2-3 min
- Timed Up & Go (TUG): frail ≥ 20 sec
- fTRST (Flemish Triage Risk Screening Test): 0-6, frail ≥ 2
- G8: 0-17, frail ≤ 14
- NRS: nutrition – BMI, weight loss, food intake. ASA: 1-5
- CACI (Charlson age comorbidity index): 0-42, frail ≥ 6

TUG: rise from chair w/o arms, walk 10 ft (3 m), turn, return, sit

- PREOP study: prolonged TUG → 50% major complications vs. 13.6% if normal
- absolute risk 50% vs. 24.8% for ASA 3-4 – TUG outperforms ASA

fTRST – 5 questions, works in the ED: cognitive decline (2), lives alone / no help (1), reduced mobility or falls in past 6 mo (1), hospitalized in past 3 mo (1), polypharmacy ≥ 5 meds (1)

- Zatzoni: score ≥ 2 predicts ↑ morbidity, mortality, LOS in emergency general surgery

Office Algorithm (< 5 min):

- > 70 yo or appears frail → G8, TUG, Mini-Cog, falls in past 6 mo
- FIT: G8 ≥ 15 AND Mini-Cog ≥ 4 AND TUG ≤ 20 sec AND no falls → usual care

- any one abnormal → geriatric assessment + geriatrician at the MDT → adapted care

ASSESSMENT DOMAINS

Function:

- basic & instrumental ADLs – prepare a meal, house cleaning, take your own meds

Cognition:

- preop impairment → ↑ post-op delirium

Polypharmacy:

- consult pharmacy if > 10 medications

Social Support:

- lives alone $\neq$ fit – it means vulnerable if function declines
- who can care for you, transportation. Involve social work & case management preemptively

Patient's Wishes:

- goals up front, presented at the MDT, and REVISITED – goals change during treatment

PREHABILITATION

Preop optimization to ↓ morbidity and speed return to baseline function. 4-6 weeks

- delay from dx to surgery does NOT compromise cancer outcome (Curtis)
- rectal CA during nCRT = the ideal prehab window

Medical:

- optimize DM, thyroid, cardiopulmonary. Smoking cessation is paramount
- ↓ EtOH (withdrawal, delirium), ↓ polypharmacy w/ a pharmacist

Physical:

- CPET → ventilatory anaerobic threshold (VAT); low VAT a/w morbidity – gold standard but often unavailable
- alternative: 6-min walk test or TUG, no equipment
- resistance + endurance + breathing, 2-3x/wk, 30-45 min. Supervised beats home – compliance

Nutritional:

- protein target 1.2-1.5 g/kg/day; whey before exercise
- sarcopenia predicts outcomes and occurs at LOW, NORMAL, and HIGH BMI
- Gillis meta: nutritional prehab ± exercise ↓ LOS, faster return to baseline function

Psychological:

- anxiety & depression ↓ by HADS w/ prehab

Minnella: pts w/ the WORST baseline walking capacity gain the most

~30% do NOT respond; Carli RCT found no benefit of prehab vs. post-op rehab

SURGICAL CONSIDERATIONS

ER pathways are feasible in geriatric pts, same benefits, no ↑ complications

- modify: AVOID NSAIDs & extreme fluid restriction w/ baseline renal insufficiency

- DM w/ gastroparesis – watch early feeding, aspiration
 - not an MIS candidate → still apply the rest of the pathway, open pts benefit too
- MIS: age ALONE is not a contraindication, incl. redo and rectal CA
- pneumoperitoneum – caution w/ severely ↓ EF or severe pulmonary dz
 - experienced laparoscopists only – AVOID long anesthesia and late conversion

Emergency surgery: much higher morbidity & mortality, worse as frailty ↑ – no window for optimization

Do NOT do a lesser operation on the basis of age

- obstructing sigmoid CA w/o mets → resect, don't just divert. Goal remains R0
- node-positive → adjuvant chemo based on POST-OP frailty
- disease-specific survival is the SAME in geriatric pts given standard-of-care treatment
- when risk truly outweighs benefit – name the futility, refer to palliative care / hospice

POST-OP & FUNCTIONAL RECOVERY

GOSAFE: ≥ 1 Clavien-Dindo complication → DOUBLE the functional decline (59% vs. 31%)

Geriatric Comanagement:

- Shahrokni, 1009 comanaged vs. 846 surgery-only: adjusted 90-day mortality 4.3% vs. 9.2%
- earlier recognition of geriatric syndromes – delirium, falls

Delirium:

- risk: prior delirium, caregiver dependence, cognitive impairment – identify PREOP, tell the family
- prevention: orientation & safety, mobilization, day-night rhythm
- meta-analysis of multicomponent nonpharmacologic prevention: OR 0.47 (0.38-0.58)
- AVOID bedrest – brief bedrest costs muscle mass that is hard to regain
- rehab starts at admission, continues past discharge

Functional Recovery:

- GOSAFE: complete functional recovery (ADL > 4, Mini-Cog > 2, TUG < 20 sec) in only 26%
- 31% of pts alive at 90 days had DECREASED functional capacity
- QOL still improved at 90 days (EQ-5D 0.76 → 0.80) – driven by pain and anxiety/depression

Organ-Specific Recovery:

- measure it – LARS score, Cleveland Clinic Fecal Incontinence Score
- fecal incontinence is a frequent reason for nursing home placement
- if continuity will mean incontinence (poor tone, low rectal CA), permanent colostomy is the better functional result

- loop ileostomy is reversed less often in geriatric pts, and readmits for dehydration / AKI
- reversal unlikely → PERMANENT COLOSTOMY up front
- permanent stoma does NOT worsen HRQL in geriatric pts (Orsini)

RECTAL CANCER – TASK FORCE (ESSO / ESCP / SIOG / ACS CoC)

Use FRAILITY, not chronologic age, for risk stratification

- mandatory screening: G8, Mini-Cog, TUG, falls hx
- frail → geriatrician on the MDT; FIT → the standard algorithms for younger pts

TME is advised for ALL rectal pts who can undergo it, independent of age

- open / lap / robotic / taTME not contraindicated by age
- laparoscopy PREFERRED, by experienced surgeons. Robotic no better, costs more
- taTME by experts only – ↓ conversion, long-term data lacking
- local excision: NOT recommended after XRT – ↑ morbidity

Locally Advanced:

- nCRT ↑ local control, but toxicity can cost the pt a curative operation
- capecitabine CONTRAINDICATED in renal failure
- contact X-ray brachytherapy: residual < 3 cm after EBRT, or monotherapy for cT1 < 3 cm
- watch & wait for cCR, expert MDT only – no reliable predictor, use size & rate of shrinkage
- adjuvant chemo after nCRT + surgery is NOT advised in geriatric pts

Synchronous Liver Mets:

- liver surgery ONLY with curative intent, neoadjuvant chemo if primary asymptomatic
- AVOID synchronous resections

68 MISCELLANEOUS: HEALTHCARE ECONOMICS

GLOBAL PERSPECTIVE

US 2018 healthcare spend: \$3.6 trillion = 16.9-17.7% of GDP, \$11,172 per person

- Switzerland 2nd at 12.2% GDP, \$7317/person. Germany 11.2%, Japan 10.9%, UK 9.8%
- only 90.8% of the US population was insured in 2018 – 35th of 36 OECD countries
- US life expectancy 78.6 yrs – LOWEST of the G7, 28th of 36 OECD
- US cancer outcomes, incl. 5-yr colon & rectal CA survival, BEAT the OECD average – but avoidable mortality is HIGHER

Total spend = quantity of resources × price

- utilization is SIMILAR to other countries – same beds, physicians, nurses
- it is the PRICES: physician & nurse pay highest of 11 high-income countries; common drugs ~2x that average
- administration = 8% of US spend, \$787/capita vs. OECD \$90 – almost 9x

US HEALTHCARE FINANCE

Federal govt = largest single purchaser. > \$1 trillion in 2018 = ~25% of the federal budget

Coverage: employer-sponsored 49%, Medicaid 20%, Medicare 14%, individual 6%, military 1%

Medicare:

- 1965, LBJ Great Society, over AMA objection. ENTIRELY federally funded
- eligible: ≥ 65 yo, ESRD, ALS, permanent disability
- Part A (Hospital Insurance): inpatient, SNF, some home health, hospice. Payroll taxes
- Part B: VOLUNTARY, medically necessary & preventive physician services. General revenue + premiums
- Part C = Medicare Advantage (1982): private insurers, capitated, covers A + B. 34% of beneficiaries in 2019
- Part D: drugs, from the MMA of 2003, private plans. 70% of beneficiaries by 2019

Medicaid:

- 1965 companion to Medicare, for the poor. Federal-state PARTNERSHIP – feds set core eligibility, states administer
- federal share 50-78% by state per capita income; 90% for ACA newly-eligible adults ≤ 138% FPL in expansion states
- CHIP (1997): uninsured children above the Medicaid threshold
- covers 20% of the population – ~half of all deliveries, > 60% of nursing home residents, 43% of enrollees are children

Employer-Sponsored:

- WWII origin – wage & price controls blocked raises, so insurance was offered instead. Pre-tax

- ACA: employers > 50 employees must offer affordable coverage or pay a penalty
- avg family premium 2019 = \$20,576, employee pays 30%; employee share ↑ 22% over 5 yrs vs. wages 14%, inflation 8%

Individual (Non-group):

- ACA Title I created state exchanges. ~6% of Americans

POLICY ISSUES

The ACA:

- 4 mechanisms: employer mandate, individual mandate, state exchanges, Medicaid expansion to 138% FPL
- NFIB v. Sebelius (2012): individual mandate valid under the taxing power, but states CANNOT be compelled to expand
- 36 states + DC expanded. Enrollment ↑ 34% in expansion states vs. 9.1% in non-expansion
- 2017: Congress cut the individual mandate penalty to \$0

Underinsurance:

- high-deductible plan (> \$1300 individual, > \$2600 family) enrollment 14.8% (2007) → 43.4% (2017)
- avg employer-based deductible ↑ 162% from 2009 to 2019
- 40% w/ employer coverage report trouble affording it; ~half postponed care or a Rx for cost in the past 12 mo
- coverage ≠ access. Accepting NEW pts: Medicaid 71.6%, Medicare 77.4%, private 92.4%

Surprise Billing:

- out-of-network charge despite an in-network surgeon at an in-network facility – the pt doesn't pick the anesthesiologist, pathologist, radiologist, or assistant
- Chhabra, 347,000 elective operations: out-of-network bills in 20.5%, mean balance bill \$2011
- more likely w/ complex procedures and w/ complications

Rural Hospital Closures:

- 170 closed since 2005, 19 in 2019. 354 more – 25% of US rural hospitals – financially stressed and at risk
- ~2/3 of 2010-2018 closures were in NON-expansion states; expansion a/w better finances and lower closure risk

PHYSICIAN REIMBURSEMENT

MACRA (2015):

- bipartisan. Repealed the sustainable growth rate (SGR) and its annual “doc fix”
- created the Quality Payment Program (QPP) – pay for VALUE, not volume
- two tracks: MIPS and Advanced APM
- low-volume exemption: < 100 Medicare pts/yr OR < \$30,000 Part B allowed charges – but your fee schedule then stays FLAT

MIPS:

- 4 categories: quality (replaced PQRS), cost (from the Value-Based Payment Modifier), promoting interoperability (replaced meaningful use), improvement activities
- final score 0-100. 2019 performance threshold = 30 points
- payment adjustment LAGS 2 years – 2017 data set the CY2019 fee schedule
- adjustments started at $\pm 4\%$, rise to $\pm 9\%$ by 2022
- BUDGET NEUTRAL – the penalties fund the bonuses, so don't expect large upside
- \$500 million set aside for “exceptional” performers – threshold 75 points, up to +10%, OUTSIDE budget neutrality
- report as an individual, as a group (same TIN), or BOTH – w/ both you get the HIGHER score
- group reporting: everyone gets the SAME score, often on irrelevant metrics – a colorectal surgeon scored on diabetic eye exams

Advanced APM (A-APM):

- 5% incentive for hitting payment or patient thresholds, and EXEMPT from MIPS
- > 90% of ACOs are NOT advanced APMs – they take no significant downside financial risk
- the incentives push small practices to join larger organizations

CODING

Two categories – diagnosis and procedure. Hospitals and physicians use DIFFERENT systems

ICD-10:

- WHO has maintained the ICD since 1946. Required under HIPAA
- ICD-10-CM: diagnosis coding, all US settings
- ICD-10-PCS: procedures in the INPATIENT setting – what the facility reports, not the surgeon

