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Cecal Volvulus and Cecal Bascule: Management

Generally, the patient with cecal volvulus presents with distention and tenderness. The plain films are sometimes diagnostic, but often they can be confusing. In general, I have not found endoscopy to be particularly helpful — it is unlikely to undo a cecal torsion.

**Cecal Bascule:** Cecal bascule is a bit of a strange diagnosis. Many people have a big floppy cecum. Because this cecum is very mobile, we can do a right colectomy, and it is one of the easiest operations we do. With a bascule, the cecum can fold on itself. We see this frequently in the OR, but we can also see it radiographically. More straightforward cases are truly obstructed and symptomatic. But more difficult cases are those that present with vague abdominal pain, and on a CT scan, the radiologist suggests the possibility of a bascule. For cases with evidence of an obstruction but the symptoms resolve on their own, I think surgical intervention makes perfect sense. But management is a little less clear for cases presenting with nonspecific abdominal pain and a suggestion of a bascule on CT. If patients have recurring abdominal pain and you are convinced that that cecal bascule is the etiology, consider a laparoscopic right colectomy. I would probably tell the patient that I am unsure if cecal bascule is the source of their abdominal pain and that I cannot guarantee that their abdominal pain will get better with the procedure.

Sigmoid Volvulus: Management in High- and Low-Risk Patients

Endoscopy is a good procedure for undoing a sigmoid volvulus. For patients with torsion, the best option is to try to undo the torsion. We often use flexible sigmoidoscopy, but rigid proctoscopy could also be used.

**Management:** After undoing the torsion, the question is how best to manage these patients. The challenge is that these are generally elderly patients who often have many comorbidities. Sometimes they are institutionalized. The recurrence rate for sigmoid volvulus is very high, and we must use good judgment regarding whom we choose to operate on. The classic teaching is that, if you have undone a true sigmoid volvulus, then an elective operation makes sense.

**Low-Risk Patients:** Low-risk patients should definitely undergo an elective operation to prevent recurrence. They can be approached laparoscopically. But usually you can just make a small incision, and because the colon is so redundant and floppy, it will pop out. In this situation, you only need to resect the redundancy and perhaps do a functional end-to-end anastomosis.

**High-Risk Patients:** With elderly patients who are very sick and frail, I sometimes elect to follow them. If the problem recurs and they are constantly in the emergency room, I know that at some point, I must deal with the problem. For most cases, we first undo the torsion. If the colon is ischemic or gangrenous or if we are in an emergency setting, then a colostomy makes sense. But for most cases of chronic recurrence in which the colon is healthier, we perform an elective operation, generally a primary anastomosis. The management of every case is individualized. If we are dealing with an institutionalized person who perhaps has bowel control issues, the best option may be resection and an end colostomy to facilitate care for that patient. For these types of patients, having discussions with the family and the caregivers make a lot of sense.

**Perioperative Considerations:** I try to perform a bowel prep for all of our patients. We use a mechanical bowel prep, generally GoLYTELY®. We also use oral antibiotics. We have found that this decreases the incidence of surgical site infections. We also give perioperative IV antibiotics.
Diverticulitis: Disease Spectrum and Management Decisions

The American Society of Colon and Rectal Surgeons’ recommendations regarding diverticulitis seem to keep changing and getting vaguer. Diverticulosis and diverticulitis are relatively modern diseases. Some of the previous recommendations were from the 1970s, at which time we recommended that patients get an operation after their second attack. However, since then, population studies have demonstrated that this recommendation was probably too aggressive. Through a number of population studies, we have learned that patients are far more likely to need an emergency operation and a colostomy after the first attack than after the second. We have also learned that subsequent attacks tend to be similar to or not as severe as the previous attacks. Because of this, each patient is now individually assessed based on the frequency and severity of their attacks and their comorbidities. The spectrum of diverticulitis covers a huge range. For the patient with perforation or fecal peritonitis, 100% of surgeons would agree that an operation is required. However, no operation would be recommended for patients who have only a little twinge of left lower quadrant pain, some mild inflammation on CT, and prompt response to antibiotics. But between these two extremes of diverticulitis, there is a huge gray zone. Surgeons argue about the threshold in that gray area when operation is required. I tend to be conservative. Therefore, if patients are not relatively symptomatic and do not have severe recurrent bouts that are confirmed by a CT, I often will follow them.

Complicated Diverticulitis: The word “complicated” is often applied to diverticulitis. What does that mean? Again, diverticulitis has a huge spectrum of disease. Generally, complicated disease includes more obvious features, such as a colovesical or a colovaginal fistula, which almost always needs operative management. If a patient presents with a large abscess that you percutaneously drain, the likelihood they will eventually run into problems is also increased. For that patient, we will generally deal with the immediate infectious problem and then consider an elective operation.

Uncomplicated Diverticulitis: Uncomplicated diverticulitis tends to be applied to patients who present with thickening of the colon with maybe a dot or two of extraluminal air. In general, this is just inflammation and thickening of the colon that responds rapidly and completely to antibiotic therapy. These are the patients that we can probably watch more closely and only offer surgery for people who really have significant recurrence at a relatively early interval.

Diverticulitis: Margins, Rectal Mobilization, and Ureteral Stents

One of my sayings is, “If you are doing an operation for diverticulitis and it is not hard, you probably didn’t need to be there to begin with.” Therefore, know that this will be a difficult operation because you will be working with a very thick and inflamed portion of the colon, which often is in the sigmoid.

Margins: When faced with an elective operation for diverticular disease, surgeons must determine their proximal and distal margins. For the distal margin, we try to get down to the top of the true rectum because we know that recurrence rates tend to be higher if residual sigmoid colon is left behind. We do not want to resect all the diverticular disease because often these patients will have diverticulosis throughout their entire colon. Therefore, I usually choose my proximal margin by locating soft and pliable colon that I can work with, usually the descending colon. I do not particularly care if diverticulum is there or not.

Mobilizing the Rectum: Do you free the rectum up from the pelvic column? I almost always try to get into the retrorectal space. First I ensure that the ureter and gonadal vessels are out of harm’s way, and then I will probably take the superior rectal artery. I tend to mobilize the proximal rectum and score the peritoneum lateral to the rectum so that I do have a somewhat mobile rectum. I think this allows easy passage for the EEA™ stapler, as well. How do we know where the top of the rectum is? Sometimes, seeing the top of the rectum can be a challenge. Classically, it is where the taeniae coli coalesce, which can sometimes be hard to see during an operation, especially laparoscopically. Often, I will pass
an EEA sizer up to the rectum. If a 29 EEA sizer does not easily pass, then you probably have residual sigmoid colon. In this case, you want to go a little bit lower so that you can easily pass a 29 EEA stapler. **Ureteral Stents:** I do not routinely use a ureteral stent. They can be incredibly helpful when you have a really bad case with something like colovesical fistulas. If, on CT scan, the patient has a large inflammatory component invading the left sidewall, I will use the stents. I will also use them in cancers if there is any question of sidewall involvement. Therefore, I try to selectively use ureteral stents because, while they can be helpful, their use is associated with some morbidity.

**Perforated Diverticulitis: The Lavage Debate**

For patients with perforated diverticulitis, surgeons have been debating the value of laparoscopic lavage. When I have operated for perforated diverticulitis, a tremendous amount of contamination is usually present, which makes me anxious. In case studies from newer European studies published in the last 2 to 3 years, I think people “got away with it” (laparoscopic lavage), but its efficacy was not proven by any randomized prospective studies. Therefore, I have not performed the procedure because it does not make a lot of intellectual sense to me. **Ladies Trial:** Some European studies have looked into this technique, of which the major one is called the Ladies trial. This study had 2 arms. One arm looked at whether you should do a Hartmann versus a primary resection and some sort of proximal diversion. This arm is still open. The second arm was to do laparoscopic lavage for purulent type peritonitis (not for feculent peritonitis). This arm of the trial was closed because the study encountered a significant amount of additional morbidity associated with laparoscopic lavage. I believe the fundamental problem with lavage is that it is hard to predict if an actual macroscopic perforation is still present. In the study, a large number of patients needed to go back for additional procedures and some needed additional emergency surgery.

**Inflammatory Bowel Disease: Differentiating Crohn Disease vs Ulcerative Colitis**

Trying to differentiate between Crohn disease (CD) and ulcerative colitis (UC) can be easy in some cases and difficult in others. **Clinical Parameters:** The dilemma tends to come when distinguishing Crohn colitis from UC. Classically UC always begins at the dentate line and always involves the rectum. If any rectal sparing is present whatsoever, then that is CD, not UC. CD also has skip lesions: if you find some disease in the rectum, nothing in the sigmoid, and disease in the transverse colon, then that is CD. The presence of any kind of perianal disease, such as fistulas or abscesses, is strongly indicative of CD. Any small bowel involvement is also strongly indicative of CD. **Pathology:** We like to believe that, if we get a biopsy, our pathologists can definitively tell us whether we are looking at CD or UC. For example, CD is often associated with granulomas. But I have found that it is actually quite difficult for our pathologists to make that call, certainly on superficial biopsies alone. If a person comes to surgery and we are unsure or they are sick, we sometimes do a total colectomy and can submit the entire colon to the pathologist. This gives the pathologist the ability to look at the full thickness of the colon and help us distinguish CD versus UC. However, even in that situation, we do not always make a definitive decision, and there is a subset of patients who will be diagnosed with “indeterminate colitis.”
Ulcerative Colitis: Management

Most people with ulcerative colitis (UC) do not require surgery. For patients who do not need an operation for perforation or massive bleeding, we work intensively with our medical and gastrointestinal colleagues to treat them.

**Medical:** Traditional treatments for UC have been steroids for acute flares and 5-ASA compounds to maintain remission. However, the drugs used to treat UC have evolved. When the biologics, such as Remicade® (infliximab), came into being, they were mostly focused on CD, but they now have a role in treating UC. Virtually every day a new medication and a new drug seem to emerge. The implication for surgeons is that our patients are being treated with increasingly more medications, and they often present to us being sicker than the patients we saw before these treatments were available. In these cases, some sort of urgent surgery is usually required, almost always a multistage procedure. Almost all of our patients, especially those that fail medical management, are on a host of medications — usually on steroids and often on biologics and a variety of different medications.

**Surgery:** As people have UC for longer times, their risk for developing colorectal cancer increases. An indication for surgical intervention with UC is any evidence of dysplasia on biopsies or a dysplastic-associated lesion or mass that is confirmed by biopsy. Any biopsies that suggest dysplasia indicate the need for immediate surgery. Most UC patients do not get these types of problems unless they have had the disease for ≥10 years. But just because a patient has longstanding disease does not necessarily mean they should have an operation. Our patients with well-controlled longstanding disease get frequent surveillance colonoscopies, during which any obvious lesions are biopsied and random biopsies are taken throughout the colon looking for evidence of dysplasia. If they do not have dysplasia, then we just continue to follow them.

**Effect of Biologics:** A few years ago, 1 or 2 published articles indicated that the use of Remicade had decreased the risk of needing operative intervention for either obstruction fistulae or concern for cancer. Is that really true? I think immune-modulating drugs have done a good job of controlling the disease. But, if UC remains uncontrolled with these medications, then patients with very severe disease tend to present to the surgeon. It is relatively rare, now, to operate on people with mild to moderate disease that is just not controlled by 5-ASA compounds. Patients with this mild to moderate disease seem to respond to the other medications and do well clinically. We seem to be operating on only the patients who have severe disease.

Crohn Disease: Surgical Considerations

When operating on patients with Crohn disease (CD), we must remember that this is not a curative procedure. Instead, it is a palliative procedure to relieve the immediate problem, which is often either a fistula or obstruction. Therefore, bowel preservation is a fundamental principle of operating on these patients. When I operate on patients with CD, there is usually a relatively inflamed area that is often associated with infection and a phlegmon. In these cases, stricturoplasty is not particularly helpful and resections are required. But if the patient has short segments of strictures without a significant inflammatory component, then a stricturoplasty is a great option to preserve bowel and limit the resections.

**Postop Fistulas:** Postoperative fistulas in these patients are very challenging. Although I’d like to blame the CD, most of these fistulas are run-of-the-mill anastomotic leaks. These fistulas are a bit more common in our patients with CD because they are often on steroids. When operating in the setting of infection, postop fistulas are not an uncommon scenario. Management of the fistula depends on the presentation. If the patient is well, their white count is normal, and they are stable, then I try to manage them conservatively. I might need to place a drain if there is any infection that is associated with an abscess. I utilize total parenteral nutrition (TPN) when necessary. Yet, I do not always use TPN: if the patient has a low-output fistula, then they can probably eat and I just monitor the fistula output and
conservatively manage the case. My hope is that, because the fistula is not associated with CD per se, it is probably more of an anastomotic leak (and a technical issue) that could close with time.

Hemorrhoids

The medical management of hemorrhoids has not really changed. Generally, we try to get people to have healthy diets, not to strain too much on the commode, and to use a lot of high fiber to create bulky but soft and easily passable stools with plenty of fluid. We try to encourage our patients not to spend a lot of time on the commode, and not to strain while there.

**Classification:** We classify hemorrhoids according to a grade. Grade I hemorrhoids tend to be just internal hemorrhoids that do not prolapse. Usually if they are in your office, the patient is at least bleeding. Grade II hemorrhoids will prolapse but will spontaneously reduce on their own. Grade III hemorrhoids will prolapse and patients will describe pushing them back in. Grade IV hemorrhoids are prolapsed hemorrhoids that seem to be incarcerating and cannot go back in.

**Surgical Treatment:** A hemorrhoid’s grade determines its treatment plan. For grade I hemorrhoids, I think that rubber band ligation is probably the best option. It is easily done in the office. Even for grade II hemorrhoids with some mild prolapse, rubber band ligation can be effective. For larger hemorrhoids (grades III, IV), operative management becomes the main treatment. The fundamental problem with hemorrhoid surgery has been pain. The “old-fashioned” open operation is incredibly effective for this disease and results in very satisfied patients. However, the problem is that the surgery hurts. For this reason, we have come up with numerous alternatives: stapled hemorrhoidectomy, the Doppler technique the laser technique, and all the other variations. I have tried most of these alternate approaches. For example, I tried stapled hemorrhoidectomy for a number of years. Unfortunately, the recurrence rates are increased with this technique, and although complications are unlikely, the risk of severe complications is also increased. In addition, the staple line is associated with chronic pain. I think the next rendition is the Doppler-directed ligation of hemorrhoids. Although I have not performed this procedure, some of my friends have, and they seem satisfied with it. It seems logical that there could be recurrences associated with this technique. This technique involves a Doppler-directed ligation of the vessels. But first, a hemorrhoidopexy is performed to elevate the tissue into the anal canal.

Anal Fissures

A surgical procedure is available to treat anal fissures, but more and more medical options are being touted in the literature.

**Diagnosis:** Before treating an anal fissure, a diagnosis must be rendered. Virtually every patient who comes to our office with anal pain and some bleeding have been told that they have hemorrhoids, but this is usually not true. The classic presentation for an anal fissure is severe pain with a bowel movement. Often this is accompanied by a little bleeding, but usually it is just on the toilet paper. These cases do not have a lot of physical findings. You must be careful to examine them — the classic location of anal fissure is in the posterior midline position. I try to avoid digital rectal exams and anoscopy in people with obvious fissures because it is extremely painful for them.

**Treatment:** Once I have visualized the fissure, I review the treatment options. The fundamental principle is to decrease the trauma to the anal canal. First, we must make sure that the patient is on a high-fiber diet and that his/her bowel movements (BMs) are passing easily. Usually a very hard BM precipitated the fissure, so the BM’s consistency must be treated. Second, we must reduce the pressure in the anal canal. Often these patients have a very tight anal canal associated with hypertonicity, and different medical options are available to treat this. A topical nitroglycerin ointment has worked well for some patients, although it is associated with headaches. As a result, we have transitioned from
topical nitrates, and we tend to use topical nifedipine, but topical diltiazem reportedly works. More recently, the use of Botox® has been discussed to relax the anal canal and allow the fissure to heal. About 70% to 80% of people should improve or heal with these types of management. However, for those who fail and have chronic anal fissures, surgical treatment must be considered — a lateral internal sphincterotomy. Some people claim that this operation is associated with a high rate of incontinence, but my experience is that, when you really talk to people, incontinence is a relatively unusual clinical occurrence. Clinically significant incontinence is associated with how much muscle you cut, and we try to cut a limited amount of muscle. We usually say, “Cut about the height of the fissure itself.” This is usually ≤5 mm, so it is not a significant amount of muscle.