CPT:

- AMA trademark, first published 1966. CMS-mandated for Part B and hospital outpatient since 1983
- 5 digits. Also the HIPAA standard for electronic transmission
- Category I: the main set – E/M plus diagnostic & therapeutic procedures
- Category II: supplemental, for performance measurement. NOT billable
- Category III: temporary tracking codes for new technology. Alphanumeric, NO work RVUs → no payment
 - e.g. THD was 0249T until Category I 46948 was created
- CPT Editorial Panel meets 3x/yr; a new Category I code takes 18-26 months

- requires proven efficacy, common US practice, FDA approval if a drug/device, ≥ 5 peer-reviewed articles in US pts
- INADEQUATE LITERATURE is the most common cause of denial

The RUC:

- AMA Specialty Society Relative Value Scale Update Committee
- assigns work RVUs (wRVUs) to new & revised CPT codes. Meets 3x/yr after the Editorial Panel
- it only RECOMMENDS – CMS makes the final payment decision
- ASCRS has NO permanent seat, holds a 2-yr rotating seat about every 4-6 yrs

DRG:

- CMS classification for HOSPITAL payment under the Inpatient Prospective Payment System (IPPS)
- assigned from the primary dx, up to 25 other dx, and up to 25 procedures

CODES BECOME PAYMENTS

Claim is judged on: covered by contract, supported by documentation, properly submitted

Timely filing: as short as 120 days for commercial payers, up to 1 yr for Medicare

Modifiers (2-digit, Level I) – 35 in the 2020 CPT list:

- –57: decision for surgery. The one used most in the office
- –22: substantially greater work than typical. Op note MUST give the reason AND the magnitude
 - “diffuse, dense adhesions from previous laparotomies ... prolonged the procedure by at least X”
- –51: the secondary procedure of a multi-procedure case – usually paid at 50%

Add-on Codes:

- 5-digit, cannot stand alone. Excluded from –51 and from multiple procedure payment reduction (MPPR)
- the colorectal ones: splenic flexure mobilization w/ partial colectomy – 44139 open, 44213 lap

Unlisted Codes:

- when no code fits. Submit the op report, an explanation, and suggested comparable procedures, then negotiate
- labor-intensive – AVOID unless no code or combination can describe the operation

69 MISCELLANEOUS: ETHICAL CONSIDERATIONS

CONFLICT OF INTEREST

DEFINITION & CLASSIFICATION

COI: when "professional judgement concerning a primary interest (pt welfare, research validity) may be influenced by a secondary interest (financial gain)"

- cannot always be avoided – MUST be handled transparently

Financial:

- most easily identified, most likely to undermine credibility of author, journal, and the science
- grants, employment, consultant fees, non-monetary support, drugs/equipment, patents, equity, gifts, trips, meals, paid expert testimony
- situational – amount, involvement, timing & place, mitigation. Even small gifts or an exchange of services shift decisions
- industry-investigator ties gave us laparoscopy, TEM, colonoscopy – preserve them, regulate the behavior

Nonfinancial – subtler, harder to measure, no less dangerous:

- Intellectual: personal views, ideas, moral convictions, strong theoretic opinions
- Professional: personal relationships (friendship or enmity), academic competition, peer recognition & acclaim
- academic pressure – funding, promotion, publication. Successful researchers are BY DEFINITION biased

Determining COI:

- Fineberg reasonable person standard – would a reasonable person read the circumstances as potentially sufficient to influence judgment
- the question is not whether a COI EXISTS but whether it "presents an unacceptable risk of undue influence or bias"
- experts WITHOUT a financial COI deserve the same scrutiny – hidden nonfinancial bias frames the argument

REPORTING & EFFECT ON OUTCOMES

Journals rely on SELF-reporting. Most require a statement; many relevant COIs still go unreported
Physician Payments Sunshine Act (2010): manufacturers must report all financial transactions w/ clinicians holding an NPI → Open Payments Database (OPD), which lets industry-reported COI be checked against self-disclosure

Industry-sponsored studies more often favor the sponsor's product – also true of meta-analyses, systematic reviews, editorials, letters

- influence runs through the research agenda, obfuscation w/ secondary studies, selective analysis, reporting, and interpretation

Vioxx: excess CV events in the Vioxx arm falsely ascribed to a "cardioprotective" effect of naproxen, the control

Cherla (OPD): 70.3% DISCORDANCE b/n surgeon-reported and industry-reported COI – nondisclosers' conclusions more likely to favor industry

100 consecutive ventral hernia articles: 82% had a relevant COI, up to 1/4 undisclosed; authors w/ COI 2x as likely to report industry-favorable results

COI reporting in systematic reviews only 17-65% – the evidence we rate highest

IOM (2011) principles for COI on guideline panels – NOT widely adopted

MANAGEMENT STRATEGIES

Disclosure:

- most used. Does NOT reduce bias, only exposes it. Post hoc, so prone to selection bias
- does NOT mean judgment was affected, that the investigator is biased, or that the relationship was inappropriate
- moral licensing – having disclosed, the individual feels entitled to say and do what they want; and disclosure by a high-profile surgeon can read as EXPERTISE, inflating influence
- necessary but NOT sufficient – judge the COI as any other bias

Elimination:

- divest the relationship, best a priori in the planning stage. Impossible for nonfinancial interests

Balance:

- two opposing groups design the trial, a NEUTRAL party collects and analyzes

Exclusion:

- excluded from evaluation & interpretation; may still take part in trial development
- equity in a company that benefits from a guideline → EXCLUDED from that guideline and its authorship
- exclude all dissent and the homogeneous remainder carries its own unintentional bias

PEER REVIEW

ICMJE "Disclosure of Conflicts of Interest" form – required by most journals, incl. DCR

Reviewers self-assess and recuse; the editor balances reviewer assignment when viewpoints oppose

Editors divest ALL COI before taking the post, recuse on conflicted manuscripts, and are the FINAL arbiter

SURGICAL INNOVATION

THE PROBLEM

Lap chole → the epidemic of CBD injuries; rate fell to open levels w/ experience, then ROSE again w/ single-port taTME → urethral injuries, virtually unknown open or lap
- moratoria in Norway, unusual locoregional recurrences in the Netherlands

Common thread: large promised benefit, rapid adoption, often after a WEEKEND COURSE – no proctor, no institutional oversight