Colorectal Cancer: Screening

For the low-risk patient, the screening guidelines for colorectal cancer are relatively clear. Age is the primary component. Anybody aged >50 years probably needs some sort of screening, and various options are available. As colorectal surgeons and surgeons in general, our bias is toward colonoscopy. Regardless of the technique, everyone aged >50 years should be screened. **FIT:** The screening option that has probably been best studied is the fecal occult blood test (FOBT). The fecal immunochemical test (FIT) tests for blood, which is probably a little bit more sensitive and specific than FOBT. Fundamentally, FIT works the same way as FOBT. Our primary care doctors will frequently use FIT in the office, but it should not be done in the office via digital rectal exam. Instead, these cards need to be sent home and the stool taken by the patient in a passive bowel movement and returned to the office. **DNA Testing:** DNA testing of the stool has not quite made it to prime time. The test requires a large stool sample, is currently not covered by insurance, and has not definitively been shown to add much to the detection of early colorectal cancer. It is also expensive. I would not recommend it as a test at this time, although the American Cancer Society has approved it. **CT Colonography:** CT colonography requires a bowel prep. According to patients, the worst part of the entire procedure is the bowel prep. As a result, I think people are trying to figure out ways to use CT colonography without a bowel prep, but we are not quite to that point. The downside of the routine use of CT colonography is that a positive finding requires a regular colonoscopy. So I do not recommend it for routine use. Instead, a regular colonoscopy makes more sense in this situation because it is diagnostic and therapeutic, and it is associated with low morbidity. I use CT colonography when we have an incomplete colonoscopy. This usually happens secondary to diverticular disease or just a redundant colon, and I cannot complete the colonoscopy despite my best efforts. Then CT colonography is a great way to clear the rest of the colon. Another time you might use CT colonography is to clear the proximal colon when an obstructing or near-obstructing tumor is present.

Colorectal Cancer: Polyp Type vs Surveillance

**Case:** On screening colonoscopy, you find 2 polyps on the left side of the colon and completely remove them. They are pedunculated and basically have adenomatous changes. What do you tell the patient? **Recommendations:** First, tell the patient that they will be okay. These 2 polyps are what patients consider to be “benign.” But they are neoplastic growths, and we consider them to premalignant — they contribute to the adenoma-to-carcinoma sequence. Because these patients are “polyp-makers,” they require more frequent endoscopic surveillance. The general recommendation is that patients who have a clean colonoscopy (no polyps) at age 50 years will not need another colonoscopy for 10 years. But once a polyp is found, the recommendations change significantly. If the patient has 2 small 2-mm polyps,
perhaps recommending a 5-year (or so) surveillance interval would be good. That interval will need adjusting based on the number and size of polyps.

**Multiple Polyps:** Patients with multiple polyps (3 to 5) require more frequent surveillance. If the polyps are small and do not appear to be aggressive, then a 3-year surveillance interval would be good.

**Large Polyps:** More frequent surveillance is also required for patients with large polyps, flat polyps, villous adenomas, and/or polyps that we take out piecemeal. These polyps need following up because we (1) are looking for the development of additional polyps throughout the colon, and (2) want to ensure that polyp removal has been endoscopically successful. I bring these patients back within 6 months to reexamine the polypectomy site. When removing a large polyp, we must also tattoo it. If you inadvertently find cancer in the polyp, then the tattoo shows you exactly where it was located in the colon. If, on surveillance, the polyp is gone, a tattoo will allow you to locate and inspect the area.

For tattoos, we use a carbon product called Spot™.

**Sessile Serrated Polyps:** Sessile serrated polyps (SSPs) of the colon are very subtle, tend to be on the right-hand side of the colon, are often very flat, and seem to represent a different genetic pathway. SSPs are actually an epigenetic phenomenon (methylation of 1 of the promoters in the gene sequence). SSPs are concerning because they are hard to detect, tend to be quite flat, and seem to have a more rapid progression to cancer. Patients with “interval cancers” first underwent colonoscopy with no cancer detected, but cancer developed before their next scheduled colonoscopy. Among patients with interval cancer, an increased number seems to be from the SSP pathway. The thought is that these cancers grow a little faster than the traditional adenoma-to-carcinoma sequence. The surveillance program needs to be more aggressive for patients in whom SSPs were found and removed, especially if ≥1 cm. They do not necessarily need to undergo colonoscopy every year, but waiting 5 to 7 years is probably too long. SSPs can be challenging to remove. We often use a saline lift technique in which a little saline is injected beneath the polyp in an attempt to lift the polyp off the submucosa and grab it with a snare, thus reducing the risk of causing a full-thickness burn.

**Post-Polypectomy Syndrome**

When we treat polyps, we frequently use a snare polypectomy followed by cautery. This can result in a full-thickness injury, resulting fever and pain (postpolypectomy syndrome). Patients can present back, even that same night, with that type of discomfort and, on occasion, elevated white counts. We evaluate them thoroughly because only a few patients with postpolypectomy syndrome need an operation.

When you first evaluate them, you are obviously quite anxious because you have done the colonoscopy and you want to ensure that the patient does not have a full perforation. However, these injuries tend to be isolated, and if there is a perforation, they tend to be quite small. With close follow-up and some bowel rest, these injuries usually resolve on their own.

**Diagnosis:** The diagnostic workup depends on the clinical presentation. If the patient presents with only a little discomfort and your examination of them does not particularly worry you, they probably do not need further diagnostics. However, if the patient arrives in the emergency department with a great deal of pain and a slightly elevated white count, then I probably would order a CT. If a large amount of free air is seem, then a large hole is probably present and the patient needs an operation. I think that CT can help differentiate operative cases from those that might be followed up nonoperatively.

**Colorectal Cancer: Genetics and Lynch Syndrome**

Approximately 70% of colon cancers are considered purely sporadic, whereas 10% to 20% have some sort of familial component. A few patients have true genetic disorders that we can define, of which the 2 most common are Lynch syndrome (associated with defects in the mismatch repair genes) and familial
adenomatous polyposis (FAP; associated with the adenomatous polyposis coli [APC] gene).

Usually FAP is fairly obvious. Patients will have hundreds if not thousands of polyps, and it is rarely a diagnostic dilemma.

**Lynch Syndrome:** Historically, to diagnose Lynch syndrome, we have relied on family history, but we probably do not do a great a job at getting good family histories. Also, as families get smaller, patients are less likely to have a family history that would alert us to a genetic disorder. Therefore, in our practice, we have begun universal testing of all colon cancers that get diagnosed — we order immunohistochemistry (IHC) to look for the evidence or absence of mismatch repair genes. Our goal is to identify families with Lynch syndrome so that we can give them proper screening and perhaps alter their operations initially to a hysterectomy or subtotal colectomy. For every cancer we resect, we receive a synoptic report, and at the conclusion of that report we now routinely get the IHC test results that look for the different mismatch repair genes.

**Family Screening:** If the paternal leader of a family is tested and the IHC results say “consistent with Lynch syndrome,” how do you address the rest of the family? I believe that if you are going to embark on a genetic evaluation, it is best to do that in the context of a program. Here at the University of Virginia, we have two genetic counselors, and we send patients diagnosed with Lynch syndrome to these counselors. A diagnosis of Lynch syndrome does require an involved discussion with the patient. This is inherited in an autosomal dominant fashion, which means that the patient’s offspring and siblings have a 50% chance of also having the defect. Once they understand this risk, most families are willing to get their children or siblings tested. If we can identify the genetic defect in the patient who has cancer, then we can use that test to identify Lynch syndrome in their affected offspring. If the test is negative, then that family member would be placed into a routine surveillance program, but if they are positive, then we must adjust their screening recommendations.

**Colorectal Cancer: Surgery and Chemotherapy**

For colorectal cancer, the fundamental aspects of surgery have not changed significantly in recent years. The emphasis is on ensuring that we use good surgical technique, which involves high ligation of the primary feeding vessels. For right colon cancer, all lymph node drainage should be adequately removed by doing a high ligation of the ileocolic vessel just off the superior mesenteric artery. If needed, do a high ligation of the middle colic vessel. For left-sided disease, especially sigmoid cancers, the inferior mesenteric artery should be ligated as close to the aorta as possible.

**Lymph Nodes:** Surrogates are being used to look at lymph node counts. Now the goal is to get 12 lymph nodes. We must make sure that we are not just saying the words, but that we are actually doing it in action.

**Mesocolic Excision:** A developing concept that has not been fully accepted is that of complete mesocolic excision (mesocolon removed without any violations of peritoneal fascia). Some data suggest that this may be a “better technique,” but this has not yet been proven.

**Laparoscopic Surgery:** The debate over open versus laparoscopic surgery for colon cancer has been put to rest. The results of the COST trial clearly showed that the oncologic outcomes of laparoscopic and open surgery are equivalent and that the laparoscopic approach has certain advantages.

**Chemotherapy:** For colon cancer, chemotherapy historically consisted of 5-fluorouracil (5FU) and leucovorin. Now, a combination of drugs known as FLOFOX is routinely used (5FU, leucovorin, oxaliplatin) and has been shown to be superior to traditional 5FU-leucovorin. A host of other directed therapies are used in the metastatic setting, such as Avastin® and Erbitux®. Because of the many recent advances in chemotherapy, surgeons must work closely with their oncologists.

**Chemo for Stage II:** About 20% of patients with stage II colon cancer will fail. Recent studies have not supported the use of routine chemotherapy for stage II. But not all stage II colon cancers are the same, so effort is underway to differentiate high-risk from low-risk stage II. Compared to T3 disease, patients with T4 disease have invasion of other organs, have an increased relapse rate, and are
considered high-risk patients. Lymph node count is another area of concentration. Relatively good data show that the recurrence rates are much higher with low lymph node counts, perhaps indicating the use of chemotherapy. Poorly differentiated tumors are also high-risk features. At our institution, oncologists now use some genetic sequencing to distinguish high-risk from low-risk tumors, but this approach is still cutting-edge.

**Neoadjuvant Therapy:** The use of neoadjuvant chemotherapy is just beginning to enter the management discussion for colon cancer. For locally advanced disease, preliminary data show that neoadjuvant therapy is associated with an increased likelihood of negative radial margins. For metastatic disease, neoadjuvant therapy is clearly indicated as long as the primary is not symptomatic. A more controversial topic is whether neoadjuvant therapy is indicated for patients with a tumor and presumed node-positive disease, but no evidence of metastatic disease. I think the answer to this dilemma is currently “no,” but that may change in the future.

**Rectal Cancer: Mesorectal Excisions**

Because the rectum is located in the bony pelvis, we believe that the rate of local rectal cancer recurrence has traditionally been higher than for colon cancer. As a result, we have looked at alternatives to surgery alone for managing rectal cancer. Over the course of 25 to 30 years, the use of neoadjuvant treatment has evolved with the goal being to either downstage tumors or to sterilize the margins so that we can do good surgical operations. Surgery includes a total mesorectal excision (MRE) with a goal of getting negative radial margins to decrease the local recurrence rates, which we have done. Local recurrence rates ranged as high as 50% in the 1970s but now are in the single digits with the combination of neoadjuvant chemoradiation for locally advanced disease followed by a good operation. **Mesorectal Excision:** The goal of MRE is to find that magic plane in the retrorectal space. We find those filmy avascular attachments, leave the fascia propria intact, and dissect in a circumferential way all the way down to the pelvic floor where the mesorectum ends. The specimen that we remove has some fatty tissue around it that has an envelope with the completely intact fascia propria and no violations of the fatty tissue or the mesorectum. That would be termed a “complete MRE.” To determine if laparoscopy is superior to open surgery for MRE, an extensive study was done with some of the best laparoscopic surgeons in the United States. A composite score was determined based on negative circumferential margins, negative distal margins, and a complete MRE. The results showed that the distal margin was almost always negative regardless of which approach was used. However, compared to open surgery, laparoscopy was associated with a higher rate of positive circumferential radial margin and a slightly higher rate of incomplete MREs. Therefore, laparoscopy, even in the hands of some of the best laparoscopic surgeons, could be slightly inferior to open surgery, based on that composite score. This finding was confirmed in the ALaCaRT study from Australia. So what does that mean? I do not really know yet. We do not know whether these minor breaches in surgical technique are associated with increased recurrence rates when you reorient and get back in the right plane. Some data suggest that these breaches might increase the recurrence rate, but we do not know the long-term outcome on those studies yet. Therefore, I think that you have to look at your own practice and decide what technique is best for you. I do not think you should feel bad about doing a good open total MRE — I think that arguably still remains the standard of care. At our institution, we are doing more of our surgeries robotically. I do think there are some limitations to laparoscopic surgery in the pelvis, but the robot with the wrist and arms seems to give you better angles. So I think that robotic surgery may be where the next evolution will be.
**Small Rectal Cancers**

Is local resection appropriate for small rectal cancers? This management question, like other management issues in rectal cancer, has been evolving over the last 20-plus years. When I was a resident, we were very enthusiastic about local excision of rectal cancers, and we would offer it to patients with T1 disease (superficial disease) or T2 disease, although we knew that patients with T2 disease needed additional therapy often in the form of chemoradiation therapy. Now we have come to the conclusion that, for T2 disease, the locoregional recurrence rates are just too high with local excision. We also previously believed that if cases recurred after local excision, we could salvage them with surgery. But several studies now show that, in cases of recurrence after local excision, the salvage rates are in the 50% range.

**Small T1 Cancers:** For T1 disease without any poor prognostic factors predicting lymph node involvement (not poorly differentiated in the major component, no lymphovascular invasion), the risk of lymph node positivity is <10%. As long as the tumor is small and amenable to techniques, then a local excision is acceptable for T1 disease.

**T2 Cancers:** For T2 disease, the locoregional recurrence rates are too high to consider local excision for curative intent. For most patients who are good surgical candidates, a total mesorectal excision is preferred. For patients who are poor surgical candidates, we can be more aggressive about local therapy because we are really providing palliative care rather than curative care.

**Parastomal Hernias**

Parastomal hernias are extremely common. Most of my patients with parastomal hernias are not terribly symptomatic, and we prefer to manage them using support systems (hernia belts), and we try not to operate on them, because the recurrence rates are high and it can be a difficult and challenging operation. However, some sort of surgical intervention is needed for that subset of patients with larger hernias, who become symptomatic, or who can no longer keep a bag on it. At one point in my young career, there was enthusiasm for relocating stomas. That never made a lot of sense to me. As it turns out, relocating the stoma does not make a lot of sense. The problem is that, if they had a big parastomal hernia the first time, some factors, probably contributed to that, and those factors often still exist after relocating the stoma.

**Mesh:** We now primarily use mesh techniques to try to fix parastomal hernias. People have described a couple of different techniques. There is the keyhole technique and the Sugarbaker technique is becoming increasingly more common and often has the best data. With the Sugarbaker technique, you essentially try to “reperitonealize” the colon by placing a piece of mesh and having the colon come underneath the mesh and then directly out the fascial hole. In our practice, we usually use a polypropylene mesh. I believe that the people who want to use a biologic mesh are concerned about the infectious complications. But I think when you are doing parastomal hernia, you should not be dividing the bowel, so I believe that the risk of infectious complications is low. Because biologic mesh tends to be extraordinarily expensive and it often stretches, we have not been particularly happy with that type of mesh.

**Enhanced Recovery After Surgery (ERAS) Protocols**

At the University of Virginia (UVA), we have developed a series of multimodality interventions for the perioperative care of patients. These interventions come under the umbrella term of “enhanced recovery after surgery (ERAS) protocols.” Our ERAS program has been highly effective. We have seen about a 2-day drop in our length of stay, regardless of whether the procedure was performed laparoscopically or open. In fact, our open operations done after implementing ERAS are going home faster than our
laparoscopic operations performed before ERAS. In addition, our complication rates have gone down since implementing ERAS — our surgical site infection rates have gone down, which we did not predict. We feared that ERAS would come at a cost of readmissions, but, in fact, they are probably lower than they were before ERAS. I know this all sounds too good to be true, but it has been very effective, more so than anything else I have ever done in practice.

**ERAS for Elderly:** Elderly patients are also treated via the ERAS protocols. However, you do have to be more aware of the elderly patients, especially when introducing a diet. Aspiration in the elderly population can be a fatal event. Therefore, when using ERAS protocols for elderly patients, you have to be more engaged than you are with younger, healthier patients.

**ERAS Components:** There are several major components to our ERAS program at UVA. We do not starve people anymore – we try to keep them in their physiologic state. We allow them to drink up to within a couple hours of surgery. They can eat until midnight or until they start their bowel prep. We do require a full bowel prep, including oral antibiotics plus IV antibiotics at the time of the operation. We do carbohydrate loading by using Gatorade® before surgery. One component that has made a major difference is that we limit narcotics by using a host of multimodality therapies. We actually use an opiate-only spinal epidural that lasts about 24 to 48 hours. We then give gabapentin and a host of other nonnarcotic medications. As a result, we have dramatically reduced our intraoperative and postoperative narcotic use. Another component that has made a major difference is the use of goal-directed therapy in the operating room. We greatly limit the amount of IV fluid that patients receive intraoperatively, and then postoperatively we administer 40 mL per hour. The total volume that patients receive postoperatively has dropped dramatically, from 4 to 5 L before ERAS to hundreds of mL after ERAS implementation. A third significant component has been our very intensive preoperative patient education program. Patients know exactly what they are supposed to do, and if they fall off the protocol, they will tell you because they are very engaged in the process. If I had to rank the 3 most important components of our ERAS program, I would say (1) patient education, (2) decreasing the amount of narcotics, and (3) decreased IV fluids.

**Gastric Cancer: Evaluation**

In the United States, most patients with gastric cancer present with more advanced disease than do patients in Asian countries. The combination of a well-performed CT scan, endoscopy with ultrasound as indicated, and a diagnostic laparoscopy are the cornerstones of evaluation.

**CT:** A well-performed CT scan is first needed to look for possible lymph node and solid tumor metastases, especially hepatic metastases.

**Endoscopy:** Next, endoscopy is critical for identifying the primary tumor’s location in the stomach, if the tumor can be seen. In the U.S., the incidence of gastroesophageal (GE) carcinoma continues to rise, while the incidence of traditional gastric cancer has been declining for the last 50 or more years. Therefore, endoscopy may entail an endoscopic ultrasound to give the endoscopist a sense of the tumor’s origin. Is the tumor’s epicenter in the stomach, the GE junction, or the esophagus? This information is important because therapy and the surgical approach can be tailored based on the primary tumor’s location.

**Laparoscopy:** Finally, a diagnostic peritoneal examination is important. At my institution, diagnostic laparoscopy is performed early in the patient’s evaluation because most U.S. patients present with T3 tumors or larger, or at least large T2 tumors. This means that the tumors are at least 2 to 4 cm in size, which means that these patients have a 40% to 60% chance of having peritoneal metastases, and many of those (10% to 15%) may not be seen on standard imaging. If patients present with malnutrition, weight loss >10%, an albumin <3.5, and swallowing difficulties, placing nutritional access should be considered at the time of diagnostic laparoscopy because the patient may be receiving chemotherapy or radiation before surgery.
Gastric vs Esophageal Cancer: Treatment

Cancer treatments differ based on whether the primary tumor is located in the esophagus versus the stomach. **Surgery:** If the primary tumor is located in the esophagus, an esophagectomy is required. The surgeon will consider a thoracic approach in which the esophagus is removed approximately 8 cm proximal to the primary. In cases of gastric cancer, a portion of the stomach may be resected. **Radiation:** One main treatment difference between gastric and esophageal primaries relates to the radiation fields that are delivered by the radiation oncologist. Both the thoracic lymph nodes and the gastric lymph nodes may be at risk, even if the primary tumor involves the distal esophagus. **Chemotherapy:** The chemotherapy regimens do not differ significantly for gastric and esophageal cancers. Today, it is not atypical to have patients with a near-complete response to preoperative therapy. The surgeon must realize that, before treatment, they must have a good understanding of the primary tumor’s location because after preoperative treatment, those indicators may be gone. What’s more, at-risk lymph nodes in the chest may not be evident after preoperative therapy. Decisions regarding the primary and at-risk lymph nodes must be made before preoperative therapy is administered. **Laparoscopy:** Laparoscopy can be performed prior to surgery. At our center, it is a stand-alone procedure and the importance is that the findings may upstage the patient to a stage 4 malignancy. Therefore, chemotherapy and radiation would not be administered in the preoperative setting because that patient is not a candidate for a resection unless it is performed in a palliative setting. **HIPEC:** The use of hyperthermic intraperitoneal chemotherapy (HIPEC) in gastric cancer is currently under investigation. Some early indicators show that HIPEC may be a useful approach for patients with early peritoneal disease and that it may actually help patients survive longer. **Neoadjuvant Therapy:** Currently in the U.S., neoadjuvant therapy is considered the first-line therapy for patients who have anything more than a T1 or maybe an early T2 lesion because the risk of lymph node metastasis progressively increases with the T stage. Therefore, patients with a symptomatic tumor that is evident and identified on endoscopy as a mass lesion in the distal esophagus will most likely receive preoperative therapy. Most of these mass lesions are adenocarcinomas, and often the patients are anemic from an ulcerating overlying mucosa. The signet ring indication is an adenocarcinoma subpopulation that has the worst prognosis.

Gastric & Esophageal Cancer: Partial Response to Neoadjuvant Therapy

Patients with gastric or esophageal cancer may respond to neoadjuvant chemotherapy to the degree that subsequent diagnostic studies do not show any evidence of disease. This phenomenon is being seen with increasing frequency. Although it is good that patients are getting outstanding responses to preoperative therapy, the subsequent management of these patients is debated. Because the associated lymph node metastasis rates are high, many centers recommend continuing to surgery for patients who are fit for these procedures. The lymph node metastasis rate is as high as 20% to 25% for a T1 or T2 lesion, and these rates continue to climb as the T stage increases. This situation is very similar to that found in patients with rectal cancer: patients can have complete responses to preoperative therapy with no evidence of the initial tumor. But for these patients, rectal cancer generally recurs in the lymph nodes in the surrounding drainage basin. Once the cancer recurs, regaining control of it can be very difficult. For patients with esophageal cancer, esophageal surgery is a difficult surgery that carries a lot of risk for the patient. Therefore, after a response to preoperative therapy, I think the decision to proceed to esophagectomy must be made on a patient-by-patient basis. Many very-high-risk patients with esophageal cancer and a complete response to preoperative therapy are watched. **Case:** A patient with esophageal cancer has a partial response to preoperative chemotherapy. What is your preferred approach to surgical treatment?
**Recommendations:** Thoracic surgeons perform most surgeries in the thoracic esophagus or above the gastroesophageal (GE) junction. For cases in which the primary tumor is found at the GE junction, general and thoracic surgeons tend to work together. At my institution, mobilization of the stomach is often carried out laparoscopically, and then the esophageal dissection can be performed either under video-assisted thoracoscopic surgery (VATS) or an open technique.

**Reconstruction:** When possible, we use jejunal conduits for esophageal reconstruction and have had great success using microvascular techniques. For the average gastric cancer case, the stomach is mobilized, the esophagus is transected above the GE junction, frozen section analysis is done to verify margins, and then reconstruction is done using a Roux-en-Y esophagojejunostomy technique. Generally, we still sew those anastomoses. Although many use staples, we find that the stricture rate is higher with staples, and I have had good luck with sewn anastomoses.

**Gastric Cancer: Cell Surface Receptors as Therapeutic Targets**

Gastric and gastroesophageal cancer are two of several malignancies in which the gene for human epidermal growth factor receptor 2 (HER2) is amplified and its protein is overexpressed. As a receptor for epidermal growth factor on the surface of the cancer cells, HER2 is present on a percentage of the primary tumors. This is important because its presence offers the opportunity to use trastuzumab, which is an antibody developed to block this receptor, essentially blocking the growth signals that come through the receptor. Therefore, this systemic therapy offers the possibility of improved responses postoperatively.

**Familial Inheritance:** A familial inheritance pattern is seen with the loss of the E-cadherin receptor, putting these patients at risk for development of gastric cancers. From a familial inheritance perspective, this is one of the only known risk factors for the development of gastric cancer, and those patients and family members under study have been offered gastrectomies as a prophylactic procedure. While this subgroup is only a small percentage of the total patient population who will develop gastric cancer, it certainly shows us that genetics and inheritance patterns that we see can be used for interventions in a disease that is almost universally lethal in patients who are diagnosed late.

**Pancreatic Cancer: Incidence and Pending Therapeutic Advances**

The mortality rates associate with lung, prostate, breast, and colon cancer are decreasing. But the anticipated mortality rate for pancreatic cancer (PaCa) is increasing. By 2020, PaCa will likely be the second leading cause of death from cancer in the United States (second to lung cancer). The reasons for this shift are (1) increasing age of the U.S. population, and (2) increasing influence of obesity on the incidence of PaCa. As the U.S. population ages and becomes more obese, it is projected that the incidence of PaCa will continue to increase. No effective therapies are currently available for PaCa. Therefore, most U.S. general surgeons will see more cases of PaCa in the next 15 to 20 years.

**Therapeutic Advances Ahead:** Early studies indicate the existence of 2 to 4 subtypes of PaCa. Several large studies have surveyed the genetic signature of numerous subtypes of primary PaCas, providing us with a “satellite view” of differing genetic signatures for various PaCas. These genetic patterns indicate that there are different drivers for these different subtypes. This means that therapy can be engineered to affect these different subtypes, potentially resulting in our progress toward improving survival. Right now, we are at the early stages for some kind of rational development of treatment. As surgeons, we are counting on the development of systemic therapies that will make more patients eligible to receive curative surgery and to benefit from the surgeries that they do receive with adjuvant or postoperative chemotherapy.
Pancreatic Cancer: Categories, Treatments, and Survival

When I discuss pancreatic cancer (PaCa), I describe 3 categories based on a continuum. (1) “Potentially resectable” describes a tumor that is small enough, is located in the pancreas away from adjacent blood vessels, and is without growth outside the pancreas such that the tumor and surrounding pancreas could be removed without leaving microscopic or macroscopic cancer behind. Surgical resection could be done with confidence that the cancer would not recur outside the pancreas in a rapid fashion. (2) The other end of the PaCa continuum is “locally advanced” cancer. In these cases, the tumor has left the pancreas, has grown around vital blood vessels, such as celiac axis and the superior mesenteric artery, and has occluded the superior mesenteric vein, which cannot be fixed. These patients are not candidates for a meaningful surgical resection. (3) “Borderline resectable” PaCa is the in-between category on the PaCa continuum. In the United States, recommendations are that most patients with borderline resectable PaCa receive neoadjuvant chemotherapy. The added use of neoadjuvant radiation therapy is being studied, but we do not currently have sufficient data to say that radiation therapy is necessary. 

Locally Advanced PaCa: Patients with locally advanced PaCa receive systemic chemotherapy, but surgical resection is not generally performed. With advances in chemotherapy and surgical techniques, some patients can be offered surgery, but this is a very select group. In general, patients with locally advanced PaCa are not surgical candidates.

Borderline Resectable PaCa: Most patients with borderline resectable PaCa should receive neoadjuvant chemotherapy. The chemotherapy regimen shows some variation between different cancer centers. Two main regimens were designed for postoperative use: FOLFIRINOX and gemcitabine plus Abraxane®. Many patients who are younger, fit, and have minimal comorbidities are offered FOLFIRINOX (a combination of 5-fluorouracil, oxaliplatin, and irinotecan). This regimen can have significant toxicity, and it requires a lot of patient oversight and monitoring. Patients who are older and/or not so fit might be offered gemcitabine and Abraxane. Abraxane is a taxane that is an albumin conjugate and has a lower risk profile than FOLFIRINOX. Again, these two regimes were developed for postoperative use, not for preoperative use, so, we are still learning about how to use them in the preoperative setting.

Our goal is to use preoperative chemotherapy that helps the patient and leaves them in good enough condition to perform surgery on them. However, about 3% of patients undergoing preoperative chemotherapy have complications from systemic therapy that prevent them from undergoing surgery. Most preoperative chemotherapy regimens have a 4-month duration.

Survival: For potentially resectable and borderline resectable PaCas, the median survival is ≥30 months. The actual survival (not actuarial survival) rate is 25%. One of the big drivers for survival in resectable patients is whether they receive adjuvant chemotherapy within 8 weeks after surgery.

Pancreatic Cancer: Venous and Arterial Resections

The resection possibilities are extending in pancreatic cancer. For example, portal veins are being resected, as are other structures around the head of the pancreas, in an attempt to get an R0 resection. Some of the original literature from the 1970s and 1980s stated that doing vascular resections at the time of a Whipple procedure carried too much morbidity and mortality to provide any cancer benefit to the patients. However today, the perioperative morbidity, blood transfusions, etc, associated with vascular resections for pancreatic cancer can be within the same range as that for patients who do not receive vascular resections.

Venous Resections: Today, almost all vascular resections are portal vein resections, meaning that the primary tumor in the pancreas is abutting or invading the superior mesenteric vein as it passes underneath the neck of the pancreas. This requires some form of vascular resection and reconstruction using an autologous vein or primary reconstruction. This can be performed safely. Venous involvement is not an indicator of a particularly virulent cancer — long-term survival can be achieved in these cases.
Therefore, venous involvement does not preclude a patient from being a surgical candidate. The main technical caveat to venous resections is that the dissection must be carried out so that the specimen is attached to the vein and is completely dissected from the retroperitoneum so that the proximal and distal vein segments are free for clamp placement. We use systemic heparin to anticoagulate the patient, place vascular clamps, resect the vein, and then reconstruct it. The general idea is that we cut the stomach, bile duct, intestine, and pancreas; we perform a retroperitoneal dissection to free it off the retroperitoneum; we cut the vein and remove the specimen; and then we reconstruct the vein. This approach is very safe and does not add undo risk to the patient. Surgeons can get into trouble when they try to dissect the vein from the retroperitoneum when they do not have control of the proximal and distal portions of the vein. This can potentially cause significant blood loss and risk to the patient.

**Arterial Resection:** Arteries have abundant nerves around them that are often a conduit for the tumor to spread outside the pancreas. Pancreatic cancers are very neurotropic. From a biologic perspective, we do not know the details of this, but it appears that pancreatic cancers are at least attracted to or grow along nerves. Often the nerve involvement is around the superior mesenteric artery (SMA), which is an indicator of a different biology as opposed to spread around a vein, which is less of an indicator and more of an anatomic proximity problem. No current data support arterial resections for pancreatic cancer. Circumferential encasement of the SMA is currently an indicator for deeming a pancreatic cancer as locally advanced nonresectable.

**Pancreatic Cancer: Postop Pancreatic Fistulas, Delayed Gastric Emptying**

A postoperative complication of pancreatic surgery is pancreatic fistulas. Is there still an argument about whether to use small bowel versus stomach for the anastomosis when reconstructing after a pancreaticoduodenectomy? Small single-surgeon reports can be found describing low leak rates from pancreatic fistulas after pancreaticogastrostomy. But at my institution, this is not our current practice. I try not to excessively mobilize the pancreas before the anastomosis. One of the dangers for pancreatic anastomosis is that over-mobilization of the transected head reduces the arterial inflow, making that edge increasingly more ischemic. Therefore, to mobilize up to the stomach risks taking too much arterial inflow. In our practice, we do an end-to-side anastomosis — physically sewing the pancreatic duct to the jejunal mucosa, performing a full-thickness small enterotomy. This procedure is done in layered manner to reduce the chances for disruption of the anastomosis.

**Pylorus Preservation:** Delayed gastric emptying is a common complication after a Whipple procedure, occurring in 25% to 40% of cases. In my experience, patients who undergo pylorus preservation have a much more normal diet at 6 months to 1 year postoperatively. However, in patients who undergo radiation therapy, I do not use pylorus preservation because the pylorus is in the radiation field, resulting in potential ulcers and poor gastric emptying. Instead, I use pylorus preservation for relatively young patients for whom I have concerns about later nutrition and who have an intraductal papillary mucinous neoplasm, a small ampullary cancer or an “other” neuroendocrine tumor.

**Pancreatic Cancer: Variations in Hepatic Arterial Anatomy**

In preparation for performing a pancreatic cancer resection, the surgeon should understand variants of normal hepatic arterial anatomy. I see a great deal of risk in the dissection in the porta hepatis when the surgeon is taking the gastroduodenal artery and also dividing the common duct.

**Right Hepatic Artery:** Most of the dominant flow to the bile duct system is through the right side of the hepatic arterial flow. “Normal” hepatic arterial anatomy appears in about 55% of patients, so about 45% of patients have aberrant inflow to the liver. The most common variant is a replaced right hepatic artery, which can derive from the superior mesenteric artery (SMA), can derive from the aorta, and can derive
as a low branch from the proper hepatic artery (it swings down toward the duodenum and then back up along the common duct). If that hepatic artery variant is transected and a biliary anastomosis is performed, that anastomosis will likely stricture or not heal and leak. By using a high-definition CT scan preoperatively, a surgeon should have very good working knowledge of the kind of hepatic artery variant they will be encountering.

**Celiac Artery Stenosis:** The other variant that can cause trouble is stenosis at the origin of the common hepatic artery — a celiac artery stenosis. This variant may not be recognized until the gastroduodenal artery is divided because the flow to the liver is retrograde and deriving from the SMA and not from the common hepatic artery. As a result, when the gastroduodenal artery is divided, there is no pulse. This can be remedied by dividing the arcuate ligament, which usually lies across the origin of the common hepatic. Before entering the operating room, the surgeon must be aware of these variants and how to solve the potential problems they pose.

**Imaging Variants:** The preoperative CT scan is extremely important for identifying these variants. The traditional view is like the bread slice view in which you are looking from the patient’s feet up to the head. We now commonly use the reconstruction views that are more like an anteroposterior view you would see in the operating room. The reconstruction views are very helpful because they provide a map that is in the same orientation as what you would see in the operating room.

**Pancreatic Cysts: Diagnosis and Management**

A Japanese autopsy study looked at the pancreas of people who died from a wide variety of causes and found that 25% of individuals aged >65 years had pancreatic cysts at the time of their death. In the United States, our prevalent use of CT scans has lead to the identification of pancreatic cysts in many different patients, even asymptomatic patients. The ultimate question is whether the patient needs surgery for the cyst because of the cancer potential. We use an algorithmic approach to determine if patients need surgery, surveillance, or nothing. Simple cysts are usually serous cysts (contain a serous fluid) and require minimal, if any, follow-up. However, mucinous cysts have malignant potential because the mucin usually comes from a metaplastic change of the ductal cells lining that cyst. These mucinous cysts require further workup to determine if they represent an intraductal papillary mucinous neoplasm (IPMN) or a mucinous cystic neoplasm (MCN).

**Surgical Criteria:** Classification criteria have been published to help surgeons know which patients need surgery. These criteria usually involve identification of structural changes around the cyst, size (≥3 cm indicates high-risk lesion), presence of a nodule, architectural changes, obstruction of the main duct, and presence of alarm symptoms (jaundice or pain, dysphagia, or early satiety). Pancreatic cysts that are accompanied by alarm symptoms and/or other suspicious characteristics need further workup. For example, patients might undergo endoscopic US examination of the pancreas with fine-needle aspiration of a solid nodule or a mass seen in relation to the cyst. The difficult cysts are the ones found in asymptomatic patients. To clarify whether the patient needs further intervention, I aspirate the cyst to determine if it is a mucinous cyst or a serous cyst. Serous lesions need no further workup.