Jaffe, 150 faculty surgeons: 98% learned a new procedure or technology AFTER residency

- what they did: scrubbed in w/ another surgeon, self-directed study. What they said works: proctorship or a mini-fellowship
- barriers: time, and the belief they could do it safely without rigorous training

Reitsma & Moreno: authors publishing on innovative procedures called it research, yet most felt IRB approval unnecessary and only a minority told the pt it was innovative

OVERSIGHT

Drugs and devices are heavily regulated (FDA) – new TECHNIQUES and PROCEDURES are largely unregulated

Society of University Surgeons (2008) – formal review if the innovation is PLANNED, AND any of:

- seeks to confirm a theory about it, OR differs significantly from accepted local practice, OR outcomes not previously described, OR entails potential risk of complication, OR specific/additional consent seems appropriate
- → local surgical innovation committee (IRB if you plan to publish), a national innovations registry, and ADDITIONAL consent specific to the innovation

IDEAL Collaboration – “no innovation without study.” 5 stages:

- 1 Idea / proof of concept: very few pts, very few surgeons. Case reports – technique, outcomes, adverse events, so dangerous methods aren't replicated
- 2a Development: a few innovators & early adopters, small numbers, prospective. Safety, technical & procedural success
- 2b Exploration: more surgeons, broader indications, refinement. Enter pts in a research DATABASE. Safety, clinical & pt-centered outcomes, feasibility
- 3 Assessment: the early majority, many pts, expanding indications. RCT ideal. Mid- & long-term, pt-centered outcomes, cost-effectiveness
- 4 Long-term: audit, regional variation, quality assurance, risk adjustment from registries. Rare events
- ethics approval CONSIDERED at stage 1, MANDATORY for 2a, 2b, and 3

INFORMED CONSENT FOR A NEW PROCEDURE

Cover the operation, indications, material risks & benefits, alternatives, and WHO will perform it

- add the surgeon's training and experience with THAT procedure – pts rank it among the most important determinants, particularly whether it is the surgeon's FIRST case
- “newer must be better” biases pt and surgeon alike – guard against the surgeon's optimism bias

- Ahmed, 244 surgeons: often did NOT discuss the novelty, alternatives, their own experience, or the risks
- IDEAL stage 1: written information, REPEAT appointments, disclose your expertise & experience and why you recommend it

OUTCOMES

Early adopters have an ethical OBLIGATION to track their own outcomes

RCT is gold standard but often infeasible – must define the learning curve and enroll only surgeons past it, unknown risks make powering hard, and equipoise is lost once adoption spreads

- use prospective registries instead – personal, institutional, or national – and PUBLISH the data

Industry drives adoption through courses, apprenticeships, proctorships and grants – recognize the COI

END-OF-LIFE CARE

Three principles: effective COMMUNICATION, accurate PROGNOSTICATION, sound DECISION-MAKING

The question shifts from CAN we to SHOULD we

COMMUNICATION

Early, often, and BEFORE any acute change in condition.

Private, ample time, no outside pressure

- you FACILITATE the meeting, you don't run it. Probe for understanding, establish the level of shared decision-making expected
- prognosis clearly, compassionately, honestly – no false hope. Elicit goals/values and fears/worries; resolve uncertainties b/n family and clinicians
- several clinicians → PRE-MEETING briefing so the message is consistent
- use a PROFESSIONAL translator, NOT a family member – family insert their own values and distort. Pictures and diagrams transcend language

PROGNOSTICATION

Address FUNCTIONAL, COGNITIVE, and

PSYCHOSOCIAL outcomes – not survival alone

SUPPORT trial: half of ICU pts got unwanted treatment, > half were undertreated for pain, many never had their preferences identified or carried out

“Clinical momentum”: a series of individually standard interventions, no one weighing the cumulative effect

Scoring systems predict short-term morbidity & mortality for POPULATIONS – not function or HRQL for an individual

Elderly, esp. w/ dementia – discuss further mental & physical decline after surgery, esp. emergent

Time-Limited Trial:

- for prognostic uncertainty – e.g. the operation plus a defined period of ICU support
- set specific functional & physiologic targets and a time frame. Failure to reach them is evidence the course will not benefit the pt

DECISION-MAKING

The four principles: AUTONOMY, BENEFICENCE, NON-MALEFICENCE, JUSTICE

- NOT hierarchical, and they conflict at end of life.
- Unresolvable → ethics or palliative care consult

Autonomy:

- advance directives – instructional (documented in advance) or proxy (a designated surrogate)
- only 30% of adults have one, the majority > 65 yo
- written without context, often silent on the actual situation – an acute solvable problem on top of a chronic terminal illness
- > 70% of surrogates thought survival after CPR was > 75%; only 20% knew brain damage could follow; only 2% knew the pt could end up on life support
- pts may limit interventions unnecessarily, fearing they will burden family. Views are NOT fixed – revisit at every acute change
- > 75% of surrogates wished to eliminate or limit their role in decision-making

Shared Decision-Making:

- NOT just picking from a menu of options. Pt/surrogate prioritizes goals & values; the surgeon gives options w/ risks, benefits, expected FUNCTIONAL outcomes, and how each aligns
- done properly it shows you when a pt is being OVERTreated or UNDERTreated
- decision aids (Cochrane): pts felt more knowledgeable, clearer on values, risk, and outcomes
- best case / worst case / most likely framework – discuss the OUTCOME, not the pathway

Beneficence:

- ordinary: reasonable hope of benefit, obtainable without excessive expense, pain, or inconvenience. Extraordinary: the opposite, or no reasonable hope of benefit
- double effect – aggressive symptom control may sedate and hasten death. ETHICAL if the primary intention is to benefit the pt
- the healthy elderly pt tolerates the operation; it is the complication that is devastating – no reserve
- age alone is NEVER a reason to deny treatment, but benefit must FAR outweigh risk

Non-maleficence:

- a standard-of-care operation may be nontherapeutic or dangerous near end of life
- narrow therapeutic window that shifts as the pt changes – REEVALUATE frequently
- moral distress: you recognize the proper course but cannot act on it → second opinion from a removed expert, or an ethics consult
- withdrawal of a life-sustaining treatment is justified if EITHER: the pt/surrogate recognizes it no longer matches their goals and values, OR the responsible physician recognizes it is no longer medically indicated