**Other Tests:** Other tests are available for pancreatic cysts. Cytology is not always helpful, but it can be useful if the cytopathologist sees cells that have dysplasia or changes that look like progression toward malignancy. Some pathology labs are using more molecular studies that evaluate mutations in pancreatic cysts, but these tests are not yet useful.
Mucinous Pancreatic Cysts: Management

Mucinous pancreatic cysts require further workup to determine if they represent an intraductal papillary mucinous neoplasm (IPMN) or a mucinous cystic neoplasm (MCN). Patient age and gender can help differentiate these two lesions. A woman in her 50s with a solitary mucinous lesion most likely has an MCN. A woman in her 70s who has several small mucinous lesions most likely has an IPMN of the side branch variety. The woman with the MCN is likely to need surgery because she will probably survive a long time, giving that lesion time to develop into malignancy. IPMNs are a marker for malignancy risk of the pancreas, but it is not certain that cancer will develop.

**IPMN:** Main duct and branch duct IPMNs differ anatomically. The main duct of the pancreas has side branches coming off of it. Smaller cysts that occur in those side branch systems are generally believed to carry less malignancy risk than a main duct system IPMN, where changes can occur all up and down that main duct. Generally, when the main duct is ≥1.5 cm, patients who are suitable candidates should undergo surgical resection. Deciding which operation to perform can be tricky. If the whole duct is affected, doing a total pancreatectomy is a tough operation for patients because of the diabetes consequences.

**Surgery:** The tissue resected in cases of main duct IPMNs depends on the portion of the pancreas (head vs tail) involved. We try to preserve pancreas parenchyma because of the diabetes side effects. For example, a patient undergoes surgery for a 1-cm dilation of the main duct in the head of the pancreas. We inspect the main duct in the surgical neck. It might be dilated because it is obstructed or because it has mucin in it. This can be difficult to differentiate. Therefore, we determine where the main duct has a normal caliber and then divide the pancreas there. A frozen section from that site is sent to pathology. We resect more pancreas ONLY if carcinoma is seen at the margin. Because we do not further resection if any other cell type appears, we recognize that other areas in the tail could eventually develop a new malignancy. Therefore, these patients need to be followed up. But it is important to try to preserve as much insulin-producing capacity as possible within the bounds of an effective cancer operation.

**Follow-Up:** For patients undergoing surveillance, their first two follow-up visits are at 6 months and 1 year, at which time they get repeat CT scans. We look for a stable cyst that is not rapidly progressing. If we see a ≥50% increase in the cyst’s size, then more investigations or surgery might be triggered. If by 1 year we do not see any big changes or anything worrisome, then patients will return in another year, and we will convert their imaging study to an MRI to reduce the radiation exposure.

Carcinoids of GI Tract: Types, Presentation, and Management

Sometime, when a patient undergoes endoscopy for vague upper gastrointestinal (GI) symptoms, such as dyspepsia, the endoscopist identifies a carcinoid tumor, perhaps in the duodenal bulb or the antrum of the stomach. For these patients, the questions become whether we are looking at gastric carcinoid and what at type of gastric carcinoid are we looking?

**Type 1 Gastric Carcinoids:** Types 1 and 2 are driven by gastrin production. Type 1 carcinoids are due to atrophic gastritis (autoimmune or other causes), resulting in reduced acid production, which in turn increases gastrin production. Excess gastrin stimulates these enterochromaffin-like (ECL) formations, which can then become carcinoid tumors. These are often treated via endoscopy rather than surgery. These patients need to be monitored.

**Gastrin Levels:** For types 1 and 2 carcinoids, the gastrin levels must be checked. Even for patients on proton pump inhibitors (PPIs), those with Type 1 gastric carcinoids have gastrin levels ≥200 pg/mL, which is well above normal. If we suspect a patient has a Type 1 gastric carcinoid, we ask them to discontinue their PPIs for 7 or 8 days before we repeat the evaluation of their gastrin levels.

**Type 2 Gastric Carcinoids:** Type 2 gastric carcinoids are caused by a gastrinoma, and patients present with symptoms of Zollinger-Ellison syndrome. The gastrinoma directly produces excess gastrin levels
that are almost always >1000 pg/mL even when we cannot see the tumor. These gastrinomas are often found endoscopically in the wall of the first or second portion of the duodenum. We often use octreotide scans in which radiolabeled octreotide binds to surface receptors on gastrinomas as well as other endocrine tumors. This test allows us to identify the primary tumor as well as metastatic lymph nodes. These patients usually have a lot of dyspepsia related to the excessive gastrin production. Generally, they cannot discontinue their PPIs because they are on a maximum dose and cannot tolerate coming off. Regardless, the gastrin level is usually so high that it is diagnostic.

**Type 3 Gastric Carcinoids:** These are sporadic carcinoid tumors usually found in the first portion of the duodenum. These tumors behave like any other epithelial malignancy: they are often associated with lymph node and hepatic metastases. We often use octreotide scans to stage these patients and determine if they have metastatic lymph nodes. To manage these, we resect the tumor and do a formal resection of regional lymph nodes. I had a patient with one of these tumors located in the first portion of the duodenum. To treat it, we performed an antrectomy (resecting the first portion of the duodenum) and then we did a formal dissection of the regional lymph nodes.

**Carcinoids of GI Tract: Small Bowel Obstruction**

Carcinoids can cause symptoms of small bowel obstruction. When taken to the OR, the surgeon finds a small bowel fixed to a mesenteric mass located within the mesentery that drains the ileum (upstream from the distal ileum, cecum, and appendix). These cases are almost always a small bowel carcinoid tumor with metastases to the drainage basin that drains along the ileocolic artery and vein up toward the pancreas. Obstruction of the small bowel is what commonly kills patients with carcinoid.

**Carcinoids & Obstruction:** Sometimes at exploration, we might identify a couple areas in the liver that contain small-volume liver metastases from carcinoids. However, these patients are more likely to die of the mass in the mesentery. Carcinoids make serotonin, and a great deal of fibrosis develops around these tumors, which pulls the small bowel into them. As the small bowel becomes trapped in the fibrosis, the veins that drain that section of small bowel become obstructed. Therefore, patients die because either they cannot use the gut to eat or they develop a bowel obstruction that cannot be relieved.

**Bowel Resection:** It is important to clear the lymph nodes whenever possible in the setting of your operation. When you encounter one of these with big tumor-involved lymph nodes, lymph node resection must be balanced with leaving the patient with enough small bowel to be nutritionally functional. I operated on a woman who had this last week, and one of the first things we did was measure how much small bowel we would leave in the patient after the resection. These small bowel resections almost always remove the ileocecal valve because we take the mesentery that drains along that ileocolic artery and vein. Therefore, at least 150 cm of small bowel needs to remain in the patient. I prefer >200 cm of small bowel at that anastomosis because, even if that patient has symptoms, we can usually control those symptoms with cholestyramine or fiber and Imodium®. Even in the setting of a patient with minimal metastases, we resect the primary tumor and the regional lymph nodes to clear the small bowel region of these tumors. Historical data mainly from Sweden, although not perfect, suggest that survival is prolonged when regional lymph nodes that have cancer in them are resected.

**Carcinoid Syndrome**

Carcinoid syndrome tends to be present in patients with advanced carcinoids. Our control of these symptoms is improved when treating with somatostatin analogs in the depot form, which can be given once a month. As a surgeon, it is important that your op notes contain data about the anatomy and the length of the small bowel remaining inside the patient. Down the road as the patient progresses, they will develop diarrhea and loose bowel movements. It can get confusing differentiating between
short gut symptoms from the small bowel resection or carcinoid syndrome in these patients. Therefore, data in your op notes can be helpful to them.

**Carcinoid Syndrome:** As surgeons, we are taught that patients without liver metastases are unlikely to get carcinoid syndrome. The one exception to this would be when mesenteric lymph nodes actually invade into the retroperitoneum. Remember, the mesentery sits on top of the retroperitoneum, so the tumors will invade and the serotonin can leach into the veins in the retroperitoneum and enter the systemic circulation. This scenario is something to consider when a patient has bulky disease, which is not common.

**Gastrointestinal Stromal Tumors**

Gastrointestinal stromal tumors (GISTs) originate from the interstitial cells of Cajal, which are neural pacemaker cells found throughout the GI tract. GISTs can occur anywhere along the GI tract. Gastric GISTs are the most common. Like carcinoids, the genetic mutations of GISTs are unique to tumors found at different anatomic locations along the GI tract. The c-kit gene encodes for a surface receptor on cancer cells, and mutations in this gene are driver mutations that contribute to tumor progression and metastasis. Surgeons have been involved from the beginning in the development and deployment of targeted therapeutics for GISTs. This has led to the rapid use of medications like imatinib, which is a small molecule that blocks this receptor in patients who have undergone surgical resection of GISTs. One of the first trials that was sponsored by the American College of Surgeons was a trial in which imatinib was used postoperatively. The results demonstrated that imatinib improved patient outcomes. Since then, imatinib has been evaluated as a preoperative therapy to shrink large GISTs and improve organ preservation. For example, a patient presented with a large GIST located in the second portion of the duodenum. I did not want to perform a Whipple procedure or some large procedure for that tumor because it was on the lateral surface. I believed that we could preserve her duodenum if the tumor was smaller. So, we gave her imatinib before surgery and, as predicted, that tumor responded and shrank. I was then able to resect a much smaller portion of her duodenum than originally needed.

**Unique Tumors:** GISTs are a unique subset of tumors: they are basically sarcomas of the GI tract, and most surgical approaches involve complete resection. They are unlike pancreatic or esophageal cancers or other epithelial cancers for which cell spreading along the organ is a critical component. From a surgeon’s perspective, GISTs lend themselves to laparoscopic resection.

**Surgical Margins:** On surgical resection of GISTs, negative margins are generally adequate. For the case mentioned above, we excised a lateral portion of the duodenum with about a 6- or 7-mm margin outside the tumor’s edge. We submitted that to pathology (no frozen section) to evaluate for adequate margins. No lymph nodes were removed.

**Postop Chemotherapy:** The postoperative use of imatinib (Gleevec®) or other targeted drugs is generally driven by the original tumor’s risk of recurrence. For example, a 2- to 4-cm gastric GIST would first be resected, unless it is in a poor anatomic position for resection. If the pathologist reports that this is a 2.5-cm GIST with <5 mitoses per high power field and clear margins, then the risk of recurrence is low, and adjuvant imatinib is probably not beneficial. GISTs with an increased risk of recurrence are generally >5 cm (depending on anatomic location), located in the small intestine rather than the stomach, and have an increased mitotic rate.

**Pancreatic Neuroendocrine Tumors**

Like the pancreatic cyst, we also see a lot of patients who have CT scans that show small pancreatic neuroendocrine tumors (PNETs). PNETs are the second most common solid tumor in the pancreas after adenocarcinoma. PNETs are either functional tumors, such as insulinoma, or nonfunctional tumors.
(do not secrete an active hormone). Of all PNETs seen, 85% are nonfunctional. These tumors have malignant potential. Like pancreatic cysts, PNETs are dividing abnormally, meaning that their mitotic rate is higher than that of normal cells. They can metastasize to lymph nodes and the liver, the relative risk of which is generally driven by the tumor’s size and whether it is a well-differentiated tumor.

**Management:** Surgical resection is recommended for any PNET >2 cm, even if it is well differentiated. These tumors are sometimes more amenable to enucleation because they are often located in peripheral in the glands rather than adjacent to the duct. According to the published literature, the risk of pancreatic fistula after enucleation ranges from 20% to 40%. Therefore, drains should be placed.

**Follow-Up:** PNETs have a blood marker that we can use for follow-up. We use a chemical called chromogranin-A, which is a very sensitive, although sometimes not very specific, marker of PNETs. Octreotide scans also will be positive. This is a nuclear medicine study that can be linked to a CT scan, which is very useful, giving us functional and structural data at the same time.

**GERD: Diagnostic Approach**

The diagnostic approach to gastroesophageal reflux disease (GERD) is a debated topic. When a patient presents with reflux, you want to work up the patient from both an anatomic and physiologic standpoint. My workup for a patient with reflux involves four major studies: upper endoscopy, pH monitoring, barium swallow, and esophageal manometry.

**Upper Endoscopy:** Upper endoscopy helps visualize the anatomy within the stomach and esophagus. It can hint at hiatal hernias, although this is not a good test for identifying these hernias. Upper endoscopy will allow you to see evidence of esophagitis, strictures from chronic reflux, and any other pathology that might be present. If you see evidence of esophagitis, especially a grade 2 esophagitis or above, or if you see evidence of Barrett esophagus during upper endoscopy, then it precludes having to do pH monitoring; such as 24-hour pH monitoring. Otherwise pH monitoring is a necessary test. Now that the miniature pH capsules are available, 48-hour pH monitoring is possible.

**Barium Swallow:** A barium swallow provides us with anatomic information. The barium gives a very good view of the structure and helps us see the presence of a hiatal hernia, which can be a challenge with repair.

**Esophageal Manometry:** The final diagnostic test is esophageal manometry, which provides a physiologic evaluation. In my institution, this is one of the hardest tests to get. People sometimes get frustrated trying to go through this process. Nonetheless, this test is very important because it provides an idea of esophageal contractility and peristalsis. Manometry helps rule out additional concomitant problems, such as achalasia or nutcracker esophagus, which may affect whether the surgeon does a full or partial wrap. If, on manometry, you find that the relaxation of a patient’s sphincter is not good, you might consider doing a partial wrap rather than a full wrap.

**Impedance Plethysmography:** Impedance plethysmography (IPG) is a test that uses a fluid bolus to both evaluate peristalsis and give a very dynamic overview of esophageal function. It also gives information about reflux that may not be pH-related. IPG is generally used at academic centers focusing on the esophagus. While IPG can give you an idea of the length and location of the lower esophageal sphincter, the barium swallow remains superior for visualizing a hiatal hernia. Therefore, even if performing IPG, I recommend that a barium swallow also be performed.

**Test Staging:** When I work up a reflux case, my diagnostic approach is to perform a barium swallow and upper endoscopy first, followed by manometry and then pH monitoring (if no evidence of esophagitis). I usually try to order manometry early in the process because it will be one of the later results to come back.
Esophagitis: Classification and Managing Grade 2 Disease

The Los Angeles classification of GERD grades the severity of reflux esophagitis using grades A thru D. Grade A esophagitis has $\geq 1$ mucosal breaks that are $<5$ mm of maximal length. Grade B esophagitis has $\geq 1$ mucosal breaks that are $>5$ mm but without continuity across two mucosal folds. Grade C esophagitis has mucosal breaks that are continuous between $\geq 2$ folds, but it involves $<75\%$ of the circumference of the esophagus. Grade D is like grade C except that it involves $\geq 75\%$ of the circumference. Barrett esophagus does not play any role in this classification system. Instead, it is all about the degree of esophagitis. Patients with grades B, C, or D esophagitis generally have acid reflux, so further testing with pH manometry is not needed.

Case: The Savary-Miller grading system is another commonly used system to rank the severity of esophagitis. A patient presents with grade 2 esophagitis (multiple erosions affecting multiple folds; erosions may be confluent) and the diagnostic studies indicate reflux. How do you manage these cases?

Recommendations: Basically, patients who do the best with reflux treatment are those who have typical heartburn-type symptoms of GERD. They generally respond to PPIs, and they either have evidence of esophagitis or have abnormal pH testing in the esophagus. In my practice, I like to give a trial of PPIs to patients who present with esophagitis. I also ensure that we’re treating the esophagitis or at least that it is improving. If the patient continues to have issues, or if typical reflux symptoms continue, I move ahead and go for a wrap.

Partial vs Full Fundoplication: Indications, Outcomes

Today, patients with GERD often present to the surgeon with continuing reflux symptoms after having first gone to a gastroenterologist for initial evaluation and two rounds of PPI trial treatment. For patients who fail 2 trials of PPIs, a Nissen fundoplication (NF) is generally indicated. NF is also indicated for patients who have evidence of esophagitis, have had a stricture, or do not want to undergo prolonged PPI treatment.

Partial vs Full Wrap: NF is a “full wrap” (360° around the fundus) antireflux procedure. While NF is still a very durable and popular procedure, surgeons have become more selective in using partial wraps, especially when evidence of esophageal motility issues is seen on manometry or impedance plethysmography. Therefore, when poor esophageal peristalsis or weak contractions are seen, then surgeons are tailoring their procedures more by doing a partial wrap, such as a Toupet fundoplication (270° dorsal wrap) or a Thal fundoplication (270° anterior wrap). If the patient has normal esophageal motility, I prefer doing NF. Comparing partial wraps versus full wraps (NF), the two procedures have similar patient satisfaction scores, degree of esophagitis, postoperative use of PPIs, and reoperation rates. However, the two types of procedures differ in that early postoperative dysphagia is greater with NF, and reflux control is not quite as good for the Toupet procedure as for NF.

Nissen Fundoplication: Technique

I learned how to do a Nissen fundoplication (NF) working with Dr. Nathaniel Soper. I think it is a nice technique in which all the short gastric vessels are taken down. In addition, this technique involves a “shoeshine” maneuver to ensure the adequate mobilization of the fundus of the stomach. The shoeshine maneuver helps position the wrap so that it’s not twisted, mimicking the grasp you have on a shoeshine cloth so that it does not twist. After the wrap, I place at least 3 sutures: two on the fundus (stomach-to-stomach) and then I include the middle one, usually taking a little bit of the esophageal muscle.
**Short Gastrics:** An important aspect of performing NF is taking down the short gastric vessels. Studies have demonstrated that the short gastric vessels should be taken down because you want full mobilization of the gastric fundus.

**Fundus:** Another important aspect of performing NF is to not mistake the cardia (the main body of the stomach) for the fundus. If the cardia is wrapped around the esophagus, this results in a “slipped Nissen.” The reason NF works so well is that you take advantage of the receptive relaxation of the fundus. When you eat, the fundus relaxes so you can take in a large meal. Therefore, when your wrap utilizes the fundus, it relaxes to help the food get in, but then it contracts and stays tight to stop reflux. If the cardia is used for the wrap, you will not have this tight wrap, resulting in dysphagia and other related problems. By first taking down the short gastric vessels, you mobilize the fundus and avoid grabbing a piece of the cardia for the wrap. As you take down all the short gastric vessels, you open up the angle of His, so that when you create the posterior window, you are high on the stomach and can really see to ensure that you grab the fundus as you bring it around.

**GERD: Alternative Devices and Techniques**

Several surgical devices allow us to reduce the signs and symptoms of GERD. One interesting device is a magnetic ring that is placed around the distal esophagus: it is designed to augment the lower esophageal sphincter (LES). The magnetic beads in the ring are Another method uses endoscopic suturing to perform endoscopic fundoplication. Yet another device, the Stretta® device, uses heater probes to thicken the esophageal muscle distally, which improves the barrier function and can reduce reflux. Many of these alternative techniques and devices have come and gone quickly as they demonstrate poor efficacy in treating reflux. I recommend reserving these less-invasive endoscopic techniques for individuals who do not have severe symptoms or signs of GERD. I recommend using our tried-and-true methods of partial or total fundoplication for individuals with more severe symptoms of disease.

**GERD: Atypical Symptoms and Short Esophagus Syndrome**

Some patients with GERD demonstrate atypical symptoms. Often, these types of patients will present to surgeons. The atypical symptoms include hoarseness and/or aspiration. They may develop pneumonias related to their persistently bad reflux. For these individuals, NF is sometimes not as effective as it is for patients with typical symptoms. Nonetheless, especially if you are at a high-volume center, patients with atypical symptoms are often referred.

**Short Esophagus:** In addition, the treatment of patients with chronic atypical GERD tends to be challenging because, with the very severe reflux, some individuals develop short esophagus. Chronic GERD involves recurring cycles of inflammation and healing, followed by subsequent fibrosis, which can lead to intrinsic shortening of the esophagus (short esophagus). For a while, the Collis procedure was a popular procedure used to lengthen the esophagus by creating a neo-esophagus by cutting out a wedge of the fundus. But now, many people believe that you can bring down enough esophagus if you do a really good dissection during NF. Indeed, this is one of the key steps: you want to bring at least 3 cm of the esophagus down into the abdomen so that it doesn’t pull back up and cause related issues. What’s more, the abdominal pressure helps with the sphincter when the esophagus is located in the abdomen. In reality, while you are dissecting, you sometimes must go into the chest and dissect out a lot of that esophagus to be able to mobilize it down into the abdomen. This is a challenge.
Barrett Esophagus: Biopsy Technique and Managing High-Grade Dysplasia Without Cancer

**Case 1:** As you perform upper endoscopy to evaluate a patient with reflux, you see what looks like a 4-cm segment of Barrett esophagus (BE) just above the gastroesophageal junction. What is your preferred technique for taking biopsies when you suspect BE? How do you manage BE with high-grade dysplasia?

**Biopsy:** You want to get a systematic four-quadrant biopsy and you want to march up at the level of the BE. Advance at least 1 cm and then get four-quadrant biopsies each time. If it’s the first time with BE, you can sometimes go 2 cm. The goal is to get good sampling because, with BE, high-grade dysplasia can occur. If high-grade dysplasia is present, then the incidence of occult cancer that you’ve missed with your biopsy can be very high.

**High-Grade Dysplasia:** In the past, we were taught that, because of the risk of occult cancer, an esophagectomy was needed if high-grade dysplasia was found. This is no longer true. Instead, several endoscopic techniques are effective at treating BE with high-grade dysplasia (without cancer). Radiofrequency ablation (RFA) is one such effective treatment. With this technique, you insert a balloon that has internal coils in 3-cm segments. You inflate the balloon and perform RFA. This is especially helpful for long-segment BE. The other main option is endoscopic mucosal resection (EMR), which is typically reserved for smaller lesions, about 2 cm in size. The principle of EMR is similar to performing polypectomies during colonoscopy during which polyps are sucked up and cauterized. With EMR, the BE region is sucked up and cauterized to excise it and prevent bleeding. With EMR, that mucosa is removed in the process, which removes the source of the BE. The issues with EMR are, if used to treat long segments, you can get skips in the treatment (EMR does about 2-cm segments at a time) and the risk of posttreatment strictures is elevated. However, with RFA, the dysplasia can be eradicated in about 90% of patients. Although continued surveillance and biopsy are needed, these procedures have transformed the treatment of high-grade dysplasia without cancer.

**Case 2:** On upper endoscopy and biopsy of the esophagus, long-segment BE is diagnosed. The patient is treated with RFA. What is the follow-up?

**Recommendations:** The patient should undergo follow-up upper endoscopy several months after RFA, looking for evidence of BE and the need for biopsies. If needed, RFA could be repeated during this endoscopic follow-up because it is just a superficial burn. The biopsy samples should be sent for permanent section.

Barrett Esophagus: Managing High-Grade Dysplasia With Cancer

**Case:** As you perform upper endoscopy to evaluate a patient with reflux, you see what looks like a 4-cm segment of Barrett esophagus (BE) just above the gastroesophageal junction. The biopsies come back with high-grade dysplasia with invasive cancer. How should we manage this patient?

**Stage 0 Cancer:** If the patient has stage 0 cancer (in situ cancer), then the cancer is just within the mucosa. If the section is small enough, the patient can be treated with endoscopic mucosal resection.

**Invasive Cancer:** If the cancer is invasive and positive nodes are present, then surgical resection is needed. Several minimally invasive options are available: laparoscopic transhiatal esophagectomy, thoracoscopic surgery, or both. I am not a fan of the two-stage intrathoracic anastomosis to join the esophagus and stomach. I am a fan of performing the anastomosis in the neck because managing anastomotic leaks is easier in the neck than in the thoracic cavity. Patients who develop an intrathoracic leak rapidly become very sick. Therefore, my preferred surgeries are laparoscopic transhiatal esophagectomy or a three-stage esophagectomy with anastomosis in the neck.
Bariatric Surgery: Patient Selection

We have all read or heard about how the number of bariatric procedures is increasing. We used to have guidelines from the National Institutes of Health (NIH) to help us determine which patients should undergo a bariatric procedure. Today, the NIH guidelines are still available, and many insurance companies are still using these guidelines. The traditional NIH guideline for bariatric surgery was a body mass index (BMI) of 35 with significant comorbidities, such as severe sleep apnea, severe or uncontrollable diabetes, or severe hypertension. I used to include people with severe joint problems who could not get their joints fixed because of their obesity. These guidelines still hold true if the patient has a BMI >40. Although not been universally accepted, there has been a movement for including diabetics with type 1 obesity (BMI 30 to 35) because of the metabolic improvement associated with these procedures, especially Roux-en-Y gastric bypass. The age range for bariatric procedures used to be 18 to 65 years, but we are now operating on older and younger patients.

**Adolescents:** To operate on an adolescent, a couple key things need to be in place. (1) Make sure the patient has bone maturity — that the patient has reached whatever bone growth they are going to reach before undergoing a bariatric procedure. (2) Make sure the patient is psychologically mature. You do not want to operate on an adolescent and then have them act out through their bypass. Therefore, do a very thorough and complete psychological profile on adolescents before operating on them. The problem is that young and obese individuals have a risk of ≥80% of becoming an obese adult. The question is whether earlier intervention is beneficial for these young individuals. Overeating is a learned habit, and I do not know if we can reset brain patterns in terms of the reward center. But the earlier we can get someone and help with the weight loss, we definitely will help with survival.

**Long-Term Benefits:** Bariatric surgery improves long-term survival. The survival benefit begins to kick in at about 11 months for patients aged ≥65 years and at about 6 months for those aged <65 years. At that time, comorbidities begin to improve. Additional evidence demonstrates that the upfront costs of the procedure are worth it because as comorbidities improve or resolve, medical expenses are reduced via less medication use, etc. One barrier to insurance companies paying for these procedures is that the short-term costs are sometimes not worth it to them because people rotate so frequently through these companies. Even in my state, it can be very difficult to get approval from certain insurance companies.

Bariatric Surgery: Sleeve Gastrectomy

Laparoscopic sleeve gastrectomy (LSG) is the most commonly performed bariatric procedure today, surpassing gastric bypass as the most popular procedure. Michel Gagner came up with the concept of LSG because, basically, a gastric sleeve is part of the duodenal switch procedure. LSG involves creating a gastric sleeve, doing the division of the duodenum, and then doing the duodenal switch with the re-anastomosis. When he started performing LSG as a staged procedure, he found that patients did not come back to get the rest of their cases because they were losing weight, and it was staying off.

**Weight Loss:** Although LSG is very popular, we do not really have the long-term data regarding weight loss that we have with the Roux-en-Y procedure. The Roux-en-Y goes back to the 1980s, so we have both open and laparoscopic data. From the current data available, LSG seems to have a mid-range effectiveness (60% to 65% excess weight loss at to 2 years), being almost as effective as gastric bypass (70% to 75% excess weight loss at to 2 years).

**Morbidity:** Compared to gastric bypass, LSG has a lower morbidity/complication rate overall and it is technically less challenging. With LSG, the surgeon removes most of the stomach along the greater curvature up to the angle of His and leaves a relatively small tube.

**Other Advantages:** For patients who must pay for the procedure themselves, LSG is associated with a very low morbidity, low mortality, reduced risks technically, and good weight loss. In addition, results of a randomized study demonstrate that diabetes resolution is the same with LSG and gastric bypass.
**Bariatric Surgery: Leaks and Their Prevention**

The Achilles’ heel with laparoscopic sleeve gastrectomies (LSGs) is leaks. The small gastric tube made during LSG creates a high-pressure system, making it very difficult to close leaks that develop along the staple line. Gastric bypass generates a low-pressure system, and adequately drained leaks will generally close on their own with time. To prevent the leaks that complicate LSG, here are a couple of helpful technical hints. (1) Ensure adequate distance from the duodenum (at least 6 cm) before doing the division of the stomach. (2) Realize that smaller gastric sleeves equate with higher-pressure systems, thus increased risks of leaks. For example, the surgeon may decide to use a 40 bougie as a guide for making the gastric tube because smaller gastric tubes result in greater weight loss. However, the leak risk is greater with a 40 bougie than with a 50 bougie because the smaller tube creates a higher-pressure system. Therefore, the surgeon must make a tradeoff: smaller tube for greater weight loss but a greater risk of leaks or larger tube with less weight loss but a reduced risk of leaks. (3) Ensure that you are not too close to the gastric incisura when dividing the greater curvature. If the tube is too narrow at the incisura, a kink will result, causing a functional blockage or stricture that can cause a blowout of the staple line up higher. (4) Ensure that you get an extremely good staple closure in the region of the fundus/angle of His. (5) Consider oversewing the entire length of the staple line or using staple line reinforcement agents that can be fired with the stapler. (5) Consider reattaching the omentum to the top, middle, and bottom of the gastric tube. The sleeve can potentially twist when the omentum is not reattached, resulting in a functional blockage and generating higher pressures.

**Prevention:** Immediately after dividing the stomach and creating the tube, the omental area that was divided off is reattached by stitching it onto the staple line. A couple of stitches along the border helps keep the omentum attached, thus helping keep the sleeve aligned. Not every surgeon advocates this step, but I do.

**Presentation:** With gastric bypass, leaks generally are an early complication, and tachycardia is a marker for leaks in the postop hospitalized patient. Unlike gastric bypass, LSG patients can present with leaks (rate, 5% to 7%) either in the immediate postop period or 2 to 3 weeks after the procedure because the anastomosis gets weaker about 1 to 2 weeks out. Once the patient goes home, tachycardia is not a good marker for leaks. Patients with a late presentation of leaks generally show up 3 weeks after surgery, are not feeling well, and have CT scans showing a fluid collection in the left upper quadrant by the angle of His where the fundus use to be.

**Esophageal Achalasia, Food Impactions, and Eosinophilic Esophagitis**

Three different esophageal conditions encountered by surgeons include esophageal achalasia, food impactions, and eosinophilic esophagitis.

**Achalasia:** Heller myotomy is the standard for treating esophageal achalasia, but peroral endoscopic myotomy (POEM) has become a treatment option as well. With POEM, a small opening is made high up in the esophagus in the mucosa and the endoscope is run between the mucosa and the muscle, cutting the circular fibers of the esophagus and creating a tunnel as it advances. With POEM, a very long myotomy can be performed — if you enter up high, you can do a myotomy the whole length down, dissecting right along the submucosa and then dividing the circular fibers. Additionally, the risk of perforation is not high because the longitudinal fibers are kept intact as you advance the scope. The results of POEM are comparable to those of traditional Heller myotomy. I believe POEM is a good option for individuals with diffuse esophageal spasm or for patients needing a long myotomy.

**Food Impaction:** Managing an esophageal food impaction requires that you first protect the airway. The best way to go about diagnosing and treating it is via endoscopy. Sometimes, food impactions will spontaneously pass. But patients with a persistent impaction generally present for help. Glucagon can be administered because it relaxes the lower esophageal sphincter, but caution should be exercised.
because too large of a dose can cause nausea and vomiting, both of which are contraindicated in these cases. Generally if the patient is salivating at the mouth, immediately go to endoscopy and either gently push the impaction into the stomach or extract it out the mouth. Do not use meat tenderizer — this is an old wives’ tale. The main thing to keep in mind when dealing with a food impaction is that 80% of the time some sort of esophageal pathology will be present, such as peptic stricture or tumor, Schatzki rings, or eosinophilic esophagitis (EoE).

**Eosinophilic Esophagitis:** EoE is a newer entity that, on endoscopy, appears as concentric rings or a white exudate in the esophagus. It is seen most frequently in young, relatively healthy people. Patients with EoE get food impactions. To treat the impaction, remove the impaction via endoscopy and let the inflammation die down. Later, take biopsies along the entire length of the esophagus at intervals and send to the pathologist to establish the diagnosis. Because EoE is like an allergic reaction to food, we should test for allergies and then patients should avoid those foods. Some patients also take topical steroids, and some require a little bit of dilatation.

**Endoscope-Related Esophageal Perforations**

Esophageal perforations can occur with a difficult passage of the endoscope at the cervical level, most commonly in elderly patients. For a relatively contained perforation at the cervical level in patients who are not showing signs of sepsis, conservative measures can be used such as nothing by mouth, IV antibiotics, observation, etc. Most of these perforations respond nicely to this supportive therapy.

**Distal Perforations:** Distal esophageal perforations related to endoscopic injury usually occur usually when a stricture is present and concurrent dilatation is being attempted. The perforation is generally proximal to the stricture. Location is important. If you have a distal stricture, but you have enough space to stent it, then I would place a stent, especially because strictures can be associated with malignancy. Therefore, I would place a stent across the stricture and institute supportive measures such as nothing by mouth and IV antibiotics. Then I would make sure that I have controlled any leakage or drained anything that needs to be drained. This approach generally works well.

**GE Junction:** If the esophageal perforation is toward the gastroesophageal junction, placing a stent can be challenging, especially in the setting of a malignancy that might be eroding into others things. My preference is to place a covered stent to cover the perforation. Migration of these stents can be a huge problem.

**Stent Migration:** Migration of stents is a huge problem throughout the GI tract. Wherever we put these stents, they can migrate. It seems like we cover the perforation with a stent and then our patients get better. Then we know when the stent has migrated because our patients start to get sick again. Retractable stents allow us to remove the stent once the perforation has healed, thus greatly reducing the risk of stent migration.

**Paraesophageal Hernias: Asymptomatic and Symptomatic Patients**

Paraesophageal hernias (PEHs) are commonly seen by most general surgeons. The typical patient presenting with a PEH is an elderly women living in a facilitated care environment who comes in with a terrible esophageal hernia, incarceration, or anemia. Typically, they have anemia or they are being worked up for GI bleeding. Often these elderly patients have several comorbidities, meaning they are not good surgical candidates. On occasion, an asymptomatic patient will be referred to us after having an upper GI that shows a PEH. Although our standard in the past was to operate on any PEH, recent data demonstrate that the risk of subsequent incarceration resulting in strangulation and necrosis of the stomach is <1.5% in asymptomatic patients. Therefore, in asymptomatic patients, I watch these cases. When patients become symptomatic, 50% start having anemia and have endoscopic evidence of
Cameron lesions (linear gastric erosions usually where the stomach is at the level of the diaphragm). Other patients have really bad reflux symptoms and may have postprandial pain or chest fullness. These symptomatic patients need a workup, and we need to proceed with fixing them if they are an acceptable surgical risk.