- prefer “no longer medically indicated” to “FUTILITY” – futility is value-based and has never been definable

Justice:

- equality = equal access to available treatments; equity = each gets the support they need, when needed
- demand exceeds resources in EVERY system. Age, social status, ethnicity, culture, disability and insurance all factor into access; marginalized communities engage late and mistrust the system
- some cultures decide collectively or through a senior family member – ask how much information they want and how involved they and their family wish to be. Use the hospital liaison service

70 MISCELLANEOUS: WELCOME TO LITIGATION

SCOPE

General surgeons: 15.3% annual probability of a claim (NEJM 2011)

- 80% sued by age 45; 99% of "high-risk" specialties by 65
- 5-20%/yr by specialty – far fewer end in an indemnity payment

No facts until the verdict form – only EVIDENCE of facts

Stages: I. Initiation (notice → records request → summons & complaint) II. Discovery (written, then your deposition) III. Trial

BEFORE ANYTHING IS FILED

PRIVILEGES

Quality Assurance:

- belongs to the HOSPITAL / medical staff, NOT to you
- attaches only if disclosure made in furtherance of a known, preexisting policy
- foundation Q at depo: "Why did you make the disclosure?"
- so know the reporting rules of hospital, med staff, risk mgmt, carrier

Attorney Work-Product:

- reports to risk mgmt & to the carrier in anticipation of litigation
- carrier = attorney's ostensible agent, even before counsel retained
- also group-practice discussion IF a written policy requires such reporting

Your Own Summary:

- write it EARLY – memories fade
- head it "prepared in anticipation of litigation, for my attorney"
- keep CONFIDENTIAL, OUT of the chart – filing it in the chart WAIVES privilege

RECORDS REQUESTS

A request from the pt or an attorney = notice litigation is coming

Produce EVERYTHING asked for – it will be compared to the later production

- any difference must be explained. Additions or deletions are devastating

EMR: the record is "created" by whoever picks the screens to print

- METADATA discoverable – entry log (who, when) and comment fields

WRITTEN policy defining your "medical record", and audit compliance

THE COMPLAINT

Pt files → summons issues → served personally

States parties, the relationship creating the DUTY, the BREACH, the INJURY, the prayer for damages

Extraordinary Causes – look on page 1:

- failure of INFORMED consent = subcategory of negligence, same elements & damages
- no consent, or a substantially different operation = BATTERY, an intentional tort
- battery, fraud, concealment, IIED → punitive damages
- punitives are NOT insurable; policy likely EXCLUDES intentional torts

Damages:

- General / noneconomic (pain & suffering) – often CAPPED, several (proportionate) liability
 - Special / economic (med expense, lost earnings, future monitoring) – JOINT liability, even at 1% fault
- Individual + PC + group = 3 summonses. Keep all 3.

Response due ≤ 30 days

At pleading stage the judge MUST accept the complaint as true – your consent form is irrelevant for now

- early legal challenges rarely succeed. Expect it to be slow

Counsel screens: who was sued, service, STATUTE OF LIMITATIONS, adequacy of pleading

WHEN YOU ARE SERVED

Keep the envelope. Note date, time, how received

Read the summons – WHO was served (you, your PC, the group)

Notify carrier/attorney IMMEDIATELY. Forward every set Page 1 → extraordinary causes. Last paragraph (prayer) → damages sought

No attorney contact in 10 days → call the carrier

Legal file SEPARATE from the medical chart

Do NOT speak with anyone – partners, staff included

Do NOT do an independent investigation or literature search

Do NOT alter or supplement the records. Ever

Ask what to assemble – records + fax, e-mail, office & cell billing to prove communications

DISCOVERY

INTERROGATORIES

Written Qs, party to party. Broad – biographical, insurance, facts, witnesses, contentions, defenses

Due ~30 days from your attorney's receipt

GOOD FAITH search of chart, your files, and by inquiry to staff

Verified under penalty of perjury → usable as an ADMISSION at trial

Omit a witness → may be BARRED from calling him. Late response WAIVES objections

YOUR DEPOSITION

MOST important part of pretrial discovery

NEVER appear without an attorney – even as a non-party witness

- that is how witnesses become defendants

Scope: anything reasonably calculated to lead to relevant evidence – a sanctioned fishing expedition. Not under oath themselves

Ask if videotaped, discuss attire. Business attire always; lab coat is a call for counsel

Percipient vs. Expert:

- percipient: what you saw, heard, and what you were THINKING at the time – fair game
- opinions formed AFTER suit = expert opinions, remain work-product
- changes only if counsel designates you an expert → 2nd deposition

Standard of Care – the tautology counsel needs:

- SOC = what other reasonably careful surgeons would do in same or similar circumstances
- you are competent & careful, you acted on the facts as they then existed → within the SOC
- best cross = prove the facts you believed were NOT the facts
- so testify to your qualifications, and to what you actually knew at the time

Preparation:

- review ONLY what counsel directs – he controls the universe you accessed
- do NOT read up on the medicine – “if it matters now, why didn’t you know it then?”
- expect to list every document reviewed. Bring the list, give it to counsel
- refresh on what was available at the time: labs, consults, imaging, type & cross

Rules at the Table:

- WAIT for the whole question. Think. Answer directly and succinctly
- ANSWER THE QUESTION ASKED. Nothing more
 - “Did you examine the abdomen?” → “I don’t recall.” NOT “...but I usually do and chart it”
- to expand, ask “May I explain?” – never just keep talking
- ambiguous words: define the word INSIDE your answer. Don’t spar
 - “emergency” = urgent to you, = not-on-the-elective-schedule to OR & anesthesia records
- estimate needs a foundation – observation, memory, or customary practice
 - no foundation → “I don’t know.” Never speculate on what another person did or thought
- refreshed memory vs. reading the chart – “Are you testifying, or is the chart testifying?”
- corrections allowed, but a substantive change will be used against your credibility
 - list changes on a SEPARATE sheet, review with counsel before touching the original
- the signed transcript IS your testimony – for trial, the medical board, and expert work years later

No right to remain silent in a civil case. Every word is an arrow for their quiver

TRIAL

Jury instructed to weigh deposition testimony exactly as if given live

Verdict Form – 3 questions:

- 1. Was the defendant negligent in diagnosis or treatment?
- 2. If so, was that negligence a legal cause of injury?
- 3. If so, what dollar amount of damages?