Paraesophageal Hernias: Surgical Repair

Paraesophageal hernias (PEHs) are challenging to repair because most will recur. Nonetheless, when we do the repair, patients get symptom relief that often persists, even if the hernia recurs. To help minimize recurrences, I make sure that I completely dissect out and remove the sac. I generally reduce the contents, incise the peritoneum on the anterior aspect of the crura, create a little peritoneal flap of the sac, and then circumferentially dissect in that plane, which is usually superior to the esophagus and in the anterior aspect of the mediastinum. I dissect out that entire plane, all the way around, dissecting off the aspect of the esophagus. Then I pull that down into the abdomen. I transect it at the level of the gastroesophageal junction, which is often where the inferior aspect of it is located, and then remove it. I next make sure that I have sufficiently mobilized the esophagus, because shortened esophagus will not work in these types of patients. Next, I take down the short gastrics, clean up the angle of His, create a posterior window, and create a circumferential dissection of the esophagus. With all this dissection, I must be careful to avoid injuring the vagal nerves on either side. Once all this is done, I proceed to repairing the crura. Typically, I first do a posterior repair, suturing the crura to one another posteriorly using at least three interrupted sutures (nonabsorbable suture material). I do not use pledgets. Next, if needed, I put a couple of sutures anteriorly. Now, I either do a partial or a full (Nissen) fundoplication because these patients have reflux afterward. When I form my wrap for the Nissen, I usually have a 50 bougie in place to make sure that I am not making it too tight. Once I bring it down, I do that posterior repair, close up the anterior, and then reinforce it with a biologic mesh. I do a keyhole procedure in which I will have the main reinforcement on the anterior aspect of the crura and then I bring the flaps down, the keyhole aspect down, and cover the posterior crura and tack it there. Sometimes I use some fibrin glue, and then I place the biologic and leave it in place to let the glue set. I do not necessarily use a lot of sutures to suture it into place. If I cannot get the crura together, I’ll do a relaxing incision (similar to that used with primary inguinal hernia repairs) to the side on the diaphragm, allowing me to get the closure, and then I cover up that relaxing incision. Do not put this incision at the site of the hiatus. When I cannot get the crura together, this usually occurs anteriorly. I do not drain the resulting dead space in the chest because I am more concerned about getting into the pleura and creating a pneumothorax. In general, I do not place a gastrostomy tube. I usually place a nasogastric tube and keep it in overnight for decompression.

Caustic Chemical Ingestions: Management

Case: A patient has tried to commit suicide by ingesting some type of household cleaner. How should these patients be managed initially as it pertains to the esophagus and stomach?

Recommendation: First, protect the airway and resuscitate the patient. Then, within 24 hours, perform upper endoscopy and gently assess the degree of injury. Typically, people ingest alkaline agents, basic household cleaners, because acidic agents hurt. Alkaline ingestion causes liquefactive necrosis, and because the compounds bind to tissue proteins, injury can extend all the way through and into the mediastinum. Ingestion of acidic compounds causes coagulation necrosis, which basically is thrombosis, resulting in scar formation. When assessing the degree of injury during endoscopy, the presence of grade 2B (focal, deep circumferential ulceration) or grade 3A injury (focal necrosis) indicates that the risk of stricture is very, very high.
**Steroids:** The use of steroids to treat the ingestion of caustic agents has gone the same way as the use of steroids for spinal trauma — they are no longer used in these situations. **Care:** Typically, we first provide supportive care (IV antibiotics, resuscitation, etc), and then we treat the stricture. With more severe grades of injury, we know that the likelihood of strictures is very high. These cases usually require long-term treatment processes in which you try to improve the stricture endoscopically, but down the road they will most likely need to be removed.

**Upper GI Bleeding: Acute Management**

**Case:** A patient presents with hematemesis, and they have not undergone endoscopy. From a resuscitation standpoint, how should these patients be managed? **Recommendations:** I approach these patients similar to any trauma patient that’s bleeding. We focus on basic principles of resuscitation, which would include airway, breathing, and circulation. Most of these patients, even though they are having issues with hematemesis, are able to maintain their airway. I do not advocate routine intubation of any patient with an upper GI bleed. However, when patients are vomiting profusely or they are so hypotensive and delirious that they cannot protect their airway, then intubation is required to protect their airway. Beyond that, from a circulation standpoint, basic principles apply. We need to ensure they have adequate IV access that might consist of a pair of large-bore IV catheters or a central access, which would include a rapid infuser or a Cordis® introducer sheath. While we are securing access, we like to make sure that our patient’s blood type is tested and cross-matched for adequate amount of blood products based on their initial presentation (range, 4 to 6 units). At the same time, recognizing that these patients may require several units of blood products, we want to ensure that they have plasma and platelets available. For patients in profound in extremis, the rapid administration of uncrossed O-negative blood and initiation of a massive transfusion protocol is sometimes necessary. Once we are satisfied that our initial resuscitation has begun, then we can begin detective work in identifying the underlying cause of the bleed. Nasogastric (NG) decompression is helpful to alleviate distress and vomiting. NG decompression can also help with diagnosis: aspiration of frank blood from the stomach can help pinpoint the bleed and let us know where to direct our next steps. **Stomach Irrigation:** With the NG tube in place, do we still irrigate the stomach? I would like to irrigate the stomach only because I think it helps our gastroenterology colleagues with endoscopy. I do not know how much utility there is in cold water irrigation to intentionally stop bleeding, but I think, with a large-bore NG tube in place, irrigation is helpful to clean out the stomach, allowing our GI colleagues to get a better look and potentially provide better therapy.

**Upper GI Bleeding: Drugs Needed for Acute Management**

Several drugs are useful in the acute management of patients with upper GI bleeding. **Antibiotics:** Antibiotics are not only helpful but are necessary in a setting of variceal GI bleeds. For patients with a history of cirrhosis or variceal disease, early administration and maintenance of an antibiotic is necessary. The antibiotic can be a third-generation cephalosporin. Patients experiencing a variceal GI bleed have a fairly significant risk of becoming bacteremic and septic, typically from a gram-negative source associated with their variceal bleed. Therefore, the use of antibiotics decreases mortality in these patients and is considered standard of care and necessary. **Acid Suppression:** A drug to shut down acid secretion is needed for patients presenting with an upper GI bleed. Maintenance therapy relies on IV administration of proton pump inhibitors (PPIs). Historically, we started patients on the bolus of pantoprazole followed by a continuous IV infusion. However, recent evidence states that, after an initial bolus, patients can be placed on twice-daily IV dosing, which has a similar outcome. Not everyone may have changed their practice at this point,
so whether the patient is started on a continuous infusion or twice-daily dosing is currently at the discretion of the provider. I have gone to using twice-daily dosing. I think it helps reduce the need for further IV access and it is easier for nursing. Data are relatively solid that continuous infusion provides no extra benefit and that the use of a PPI actually decreases mortality. PPIs are preferred to therapy with H2 blockers.

**Portal Pressure:** In the variceal bleeders, something to offload portal pressure is necessary. In the United States, the 2 medications available are octreotide or vasopressin. Based on data from European studies using terlipressin, the preferred agent right now is to use octreotide as a continuous infusion.

**Summary:** In patients with an upper GI bleed, we rely mostly on acid suppression for nonvariceal bleeds. In patients with a variceal GI bleed, octreotide and antibiotics are the 2 additional medications we rely on.

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**Upper GI Bleeding: The Need for Repeat Endoscopy After Initial Management**

In patients presenting with upper GI bleeds, endoscopy is the diagnostic procedure of choice.

**Case 1:** A patient presents with an ulcer that undergoes initial management with either injection or clipping of a visible vessel. Is repeat endoscopy for surveillance (the patient has not rebled) of any value for in these patients, acutely at least?

**Recommendations:** I know that some of our gastroenterology colleagues still practice repeat endoscopy for surveillance. However, depending on the visualization they achieved the first time and the adequacy of their therapy, I am not sure how often additional findings are found on repeat endoscopy. I do know that several of our colleagues will repeat the endoscopy in several days, assuming the patient has maintained their hemodynamic stability.

**Case 2:** A patient presents with an ulcer that undergoes initial management with either injection or clipping of a visible vessel. The patient rebleeds. Does the patient automatically undergo repeat endoscopy?

**Recommendations:** When endoscopy is repeated depends on visualization and how the gastroenterologist felt with the first endoscopy. If the first endoscopy was exceedingly difficult and the gastroenterologist felt that there is nothing more they can do, then they often will plan ahead of time to not repeat their endoscopy. However, for most of these patients, we find on repeat endoscopy that we can do definitive therapy on a repeat endoscopy, even if the therapy on the initial endoscopy was inadequate. Therefore, except for some exceedingly complex cases, most patients who rebleed benefit from repeat endoscopy.

**Rebleeding Risk:** At the Medical College of Wisconsin, we have used the Forrest classification system to help predict the risk of rebleeding in patients treated for upper GI bleeding. The Forrest system is based primarily on the endoscopist’s visualization of the ulcer bed and whether they see a small clot, an adherent clot, or a visible vessel. Another scoring system, the Glasgow-Blatchford bleeding score (GBS), utilizes things like hemoglobin, BUN, blood pressure, and some other items in the medical history to predict the risk of rebleeding. I have not personally used this score. Some studies show GBS to be more useful than Forrest classification, but I have not based any therapy on a patient’s GBS recently.

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**Upper GI Bleeding: Angiography After 2 Failed Therapeutic Endoscopies**

**Case:** A patient presents with upper GI bleeding and undergoes initial endoscopy with treatment. The patient rebleeds, and a second endoscopy was performed. The gastroenterologist thought that things looked good and performed a second small intervention. The patient rebleeds again. How should we manage this patient?

**Recommendations:** At times, the gastroenterologist considers a third endoscopy because they believe that sedation was inadequate and that perhaps something could be done differently. But when a patient
fails after two good attempts at endoscopy, I think it is time to move on to one of our other adjuncts: either further contrast imaging or surgery, depending on the patient.

**Angiography:** CT angiography for localization prior to traditional angiography is often beneficial for such cases. Our interventional radiology colleagues feel strongly that, if they can localize the bleed with CT angiography, then they can spend reduced time on the IR table and reduce overall contrast load despite having two studies.

**Peptic Ulcers:** In cases of peptic ulcer, is angiography around the duodenum successful with our newer selective microcatheters? Collateral flow is the main issue with the duodenum along the gastroduodenal artery (GDA). If you take out just the artery proximally, flow through the pancreatic arcades sets you up for rebleed. Despite knowledge of the anatomy and understanding the potential for collaterals, I have seen highly selective angiography of bleeds of the GDA be successful. Our interventional vascular colleagues have several new ways of embolizing vessels — it’s not just coiling anymore. They now have different types of coil and thrombogenic agents. They have different gel foams and different materials to inject into the vessels. With some of these newer agents, including some of the injectable beads that can travel downstream, they are more successful than they have been in the past. I tend to reserve angiography for patients who are older or who would not tolerate a full laparotomy as well as some of the younger patients. Therefore, I think it is worth it to just take them to the operating room, deal with the bleed definitively, and avoid further transfusion of blood products.

**Peptic Ulcers: Management**

I think the classification scheme for ulcers is still critical in the management of ulcers. The classification of the five types of ulcers is generally based on their geography, which helps determine whether ulcers are related to high acid states or to loss of the mucosal barrier. More patients are receiving various NSAIDs and other medications known to break down the stomach’s mucosal barrier. Therefore, limiting the reversible risks for non–high-acid-state ulcers is useful, thus avoiding an acid suppression operation. For patients with ulcers related to high acid states, we need to think critically about how we will blunt that acid secretion, whether through lifelong PPI therapy or some sort of surgical intervention.

**Type V Ulcers:** Type V ulcers (secondary gastric ulcers) are usually associated with NSAID use. For example, an elderly patient receiving NSAIDs that cannot be discontinued presents with a bleeding ulcer that has failed the first endoscopic treatment. In such cases, we need to focus on rejuvenating the mucosal barrier of the stomach. In these patients, we have used prostaglandin therapy (misoprostol) to help fortify that mucosal barrier. Additionally, older medications, such as Carafate® and other medications aimed at improving the stomach’s lining can be beneficial. Ideally, we would have our patients discontinue the offending medication, but this is not possible in all patients. In those who cannot discontinue their medications, treatment is a balance between acid secretion and the mucosal barrier. The critical defect in these patients is the lack of a mucosal barrier. We can tip the scales in their favor with some sort of vagotomy: a highly selective vagotomy would be the procedure of choice in patients who can tolerate it. But I think a trial of improving the lining of the stomach is the first step.

**H pylori:** We all worry about ulcers being an infectious disease. To make the diagnosis, we have found that the serum antigen test for *Helicobacter pylori* has a good positive predictive value but a poor negative predictive value. If negative, then we next do a c13 urea breath test for *H pylori*. Unfortunately, no single medication can cure *H pylori*. The treatment lasts 1 to 2 weeks, depending on the regimen chosen, but we typically use a PPI and two antibiotics.

**Perforated Peptic Ulcers:** I do not believe that the number of patients with perforated peptic ulcers is decreasing. Instead, I believe we are seeing a different group of patients who are perforating compared to what we saw 20 to 40 years ago. More NSAID medications are now available, so we must take a good history before whisking patients with perforated ulcers to the operating room. The ideal operation for patients with perforated peptic ulcers is the Graham patch, especially if NSAIDs can be discontinued.
Once the perforation heals and the patient discontinues the offending agent, they typically do not have an issue with recurrence.

**Duodenal Ulcer: Operation for Recurrent Bleeds**

**Case:** An elderly patient receiving NSAIDs that cannot be discontinued presents with a bleeding ulcer that has failed two endoscopic treatments. The patient cannot be adequately hemodynamically resuscitated sufficient to get another study. They are hemorrhaging and have persistent hypotension. You know they have an ulcer in their duodenum that had a visible vessel seen by the endoscopist in two attempts to try to stop it. You take the patient to the OR. What procedure do you perform?

**Recommendations:** I open their duodenum and typically carry that onto their pylorus, ligate the vessel in the traditional fashion, taking care of that side branch. You need an intimate knowledge of anatomy because there are some other anatomic considerations given the patient’s bile duct, etc. So we suture ligate that gastroduodenal vessel from within the duodenum. I typically close those patients transversely with a pyloroplasty. Whether I do a vagotomy at that time depends on the patient’s history and the stability. My preference for the unstable patient is to get out of the OR, have further resuscitation, and deal with any other operations further down the road. However, in other settings, an acid-suppressing procedure could be done at that time as well. This procedure is usually a vagotomy. This approach is certainly a departure from the past when no one would think about leaving the OR without the vagotomy. However, stopping the bleeding is the first issue, and then making the surgical judgment about whether the vagotomy is beneficial (considering the risk) should follow.

**Variceal Bleeding in Cirrhotic Patients: Management**

The standard of care for variceal bleeding is endoscopic banding and ligation if possible. The risks of sclerotherapy in variceal bleeding are higher than with endoscopic banding. Therefore, if the varix can be banded, that is the ideal therapy. If the varix cannot be banded, then utilization of sclerotherapy endoscopically would be the next step. I do not have to use balloon tamponade because I work in an institution that affords me the ability to offload the portal system via a transjugular intrahepatic portosystemic shunt (TIPS) procedure in interventional radiology. But we keep a Minnesota tube in the hospital, and I teach the residents about its existence. However, in my career I have yet to have the need to use balloon tamponade. When we have the complete package available, the Minnesota tube or the Sengstaken-Blakemore tube is used as a temporizing measure in the patient who is massively bleeding from varices until a TIPS can be performed. Whether the tube is used to temporize a patient for transport or to temporize a patient while you mobilize the interventional radiology suite, I think the Minnesota or Blakemore tube is purely a temporizing measure to get to TIPS. I think the days of a routine surgical portosystemic shunt have fallen by the wayside.

**Appendicitis: Management**

A debate is raging about whether appendicitis is a surgical disease, a medical disease, or some hybrid form. This is an emotional topic, and it is easy to fall back on how we were trained. Nonetheless, I think each of us must critically evaluate the literature. My personal feeling is that we will not replace one therapy for another, but we will end up with two viable therapies that we can tailor to our patients on a case-by-case basis. This debate revolves around the treatment of uncomplicated appendicitis. Compared to uncomplicated appendicitis, complicated appendicitis (includes perforation,
abscess formation, or phlegmon formation) is a completely different disease process. Therefore, this discussion about antibiotic versus surgical therapy only includes uncomplicated appendicitis.

**Uncomplicated Appendicitis:** To accurately diagnose uncomplicated appendicitis, we need axial or CT imaging along with physical exam and lab work. CT is necessary if we are going to use medical therapy, but this opens up a debate of who needs a CT scan, which is another completely separate discussion. Features diagnostic of uncomplicated appendicitis include no fecalith, some wall thickening, some periappendiceal stranding, an absence of a substantial amount of free fluid, no free air, no phlegmon or abscess, and disease primarily relegated to the appendix itself, save for some mesenteric or periappendiceal stranding.

**Current Treatment:** When uncomplicated appendicitis is identified in a patient, I have a discussion with them and let them know the alternatives. To have this discussion, I must be knowledgeable of the available data regarding outcomes with medical treatment. The risk of medical treatment failure is higher than that of surgical therapy. According to the newest data, the failure risk is in the 9% range. However, the risk of perforation or risk of presentation with complicated appendicitis is no different than that for the general population (just under 3%). Length of stay tends to be longer for patients undergoing medical therapy. Return to work tends to be shorter because they do not deal with postoperative issues. For other complications, the rates tend to be the same for medical and surgical therapy.

**Laparoscopic Appendectomy:** With laparoscopic appendectomy, assuming that everything goes right, the risk of recurrence is 0. The patient generally goes home the same day. When the patient says, “Gee, why would you think of treating me with antibiotics where I have a risk of recurrence, I’ve got to stay in the hospital at least a day, and then I’ve got to take those drugs?” I validate my patient’s concerns and say those are all good reasons to take the surgical risk and go ahead with appendectomy. I do discuss the risk of surgical site infections and intraoperative complications with patients. But for most of my young, healthy patients presenting in the typical appendicitis demographic, we usually end up in the OR after having had an honest discussion of the outcomes of medical therapy.

**Laparoscopic Appendectomy: Tips, Tricks, and When to Convert**

Laparoscopic appendectomy is one of the most common procedures a general surgeon performs. Our group has 10 surgeons, so we have about 10 different ways to put ports in for an appendectomy. My personal approach is to gain entry through the umbilicus, and I use two left-sided ports to go after the appendix. I decompress the bladder because it can often get in the way, although this is not completely necessary. My preference is to use a 5-mm port in the umbilicus and a 10- to 12-mm port in the left lower quadrant for my stapling device. In obese patients, the left lower quadrant port site is sometimes too far away from the appendix, so I place that port in the suprapubic region. At times, a right upper quadrant port can be very useful to help retract the right colon. At times, placing this fourth port in the right upper quadrant has saved me from having to convert to an open case, so I think it is a good trick. Angled scopes are very useful. I do not irrigate — some data support my concern that irrigation in the setting of appendicitis can cause infectious complications remote from the original site. Therefore, all I do is suction effluent away from the original operative site.

**Converting to Open:** Standard factors can cause me to convert to an open procedure: inadequate visualization, an inability to safely dissect the appendix off of the surrounding structures (whether the underlying ureter or iliacs), or sometimes the appendix is both retrocecal and completely stuck to the now-overlying cecum. Before converting to an open procedure, I take steps to help myself. Sometimes, I will do a full mobilization of the right colon starting from the hepatic flexure and working down — I start away from the inflammation to take advantage of unobstructed or unviolated planes. However, if I cannot safely get a stapler across good tissue, be it the base of the appendix or some of the cecum, or if I cannot safely dissect the appendix off of the surrounding structures, then I will convert to open.
We do not run into it very often, but occasionally the appendix is acutely inflamed all the way to its base. If I can see enough cecum that I can safely fire a stapler across it without affecting the terminal ileum, then I have no problem firing that staple line.

**Complications:** When performing laparoscopic appendectomy for uncomplicated appendicitis, the risk of wound infection is always a concern. Comparing laparoscopic to open cases, the risk of an organ space surgical site infection or intraperitoneal abscess is higher with laparoscopy, but the risk of superficial surgical site infection and, subsequently, hernia is lower with laparoscopy.

**PEG Tubes: Placement Tips**

General surgeons often find themselves placing a percutaneous endoscopic gastrostomy (PEG) tube. I think because this is such an easy procedure, we tend to downplay the potential complications that can occur. While complications are not common, I would not call them rare.

**Avoiding the Colon:** When placing PEG tubes, one of the fears we face is that of going through the colon before entering the stomach. I have a few tips to help the surgeon avoid the colon. First, I dig through the patient’s record to review any axial imaging of the abdomen. I do not need to obtain such imaging before going into the operating room, but if it exists, I like to take a good look at the location of things. The anatomy of the peritoneum will change with insufflation of the stomach, but it is good to get a general sense of where things are before I start, to ensure that the patient does not have a large paraesophageal hernia or something else that could get in the way. Next, during the procedure, we do a full insufflation of the stomach and make sure all the rugae disappear, which indicates that the stomach is well insufflated. Then I look for a good light reflex on the anterior abdominal wall. I like to see the light from the endoscope fairly clearly on the anterior abdominal wall. Then with palpation of the anterior abdominal wall, I want to see that impulse immediately transmitted to the lumen of the stomach. Although I tend to keep the patient flat, some surgeons place the patient in a bit of a reverse Trendelenburg position.

**Avoiding the Liver:** To avoid the liver during PEG tube placement, I again look for it on preexisting axial images before I go to surgery. I review the image to see the size of the left lobe of the liver. I make sure that is it not draping all the way over the greater curve of the stomach. Going through the liver is not nearly as big a deal, however, as going through the colon. Traversing the liver is a problem if the patient has cirrhosis with expected liver changes.

**PEG Tubes: Buried Bumper Syndrome and Replacing Tubes**

After gastrostomy tube placement, the surgeon may see the patient again for buried bumper syndrome or to replace a tube that has fallen out.

**Buried Bumper Syndrome:** Buried bumper syndrome occurs when the bumper (the flange or mushroom) from the PEG tube has eroded through the wall of the stomach and resides in some layer within the abdominal wall. The typical presentation is pain with administration of tube feeds or with flushing of the tube. This occurs well after a solid gastrocutaneous fistula has formed and the stomach is nicely adherent to the anterior abdominal wall. As the tube erodes through the stomach, the stomach does not fall away, so we do not get findings of peritonitis or leakage of tube feeds into the peritoneum. Patients present with either a local cellulitis or pain with flushing of the tube. If the patient no longer needs the tube, I will simply remove it. If the patient still requires feeding access, then the best way to deal with this tube is in interventional radiology or fluoroscopy. Because the stomach is still adherent to the anterior abdominal wall, we can typically gain access to the stomach percutaneously, through that tract, after the tube has been removed. We can typically get a wire into the stomach, and then using a dilator and a split-sheath introducer, we can insert a new gastrostomy tube. If the anterior abdominal
wall has too much cellulitis, too much local inflammation, an abscess, or something in the way, then the
treatment is to remove the tube, treat the local site, and either to get a temporizing nasogastric tube
(or nasojejunal tube) or to insert a gastrostomy tube at a different site. Although the anterior abdominal wall
can sometimes prevent us from inserting a new tube, we can usually insert a tube into the same location.

**Replacing Tube:** Sometimes an established gastrostomy tube falls out. The patient comes to the
emergency department within 4 to 6 hours of it falling out. Our goal is to reinsert another tube in a
timely fashion, even if it is a temporary tube, so that the gastrocutaneous fistula does not close.
Our residents are all trained in this procedure and they insert a Foley catheter, a red rubber catheter,
or something to keep the tract open until we can insert a typical gastrostomy tube. If the replacement is
uncomplicated and gastric contents are aspirated, then I do not require a study to confirm placement.
If the placement is difficult or we are not getting gastric contents back, then we send the patient to
fluoroscopy for injection of a little contrast to confirm placement into the stomach.

**Colonic Pseudo-obstruction (Ogilvie Syndrome): Management**

Ogilvie syndrome (colonic pseudo-obstruction) is basically a massive dilation of the entire colon.
On abdominal x-rays, the cecum is >10 cm but no frank mechanical obstruction is seen.
Because the entire colon is dilated, the risk for perforation is high if we cannot decompress the patient.
Patients are typically either older and on anticholinergic medications for neurologic disorders,
or they are “institutionalized patients” receiving chronic psychiatric medications and have colon motility
issues secondary to some of those medications.

**Management:** Our priority is to get the patients decompressed as quickly and safely as possible.
The more dilated the colon gets, the greater the wall tension, especially on the cecum, and the greater the
risk of perforation. We typically recommend discontinuing the offending agents, but the medications are
usually on board for a reason and cannot be discontinued chronically. These patients are made NPO,
and decompression with a nasogastric tube can be helpful. However, to decompress them, we either do
colonoscopic decompression or administer neostigmine. We usually manage the patient collaboratively
with our gastroenterology colleagues. The use of neostigmine is associated with several risks. First,
we must confirm that no distal obstruction is present. Then, we must be prepared because neostigmine
administration may cause bradycardia and bronchospasm. Because the colon is massively dilated,
it is usually fairly easy to do a flexible sigmoidoscopy. Often, the gastroenterologist can insert a small
decompression tube up through the transverse colon and hepatic flexure. Because we are dealing
primarily with gas, we can successfully decompress the colon that way too. At our center, when we
decide to use neostigmine, the patient is brought to the intensive care unit and placed on telemetry.
I always make sure that a dose of atropine is at the bedside in case the patient becomes profoundly
bradycardic. Then I try to walk away because when neostigmine works, it works right away. If the first
dose of neostigmine does not work, a second dose may be given. I typically try a second dose a little bit
later, and if that does not work, then we switch to colonoscopic decompression. The gastroenterologists
use CO₂ for colonoscopic decompression. Because decompression is the goal, the last thing you want to
do is add more insufflation. I think trading the air in the colon for CO₂ on the “way in” is beneficial
because the body will reabsorb the CO₂ fairly quickly. If neostigmine is administered, I find that it has
worked nearly every time I have used it. Most information says that the success rate is 85% with the first
dose and perhaps a little more with the second dose. This is certainly a fairly high success rate for a
troublesome condition.
Meckel Diverticulum: Management

Case: You are performing a laparoscopic appendectomy on a young individual with appendicitis. As you are looking around, you see a Meckel diverticulum (MD). What do you do?

Recommendation: Because the patient has appendicitis, then I do not do anything with the MD. I take their appendix out and call it a day.

Symptoms: In the adult, symptoms are more commonly that of a Meckel diverticulitis rather than bleeding. But either of these two sets of symptoms will generally send the patient to the OR. If the patient has an inflamed wide-base MD, I believe that the best way to get that out is with a local small bowel resection and primary anastomosis. With a wide-base MD, just stapling off at the bottom is not an appropriate way to deal with these patients.

Finding MD During Trauma Lap: If you are performing a trauma laparotomy and have finished fixing the small bowel injuries, how do you manage the discovery of an isolated MD that is located away from the injuries? During a trauma lap, I will leave the MD alone. However, I will inform the patient of the MD and I will note it in their chart in case the patient ever presents with some vague abdominal symptoms.

Intestinal Pneumatosis: Management

Historically, pneumatosis intestinalis is an ominous sign that is indicative of ischemic or necrotic bowel, and we were taught to find ourselves in the OR as quickly as possible with one of these cases. However, cases of benign pneumatosis intestinalis exist.

Benign Cases: We must trust our physical exam to identify benign pneumatosis and prevent an unnecessary operation on what are typically somewhat fragile patients. Patients that get benign pneumatosis are not patients that you want to operate on if you do not have to. Causes of benign pneumatosis include chronic obstructive pulmonary disease, emphysema, and some causes of bacterial translocation.

Severe Cases: Some cases of pneumatosis intestinalis are related to ischemia or necrotic bowel. Patients present with signs and symptoms of an intra-abdominal catastrophe: they are tender and may have a metabolic acidosis and lactatemia. Patients who present with signs and symptoms of peritonitis need an exploration. Patients with a completely benign abdomen who are otherwise hemodynamically normal and do not present with signs and symptoms of sepsis and peritonitis are the patients that can undergo observation. Therefore, besides the pneumatosis, the clinical exam gets you into the OR with these cases. Laboratory findings indicative of severe pneumatosis include an acidemia or acidosis, pH <7.3, low bicarbonate, elevated lactate. These labs along with the presence of other findings on CT (mural necrosis or free air, free fluid, portal venous gas, other findings) may go along with intestinal necrosis.

Neutropenic Enterocolitis: Management

Surgeons sometimes encounter cases of neutropenic enterocolitis (NEC), especially at hospitals that have a bone marrow transplant unit. Neutropenic patients typically have very low absolute neutrophil counts and they get NEC or neutropenic typhlitis (inflammation around and near the cecum and the fat surrounding the cecum). NEC presents very similar to appendicitis (right lower quadrant pain), but most NEC patients are managed with antibiotics, conservative therapy, bowel rest, and time. Because these cases can progress to perforation or other problems, we must keep a close eye on them. However, short of findings of perforation, the mainstay of therapy is antibiotics and bowel rest.

Imaging: CT images of NEC or neutropenic typhlitis typically show inflammation in the area surrounding the cecum and in the fat and the mesentery surrounding the cecum. Some mural thickening in that area is seen as well.
Management: We test patients for *Clostridium difficile* and other things that can cause colitis in neutropenic patients, such as cytomegalovirus. Treatment is basically antibiotics, fluid resuscitation, and bowel rest. Chemotherapy is usually delayed until full recovery is achieved. Some of the more severely ill patients may receive granulocyte colony stimulating factor to help accelerate the leukocyte recovery because this typically happens during the nadir of their white count during their chemotherapy regimen. The real dilemma is trying to stay out of the OR with these patients because these patients present every surgeon’s worst nightmare. They have all sorts of infectious and healing complications that we would rather not get into. Plus, if surgery were performed, it would further delay their chemotherapy.

**Hepatic Focal Nodular Hyperplasia**

Focal nodular hyperplasia (FNH) is certainly more common than we have appreciated in the past. For the most part, FNH has very classic radiographic features with a stellate scar, and this disease is often picked up incidentally — it is not typically associated with any systemic findings for the patient. From a surgeon’s perspective, FNHs do not have a malignant potential, and they are not likely to bleed. The diagnosis is generally made radiographically, especially using MRI and CT. Generally, diagnosis does not require a biopsy. Once diagnosed, patients with FNHs do not need further imaging follow-up if the lesion is a classic-appearing FNH. Often, patients with FNH are not even referred to surgeons anymore. But if they are referred, the patient can be comforted and does not need any intervention. **Oral Contraceptives:** Are FNHs associated with the use of oral contraceptives (OCPs)? This is a very loose relationship. Because we tend to see FNHs a bit more frequently in women than men, the data suggest a minor association may exist with estrogen exposure. But if a woman is on an OCP, she does not need to discontinue its use when FNH is diagnosed. The same thing is true for women receiving supplemental estrogen.

**Hepatic Adenomas**

Other than cysts, hepatic adenomas (HAs) and focal nodular hyperplasia (FNH) are the two most commonly discussed lesions in the benign category of liver tumors. But HAs are certainly different than FNHs. These lesions are more common in women than men, and they have a direct correlation with estrogen exposure. Therefore, prolonged OCP use is associated with HAs. These tumors tend to grow rapidly in two different populations: (1) during pregnancy with spikes in estrogen levels, and (2) in older in women who are receiving estrogen supplementation. The frequency with which women on estrogen supplementation present with HAs has decreased because of how estrogen supplementation is currently used. **Management:** If an HA is diagnosed that is <4 cm in size, then the general recommendations are that estrogen exposure be withdrawn (discontinue OCPs) and to encourage patients not to get pregnant during this time. By reducing estrogen exposure, a percentage of HAs will spontaneously regress. The management challenge comes when a young female with an HA is planning to get pregnant. Then, the indication for surgery must be balanced with their pregnancy, but if the lesion is <4 cm, the indication for surgery is not strong. A typical HA has very characteristic imaging features, especially with MRI. If the lesion is >4 cm, then surgery is recommended because the risk of malignant transformation within the HA is approximately 8% during the patient’s lifetime. The second indication for surgery is if the adenoma has bled into itself because follow-up becomes more challenging after the hemorrhage resorbs and the imaging features are no longer typical for HA. The third indication for surgery is a symptomatic patient. **Males:** HAs are very rare in men. But when HAs occur in men, the incidence of malignant transformation increases significantly. Therefore, resection is recommended for all HAs diagnosed in men.
Natural History: We have learned that not every adenoma is the same: there are multiple subtypes based on their histology and gene expression. Some HAs are at higher risk for developing malignancy than are others. When they undergo malignant degeneration, HAs most typically transform into a hepatocellular carcinoma (HCC).

Hepatic Adenomatosis

In the United States, hepatic adenomatosis is being seen with increasing frequency. This is diagnosed in patients with multiple hepatic adenomas of varying size. Although these lesions were not well reported through the 1980s and early 1990s, the hepatobiliary community is certainly seeing more of these patients now.

Management: Hepatic adenomatosis is more complex than are HAs. To decide how best to manage hepatic adenomatosis in a patient, first look at the individual lesions, and then try to approach them similar to the management recommendations for HAs. Sometimes if you see a cluster of adenomas, a more formal hepatectomy may be required. But this can be a challenge when bilobar disease is present. The question comes up of when a transplant plays a role in management of hepatic adenomatosis.

As of yet, no definitive guidelines are available for managing these lesions. But, I think general surgeons should refer patients with multiple adenomas to a liver center. Regardless of what primary treatment patients undergo with a general surgeon, these patients require long-term monitoring and follow-up because they are potentially going to have residual adenomas after the initial treatment.

Characteristics: For a given case, multiple adenomas will each vary in size and typical patients will have 8 to 15 adenomas in their liver. While it is rare for the adenomas to be the same size, you may see a couple of very large lesions or maybe a lesion that has hemorrhaged into itself and then you may see some intermediate-sized lesions (3 to 5 cm) and some small lesions (1 to 2 cm). On imaging, the adenomas have classic imaging features consistent with being an adenoma. They also have classic histologic features consistent with adenomas.

Risk Factors: The main risk factor for hepatic adenomatosis is estrogen exposure, such as that seen with polycystic ovary syndrome or women who have had a higher estrogen exposure during their lifetime.

Hepatic Hemangioma

Hepatic hemangiomas are very common benign liver tumors that occur in approximately 10% of the population. Many of these lesions are small. Hepatic hemangiomas have classic imaging features (an encapsulated-type lesion filled with blood) that MRI and CT are very good at detecting. Before these advances in imaging, we recommended surgery for “giant cavernous hemangiomas.” However today, if patients are asymptomatic, we do not recommend surgery. The two exceptions to this recommendation are (1) symptomatic hemangiomas and (2) lesion location. Sometimes if the lesion is exophytic in location, the surgeon will discuss the increased risk of trauma-related bleeding due to seatbelt injuries in motor vehicle collisions. However, things have changed so much with motor vehicle collisions, that the collision in and of itself would not be reason for surgery. Hepatic hemangiomas do not have a malignant transformation or degenerative phase.