Burden = “more likely true than not true”. Evenly balanced → verdict FOR the defense

What the Jury Is Told:

- negligent = failed to use the skill, knowledge & care other reasonably careful surgeons would use
- SOC is a standard of CONDUCT, not of RESULT – a bad outcome is not evidence of negligence
- an unsuccessful effort, or a reasonable error, is not necessarily negligence
- choosing one accepted method over another is not negligence, even if wrong in hindsight
- on SOC the jury may consider ONLY expert testimony

An opinion is worth no more than the facts under it – break the facts and it collapses

Know both sides’ experts and the factual basis of every opinion

“Alternative methods” defense: knew them, considered them, chose for stated reasons

In the Courtroom:

- BE THERE – throughout, and certainly for voir dire. The plaintiff will be; the jury is ordered to be
- phone off. No texting, gestures, or reactions
- answer to the QUESTIONER – not the jury (patronizing), not your counsel (weakness)
- worst juror verdict on a witness: evasive, argumentative, nonresponsive
- if they read from your depo, do NOT object or cry “out of context”

Trials rarely run 5 days/wk – ask which days are “dark”.

Flag vacations early

Settle early where the misadventure is indefensible (wrong-site)

- the state medical board investigation is a SEPARATE matter that follows

BOTTOM LINE

Preparation starts the day of the adverse event, not the day you are served

Answer the question asked, and nothing else

71 MISCELLANEOUS: QUALITY

DEFINING QUALITY

NAM: “the degree to which health services increase the likelihood of desired health outcomes and are consistent with current professional knowledge”

Crossing the Quality Chasm – 6 domains:

- Safety – avoidance of harm during care
- Patient-centeredness – respects pt preferences
- Effectiveness – services with a basis in evidence
- Efficiency – no waste of resources
- Timeliness – prompt care, no harmful delay
- Equity – optimal care for ALL pts

DONABEDIAN MODEL (1966)

Structure + Process → Outcome

Structure:

- the context – facility & services, workforce, payment structure
- “Are the right services, equipment, incentives and people available?”
- vaguest domain, hardest for a clinician to move directly
- best-supported structural factors: nurse staffing ratios and VOLUME

Process:

- application of those tools and policies to pts
- “Are the right tools, policies and equipment used for ALL pts?”
- needs (1) consensus on optimal practice and (2) a real GAP between optimal and current
- fails when the evidence is unclear, or compliance is already near 100%

Outcome:

- the result on the pt – SSI, UTI, VTE, mortality, readmission, LOS
- highest face validity, most meaningful to surgeons
- does NOT generate a path to improvement – use it to DIRECT effort, not to fix
- better suited where undesired outcomes are common and standards are not established

Three for any metric: internal QI, external reporting (pts & payors), financial incentive/penalty

SCIP – THE PROCESS EXEMPLAR

Joint Commission + CMS, mandatory, publicly reported on Hospital Compare, tied to payment

Measures: abx timing / selection / discontinuation, normothermia, VTE chemoprophylaxis, Foley out by POD

At launch < 60% of pts were getting preop abx

Drove uptake of evidence-based SSI prevention – but studies linking SCIP performance to complication rates were LACKING

RETIRED Dec 2014, CMS moving to outcome-based measures

Other process measure: adjuvant chemo within 3 months of surgery for stage III colon CA

CHOOSING THE MEASURE

ADMINISTRATIVE VS. REGISTRY DATA

Administrative (“claims”):

- generated in the normal course of business, the basis for reimbursement
- ICD-10 CM codes. Only what is DOCUMENTED can be measured
- cheap, but abstracted by staff with no clinical background → inaccurate, misleading

Registry:

- purpose-specific abstraction, separate from the hospital's financial machinery
- trained abstractors, tight definitions, audited → better data
- significantly higher cost

The two give DIFFERENT pictures of the SAME outcome

- definitions differ, and so does the accuracy with which each applies its own definition
- the choice is a tradeoff of data quality vs. cost

EVALUATIVE VS. TRANSACTIONAL DATA

Evaluative: a judgment was applied – e.g. SSI. Someone decides, by criteria → false-positive & false-negative possible

Transactional: generated automatically, no judgment – reoperation, readmission, LOS

More outcomes are measurable with evaluative data, but institutions differ in HOW they evaluate → biased reports

- US News uses transactional data only. NSQIP and CMS do not

COMPOSITE VS. SINGLE OUTCOMES

Complications are rare → small denominators → noise reads as signal

- pressure ulcer 0.1% → 0.3%: a tripling, or 2 events?

Never read an O:E ratio without the NUMERATOR and DENOMINATOR

Fixes: aggregate over a longer period, or aggregate outcomes into a composite

PSI-90 (CMS Value-Based Purchasing) – composite of 10: pressure ulcer, iatrogenic pneumothorax, in-hospital fall w/ hip fx, periop hemorrhage/hematoma, postop AKI needing dialysis, postop respiratory failure, periop PE/DVT, postop sepsis, wound dehiscence, unrecognized puncture/laceration

Composite is more sensitive to a global problem, but gives NO actionable target

RISK ADJUSTMENT

Complications track case complexity and comorbidity
Adjust poorly and the surgeon who takes the hard cases looks worse

NOT always worth it – where the outcome is directly caused by a process failure (specimen labeling error, iatrogenic pneumothorax) adjustment adds nothing

Charlson Index: scores each comorbidity to predict risk of dying

EVIDENCE GRADING (ASCRS STANDARDS COMMITTEE)

Level I: meta-analysis of multiple well-designed controlled studies; RCTs with low false-pos & low false-neg (high power)

Level II: ≥ 1 well-designed experimental study; RCT with high false-pos and/or false-neg (low power)

Level III: nonrandomized, controlled, single-group, pre-post, cohort, time, or matched case-control series

Level IV: well-designed case series

Level V: case reports and clinical examples

Grade A: type I evidence, or multiple II/III/IV. B: generally consistent II/III/IV. C: II/III/IV but inconsistent. D: little or no systematic empirical evidence

QUALITY OF LIFE INSTRUMENTS

Reliability: same result on the same population

Validity – 3 types:

- Criterion: measured against a gold standard
- Content: how much of the spectrum within the scale is covered
- Construct: how well it measures an unobservable phenomenon

Responsiveness: sensitivity to change – detects TRUE differences

No metric is perfect. On the horizon: pt-reported outcomes, return to health, pt satisfaction

REGISTRIES & ACCREDITATION

ACS NSQIP

“Best in class” surgical registry. Trained abstractors, periodically audited

Origin: VA (National VA Surgical Risk Study), 1990s, to benchmark 30-day outcomes

- risk adjustment central – VA population older, male, more comorbid

2001: extended to non-VA hospitals by ACS with AHRQ support

Colorectal a key focus – colectomy and proctectomy procedure-specific variables

Participants get RISK-ADJUSTED outcomes across a wide range of complications

Data alone does not improve quality → ACS Quality & Safety Meeting, regional collaboratives, pilots

ISCR (Improving Surgical Care and Recovery) – ACS-NSQIP + Hopkins Armstrong Institute, > 300 hospitals.

↓ LOS, ↓ complications, faster return of bowel function

ACCREDITATION

MBSAQIP (bariatric): ACS + ASMBS. The most mature surgical accreditation program

- standards for physical & human resources and practice; site visits; mandatory outcome reporting
- CMS mandated accredited centers for Medicare bariatric surgery, then DISCONTINUED the

endorsement in 2013 – no benefit in the Medicare analysis

NAPRC: ACS Commission on Cancer + OSTRiCh, 2018

- 22 standards, effectiveness verified by site surveyors
- dedicated, highly trained surgeons, pathologists, medical & radiation oncologists
- structured, DOCUMENTED multidisciplinary tumor board reviewing every rectal CA pt
- ongoing quality metric monitoring and QI activity required
- only 12 hospitals verified, 5 in process at publication

Accreditation → improved outcomes relationship remains UNCLEAR

IMPROVEMENT

CULTURE

Surgical culture works AGAINST a safety culture:

- error equated with incompetence → errors not discussed
- effect of stress on decision-making minimized
- steep OR hierarchy → nurses and staff will not call out an error
- professional autonomy prized over cooperation
- safety held to be important, but never promoted from a priority to a VALUE

ALTERNATIVE PAYMENT MODELS

Payment shifting from services rendered to value. CMS:

APMs are the long-term strategy

APMs hold providers AND hospitals accountable for quality and TOTAL cost

- fundamentally an outcome strategy – cost is the proxy for quality, being easier to measure
- upside from savings, and DOWNSIDE RISK from excess cost

BPCI: 90-day episode cost after selected procedures.

Voluntary now, mandatory over time

- total joint arthroplasty already mandatory in 67 markets
- a major bowel surgery bundle is in development
- concerns: population heterogeneity, and few levers to control cost

DOING IT

QI must cover the whole continuum – from the decision to operate to full recovery at home

Requires a multidisciplinary team that is LED and RESOURCED, and aligned with institutional leadership

Use a scientific approach to design the intervention and to assess its impact

Measurement that is not translated into improvement is wasted

72 MISCELLANEOUS: PRACTICE MANAGEMENT

PRACTICE MODELS

For the first time, more physicians are EMPLOYED than independent

- AMA: 47.4% employed vs. 45.9% practice owners
- 70% rise in hospital-based physicians 2012 → 2018
- Midwest 55% employed, up from 34% in 2012

INDEPENDENT – SINGLE SPECIALTY

Predominant colorectal model until the last decade

Common focus → cohesive culture, easy consensus, standardized care across sites

Vulnerable if real competition appears – no diversification, no health system behind it

Survival depends on ANCILLARY revenue (imaging, lab, ASC) to carry the overhead

INDEPENDENT – MULTISPECIALTY

Grown as both a growth strategy and a DEFENSIVE response to system consolidation

More specialties → better support for ancillaries, esp. lab and imaging

Risk: service lines with competing capital demands → disharmony

- a strong GOVERNANCE model is mandatory

Before joining, understand: vision, culture, governance, financial strength, path to partnership, shareholder obligations

PRIVATE EQUITY / EXTERNAL INVESTMENT

Most states bar non-physicians from owning a practice → PE forms a management services organization (MSO)

- practice pays the MSO a flat fee or a % of revenue
- a % arrangement must AVOID structuring as fee splitting

PE takes majority of the MSO, pays partners a large upfront cash payment

- priced on EBITDA, a 5-15× multiple
- partners accept a SALARY CUT for a period, and contribute their ancillaries to the MSO

Payout comes years later when PE sells its position – you get a new partner you did not pick

Most deals to date in dermatology; now expanding into GI

- a colorectal group with a strong ASC and lab is an attractive target
- the group ultimately LOSES control of its destiny

EMPLOYED MODELS

Drivers: flat/declining professional reimbursement vs. medical inflation in overhead

- systems will pay up because they capture the downstream contribution margin
- plus EMR and technology cost, value-based transition, consumerism/retail clinics
- and less entrepreneurial appetite in trainees carrying large debt

Better structure = the DYAD – a management expert paired with a physician executive

- preserved autonomy + system support is the offset to BURNOUT

Nontraditional employers now in the market – insurers, Amazon, Apple, Walmart

- largest employer of physicians in the US = OptumHealth (UnitedHealthcare), 50,000 physicians

BEING ACQUIRED

Build partner consensus first. Be clear on the

MOTIVATION to sell, and prioritize objectives, before negotiating

Agree BEFORE signing on: purchase price for hard

assets, contract length, compensation model (guarantees + productivity incentives), governance, staffing models and ratios, stipends, decision rights

- ALL leverage passes to the acquirer once the contracts execute

Federal statutes govern: fair market value and commercial reasonableness

- so the system is not buying referrals. Compensation can NEVER be tied to downstream revenue

FINANCE

Revenue – expenses. That is the whole model, in every practice type

Large systems command higher commercial rates – partly for their obligation to treat the uninsured

Revenue cycle: small-to-mid practices → OUTSOURCE, to a company incentivized on collections

CODING

NOT taught in residency or fellowship – get formal training at orientation

Every encounter needs a DIAGNOSIS code and a PROCEDURE code tied to it

- ICD-10 (WHO) – diagnoses, extensively revised 2015
- CPT – services rendered: E/M codes and/or procedure codes

EOB from the payer documents allowable, adjustments, denials, and pt responsibility

Routine AUDITING of the surgeon's billing is best practice – compliance and fraud risk

COMPENSATION

Salary should correlate to work performed and align incentives with practice goals

Judge the WHOLE package – benefits, work hour expectations, call

Revenue-minus-expense:

- but expense allocation varies wildly (fixed vs. variable), and startup cost distorts a new hire
- breeds “eat what you treat”, and undervalues non-remunerative contribution
- fix by pooling excess at the specialty or group level, then distributing by formula

RVU-based:

- almost ESSENTIAL in a multispecialty group – it standardizes work across specialties

- accounts for time, skill, judgment/experience, and liability risk
 - 3 components: work effort, practice expense, malpractice expense – adjusted geographically
 - CMS sets RVU values per CPT code, with society input on new procedures
 - “PAYER BLIND” – value does not vary by insurance. Key for a not-for-profit mission
 - decouples pay from revenue cycle performance
 - nonclinical work (committees, service line development, mentoring) → stipends or reduced thresholds
 - now the preferred academic model: protected % time, guaranteed salary + RVU-based clinical pay
- Ancillary income: distributed EQUALLY among shareholders, generally
- it is ILLEGAL to tie income of any type to the volume of referrals

REFERRALS & MARKETING

The four A's: availability, affability, ability, ACCOUNTABILITY

Top 5 resources adults use to pick a physician:

- word of mouth 65.9%
- insurance provider directory 51.9%
- physician rating sites (Vitals, Healthgrades) 22.8%
- hospital website 10.8%
- consumer review sites (Yelp) 10.5%

Say yes to consults. Use a slow early schedule to build relationships

Meet referring providers in person, briefly. Bring food for the WHOLE office

- know the established referral patterns of your partners FIRST
- ask what would make their lives easier – fax referral forms, laminated originals
- offer same-day slots for acute rectal pain, abscess, fissure – keeps their pts out of the ER
- offer same/next-day diagnostic colonoscopy
- target providers like you – gender, race, language.

Minority pts tend to choose providers of their own race

Referral coordinators control where referrals go – appreciate them

“Leakage”: the EMR tracks every referral. Demonstrate quality and value to the system, or be treated as an outsider

Contracts for employed physicians MUST include a provision that pt choice will be respected

MARKETING

Adults 50-64 use media MORE than any other group – 51% TV, 31% digital. Not just millennials

Facebook Ads = the digital billboard – target by age, location, gender, education, interest

Healthgrades (~100 million searches/yr) and Vitals are the first stop for most pts

- reviews CANNOT be deleted, only pushed down – solicit good ones from satisfied pts
 - reply to negative reviews, and thank the positive
- Also: OTT (streaming ads), SEO, YouTube channels for operative and periop content

THE OFFICE

Staff must practice at the TOP OF THEIR LICENSE

- medical assistants are the cheapest staff – put data entry (Hx, allergies, meds) on them
- you cover HPI, exam, assessment, plan

30 pts/day model: 1 checking in, 1 checking out, 3 MAs – 1 chaperoning, 2 rooming & entering data

2-3 exam rooms plus a SEPARATE procedure room

Nail down staffing support IN YOUR CONTRACT – surgeons get fired for productivity the employer never staffed for

OFFICE-BASED PROCEDURES

An assistant is essential. Prone jackknife or left lateral Anoscopy, rigid proctoscopy, flex sig, I&D of perirectal abscess, banding, IRC, tag excision, thrombectomy

Medicare allowable: flex sig \$160.70 (w/ bx \$251.92, w/ snare \$263.39); rigid \$114.46; anoscopy \$95.07 (w/ bx \$235.68); IRC \$199.96; hemorrhoid banding \$257.43; excision skin tags \$276.97; thrombosed hemorrhoid \$185.16; I&D perirectal \$202.95

- global period 0 for scopes, 10 days for most office excisions, 90 for IRC

Anoscopes/proctoscopes sterilize easily – small autoclave + Cidex. FLEXIBLE scope reprocessing is a real burden

- disposable anoscopes and proctoscopes exist if staff time does not

ANCILLARY SERVICES

Categories: diagnostic (imaging), therapeutic (PT/OT, nutrition), custodial (hospice, SNF, urgent care)

Well-suited to colorectal: pelvic floor center with anorectal manometry, PT, ASC, pathology lab

Do NOT embark on a project SOLELY to increase revenue

Risks: losing money, STARK violation, damaging referral relationships, over-testing, lawsuits

Stark (Social Security Act §1877):

- bars a physician from referring designated health services (DHS) payable by Medicare to an entity he or an immediate family member has a financial relationship with
- bars the entity from billing for those referred services
- DHS: clinical lab, PT, OT, outpatient speech, radiology & imaging, radiation therapy, DME, parenteral/enteral nutrition, prosthetics/orthotics, home health, outpatient drugs, inpatient & outpatient hospital services
- does NOT ban self-referral to specialty hospitals, ASCs, or office-based surgical/imaging procedures

- compensation can never reflect the value or volume of referrals

- see a healthcare attorney BEFORE investing

Before adding a service: analyze what you currently send out, total investment incl. staff & training, expected reimbursement, and payer qualification requirements (e.g. CLIA)

Manometry example: machine ~\$20,000, reimbursement \$700, disposables \$115 → \$585 net per study. Fast ROI, and a referral hook for GI

Watch modifier –25 (separate E/M on the same day as a procedure) – many payers, incl. Medicaid, deny it

AMBULATORY SURGERY CENTER

For outpatient cases needing more time, equipment, or staff than the clinic

Restrictions by BMI and ASA class – but all but the sickest, largest pts fit

Revenue is FACILITY FEES per procedure → VOLUME is the driver of profitability

JCAHO accreditation, and state licensure often requires it

- insurers usually require state licensure to pay facility fees
- Medicare & Medicaid REQUIRE state licensure; commercial payers may be more flexible

Certificate of Need (CON) required in 36 states + DC, with wide variation

- no CON → contract for OFFICE-BASED procedures instead
- get counsel with specific CON experience in your state

Patient wins too – high-deductible plans, thousands of dollars' difference vs. the inpatient facility

CAREER

Committee work locally → medical executive committee, peer review → state and national appointments

- matters when a credentialing issue hits you or a partner
- keeps you current on tort reform, fee schedules, CON, Medicaid expansion

Transition out of clinical work may be by choice, or forced by illness, age, or loss of privileges

- diversify your skill set the way you diversify a retirement plan

Quadruple aim: improved pt experience + increased quality + cost efficiency + PROVIDER health and wellness