Atypical Hemangiomas: Good cross-sectional imaging is important to identify atypical hemangiomas. I recommend that hepatic hemangiomas be imaged at a center that does a lot of liver imaging diagnostic work because you do not want to miss an atypical hemangioma, which is in the variant of a low-grade sarcoma. It is an angiosarcoma. Remember, hemangiomas occur along a spectrum — hemangiomas do not convert to low-grade angiosarcoma. As such, if the patient undergoes imaging at a low-volume center, then a low-grade angiosarcoma of the liver could be misinterpreted as a hepatic hemangioma. A low-grade angiosarcoma is not a common disease, but you need to ensure that these are not present via
the use of good imaging and experienced readers. If imaging shows a typical hemangioma with the classic uptake of contrast in an asymptomatic patient, then no recommendation for follow-up imaging is made.

**Preop Considerations:** If imaging shows a very large hemangioma, we do not use embolization in an attempt to shrink the lesion. These lesions do not shrink very well. This lesion contains a nest of blood vessels in and around the hemangioma, so with preoperative embolization, we will not see involution because the lesion has one main feeding vessel. If you do try to embolize the lesion, the most common result is partial embolization, followed by partial necrosis of the lesions. Therefore, the patient will become febrile and experience pain. Therefore, even if the hemangioma is a very large lesion, I evaluate the risks associated with the lesion’s location, the rupture risk (these lesions do not spontaneously rupture), and the patient’s symptomatology before making a final decision for or against surgery.

**Benign Hepatic Tumors: Surgical Approaches and Managing Biliary Fistulae**

When faced with a benign hepatic tumor that requires surgical intervention, the decision to use a formal anatomic resection (formal right or left hepatectomy) versus a nonanatomic resection (segmentectomy or a multiple-segment segmentectomy) is based, to a large degree, on personal preference. Because these are benign lesions, obtaining negative margins is not necessary. The key to performing surgery on these benign lesions is to use an approach that will make the operation smoother and quicker for the patient. The biggest trend we see is doing these cases laparoscopically and/or robotically, which, in the literature, have been demonstrated to be safe in the right hands. Proper case selection is important.

**Biliary Fistula:** Bleeding and bile leaks are potential complications of nonanatomic resections. To reduce bleeding, low perioperative central venous pressure is the recommended approach for elective hepatic surgery. Regarding bile leaks, the first approach is to perform a dissection to separate the parenchyma from all the vascular and biliary structures, even small ones, then clip and divide these structures to keep bile leaks to a minimum. Surgeons use a number of different parenchymal dissection techniques to reduce bile leaks, such as water-jet type therapies, high-energy precoagulation, a finger fracture dissection method, or stapling through the hepatic parenchyma.

**Treatment:** Occasionally, bile leaks will occur. When identified intraoperatively, several approaches are available. Intraoperatively, topical sealants can be used safely. Other intraoperative options are to clip the bile duct or to use surgical ligatures. Omental flaps are yet another option: some surgeons like to put a layer of omentum over the raw surface to offer some protection if they plan to place a drain. Not all surgeons drain their liver surgical resections, so an omental flap provides a living, breathing “biomatrix” to help with sealing the bile duct.

**Drains:** Selective drainage may be used in nonanatomic hepatic resections. For patients with normal liver parenchyma, I do not routinely drain after doing a segmental or subsegmental resection with a dry field at the end of the case. I am more inclined to drain anyone who undergoes a liver resection and who has underlying chronic liver disease, such as fibrosis and/or cirrhosis. These patients have an increased risk of bile leaks and of developing new-onset ascites temporarily after surgery.

**Treating Fistulae** After recognition of a biliary fistula, the first-line approach for most surgeons is to perform endoscopic retrograde cholangiopancreatography (ERCP) and sphincterotomy. Sphincterotomy and stenting often stop smaller biliary fistulae because resistance is lowered at the sphincter, and bile flows along the path of least resistance. If a biloma has developed at the raw surface, drainage is needed. If ERCP is not effective, then the surgeon must consider a percutaneous transhepatic cholangiogram (PTC), cholangiocatheter placement to decompress the biliary system, and stenting the biliary duct. Usually these techniques successfully control the biliary fistula without having to return to the OR. Although I have little experience using glues via an endoscopic approach to control biliary leaks, more case reports are being published that describe the use of BioGlue®. Because it can be difficult to manipulate these glues in the endoscopes and through the catheters, stenting is a more viable option for some.
Hepatic Metastases: Detection

Case: A patient presents with stage II liver cancer. Which imaging modality is best for detecting metastatic lesions?

Recommendations: Either multidetector CT or MRI is best for detecting metastatic lesions. This is somewhat dependent on the center where the imaging is performed. For detecting liver metastases, the sensitivity for MRI ranges from 90% to 95%, and that for multidetector CTs is approximately 75%.

PET: PET is still in evolution when it comes to liver lesions and diagnosis. Some newer contrast agents have been developed, but they are still very new. PET is not great for detecting either colorectal metastases or primary cancers of the liver.

Functional Tests: With respect to functional studies of the liver, Japanese, Asian, and some European centers have done a lot of work in this area. In North America, studies to gauge function are not routinely done. Indocyanine green is what people use, although it is used more for clinical research than for practice-related applications. It is not uniformly being used partly because of how most surgeons are approaching liver function and analysis. First, before going to surgery, they are trying to determine if the functional liver remnant is adequate. If the functional liver remnant is small, then portal vein embolization may be performed before liver resection surgery. Although indocyanine green studies can help gauge the size of the functional liver remnant, an alternative is to evaluate some acute phase labs, such as coagulation factors, acid-base balance, etc. These are clinical indicators of how the liver is performing overall that can be added to the data obtained from of bilirubin and albumin assessments.

Preoperative Assessment: The Functional Liver Remnant

Before doing liver resections, we need some method to measure how much functional liver will remain with the patient. Volumetric measures can be done using preoperative CT or MRI images. Software analyzes the images and can help calculate total liver volume. Other software allows the surgeon to map out the surgical planes and determine the functional remnant. In patients with normal liver function, the functional remnant must be at least 20%, although most surgeons get anxious at 25% because we do not always know if patients truly have normal underlying liver. Many patients have a little bit of underlying liver disease, such as fatty liver, that may not be detected preoperatively. If the patient is about to undergo adjuvant chemotherapy or if you are concerned about some underlying liver disease, then a 33% remnant is preferred. Remember, the functional liver remnant is not the same as the total liver volume remnant.

Cirrhosis vs Functional Remnant: In the cirrhotic liver, the same tenants still apply. Resections on cirrhotic livers are challenging. First, you cannot perform large-volume liver surgery on a cirrhotic, even if they do not have portal hypertension, because it is hard to gauge what their residual function will be. You can certainly do segmentectomies on a cirrhotic liver, but even that will stress these livers. If a cirrhotic liver has significant portal hypertension, the liver will not do well after a hepatic resection. As a surrogate for portal hypertension, a platelet count of <100,000 platelets/mcL and a bilirubin of >2 mg/dL can be used to determine when the risk of complications is significant.

Hepatic Tumors: Treatment of Colorectal Liver Metastases

Case: A relatively healthy 60-year-old patient with normal liver function presents with nonobstructive cancer of the sigmoid colon and a single metastatic lesion in the right lobe of the liver. Are synchronous resections an option, or should the colon cancer resection and hepatic metastasis resection be staged?

Resection: A synchronous resection is okay. For nonobstructive cases, most surgeons would probably resect the colonic lesion first, and if that went smoothly, then they would resect the single lesion in the
liver in the same setting. However, you have a lot of options, and there is no bad option. If the liver lesion is 2 to 4 cm in size, the liver resection could be done before the colon resection. With small liver lesions, the resection will be a segmentectomy or a nonanatomical resection, which could certainly be done in stages with the colon resection if the patient is not progressing well during whichever operation is done first.

**Chemotherapy:** The best treatment for liver metastases is surgical resection. But if resection is not an option, a number of other therapies are available. Standard chemotherapy and neoadjuvant chemotherapy may be helpful. The FOLFOX regimens (oxaliplatin with fluorouracil and folinic acid) are the standard chemotherapies for colon cancer. New agents are currently being introduced for treating colorectal metastases, such as vascular endothelial growth factor (VEGF) inhibitors and small molecule targeted therapies.

**Ablation Therapies:** Ablation of colorectal hepatic metastases is another treatment alternative, such as cryotherapy, radiofrequency ablation (RFA), and microwave ablation. Cryotherapy has not been used as much as other ablative therapies for the liver, partly because patients develop systemic inflammatory response syndrome (SIRS) with cryotherapy. About 2000 to 2003, ablative therapy became a favored approach for treating patients with colorectal metastases. But the resulting data were not as good as expected, and the use of ablative therapies began to wane. In retrospect, I believe the data were poorer than expected partly related to selection criteria. Ablation is a functional resection. RFA is good, but it has its challenges. Early generation RFA devices had problems with heat sink. If the tumor is near a major blood vessel, the temperature of the tissue would not get as hot, leaving behind residual tumor. In addition, different radiofrequency generators each have their unique ablation geometric patterns.

For microwave ablation, the microwave energy sources are not susceptible to heat sink issues, and they can coagulate across smaller vessels. I think surgeons are now using ablation adjunctively when doing larger resections. Growing evidence in the literature shows that, for a single lesion with a colorectal metastasis, ablation is a good alternative when patients are not a good candidate for resection.

**Insufficient Future Functional Hepatic Remnant: Portal Vein Embolization**

**Case:** A patient needing to undergo a large hepatic resection is predicted to end up with a functional liver remnant that is too small. What are options for managing this patient?

**Recommendations:** Selective portal vein embolization is a good option for patients needing a larger resection resulting in concerns about functional remnant volume. This procedure is performed by interventional radiologists. Typically, in 2 to 4 weeks, the surgeon can see if appropriate hypertrophy of the potential remnant has occurred. Not all patients respond the same to portal vein embolization — sometimes selective portal vein embolization does not result in the clinical response you were looking for, so you could not proceed with the resection. A corollary to portal vein embolization is the staged embolization hepatectomy procedure. This procedure is not uniform across the hepatobiliary group, but in general, the portal vein to the remnant that will be removed is surgically ligated, and then the patient is closed up. This procedure produces a more robust hypertrophic response than does selective portal vein embolization. As such, if the hypertrophic response is adequate, the patient may be able to undergo completion hepatectomy after 48 to 72 hours, maybe even 5 days.

**Hepatocellular Carcinoma: LI-RADS**

The **Liver Imaging Reporting and Data System** (LI-RADS) has standardized the reporting and data collection of liver findings on CT and MRI for hepatocellular carcinoma (HCC). I believe it was developed to give better guidance to the transplant community. Typically, HCC is found in patients with chronic liver disease. Those at highest risk of HCC include patients with hepatitis infections,
chronic alcohol exposure, and/or nonalcoholic fatty liver disease. Cirrhosis is also associated with the development of HCC.

Need for LI-RADS: Typical HCCs have very classic radiographic features. Therefore, historically, we have said that no biopsy is needed for HCC in a cirrhotic liver to define whether we will treat the patient’s liver cancer. A subset of patients on the transplant list has HCC and has become a priority group on the list. While MRI experts were very confident of their diagnostic abilities, the transplant community needed more guidance and uniformity in defining HCCs and liver lesions in the spectrum of HCCs.

LI-RADS 1, 2: LI-RADS consists of 5 categories. LI-RADS 1 and 2 are either definitely or probably benign lesions. For example, the livers of patients with cirrhosis have nodules that are premalignant in their pathology. These patients need to be watched.

LI-RADS 3: Nodules classified as LI-RADS 3 may or may not need treatment, but we do not push for tissue diagnosis because the nodules are typically ≤2 cm. Unlike a metastasis, which is uniform across the entire lesion, a 2-cm LI-RADS 3 nodule, if sampled, might come back as “dysplastic liver tissue” or with some mitotic cells suspicious for HCC. Therefore, with LI-RADS 3 nodules, we recommend imaging follow-up at 3-month intervals. The lesion will quickly identify itself one way or the other.

Hepatocellular Carcinoma: Treatment

For the small- to medium-sized (<4 cm) HCC nodules, local ablative therapy for single nodules has actually been a very effective treatment with good 1-year no evidence of disease and patient survival. Beyond 1 year, the recurrence risk increases because the patient still has underlying chronic liver disease.

TACE: For larger lesions and multifocal lesions, the best data has come with transarterial chemoembolization (TACE), which typically uses cisplatin beads. While TACE is not curative for HCC, it is palliative. Sometimes a combination of TACE and ablation can be used to treat HCC.

TARE: Trans-arterial radio-embolization (TARE) uses radioactive beads to treat HCC. Because the beads are smaller, they are better for patients with a portal vein thrombus because they do not cause as much ischemia within the liver. Like TACE, TARE is not curative but is definitely very effective.

Chemotherapy: For systemic chemotherapy, sorafenib is the only available agent that has an indication. However, only a certain population can tolerate sorafenib. While it is not curative for HCC, sorafenib is life prolonging in a series of months. Some new ongoing trials may soon give us some new agents to help manage HCC.

Sclerosing Cholangitis

Sclerosing cholangitis can be diagnosed noninvasively via imaging using either endoscopic retrograde cholangiopancreatography (ERCP) or magnetic resonance cholangiopancreatography (MRCP). Both are very good at detecting primary sclerosing cholangitis (PSC). The diagnosis can also be linked to a strong
family history of autoimmune disorders. But MRCP is very good at detecting the early biliary changes of PSC, similar to what we used to do with ERCP. Therefore, fewer patients are getting early ERCPs. But once PSC is diagnosed via MRCP, patients will get an ERCP, usually with brushings because we are concerned about the potential development of cholangiocarcinoma if they have a dominant stricture(s).

**Value of Stents:** If the brushings come back negative for carcinoma but the patient still has evidence of biliary obstruction, biliary stents within the biliary system are not very helpful. Because PSC is typically a diffuse autoimmune disease of the biliary tree, getting up to the first-level bifurcation with a stent does not help or resolve the disease at the more distal levels — the radical branches — that tend to sclerose first.

**Transplant:** Patients who develop that level of disease and dysfunction really need to undergo transplantation. Often, these patients are very stable with their PSC, and then they appear to step off a cliff. For some time, they do not have stigmata or signs of liver disease until suddenly their bilirubin is 12. Even then, they are still functioning because they do not have ascites or a lot of the other complications of their portal hypertension. They tend to present with their jaundice. Often their admissions to the hospital are related to episodes of cholangitis related to the PSC.

**Posttransplant Recurrence:** After transplanting a patient and everything goes right, are they at risk for a recurrence of sclerosing cholangitis in the transplanted liver? Much like with any autoimmune disease, sclerosing cholangitis involves the body’s self-recognition against the biliary tree. Even though you are transplanting an allogeneic biliary tree, it is still a biliary tree. Therefore some patients with PSC, but not all patients, develop secondary sclerosing cholangitis at 10 to 15 years after successful transplantation. The immunosuppression administered for the transplant does help prevent this to some degree because not all transplanted patients develop secondary sclerosing cholangitis.

**Polycystic Liver Disease vs Solitary Hepatic Cysts**

Among patients with polycystic liver disease (PLD), it is difficult to determine what percent present as symptomatic because there are clearly more individuals in the population that have hepatic cysts than we recognize. Patients with PLD are usually diagnosed earlier than those with solitary hepatic cysts. Therefore, it is important to differentiate between hepatic cysts and PLD.

**Polycystic Disease:** PLD is a genetic autosomal-dominant disease, and patients with the disease will often be in a kindred. PLD is often associated with polycystic kidney disease, and these patients have hepatomegaly because they have cysts of all shapes and sizes distributed throughout the liver. They are more likely to be symptomatic because of the volume of their liver from their PLD. Patients with PLD can feel very good for a long time, and then suddenly they just feel like they are gaining weight and cannot lose it. Not everyone with PLD and hepatomegaly has large polycystic kidneys, and sometimes one can be more involved than the other. While replacing the polycystic liver is really the best thing for the patient from a symptomatic standpoint, it is harder to get a liver for them because they are not as sick as other patients on the transplant list.

**Managing Cysts:** Surgeons use different approaches for managing the cysts. For PLD cases with asymmetric distribution of the cysts and defined symptoms, “hepatoreductive surgery” with the more involved segment, based on symptoms, appears to be a good choice. With this approach, you must be aware that you are leaving normal liver and polycystic liver disease behind in the patient. What you are actually removing is mostly disease (mostly a conglomerate of cysts), and you are not removing much liver tissue. Therefore, you do not get the same liver regeneration response like you would if you were doing a left or right hepatectomy. Nonetheless, when they regenerate their liver, they will still have cysts there and those cysts can certainly grow in size.

**Solitary Cysts:** Do solitary liver cysts ever need surgery? This depends on cyst size, symptoms, and features. A simple cyst is just that: it has a pseudoepithelial lining, is not a cystadenoma, and contains a straw-colored fluid. Most surgeons manage these by first defining a good history. They next ask themselves if they feel confident that a cyst is the cause of the patient’s symptoms,
because usually they operate on those cysts for symptoms. If the patients are symptomatic and are good surgical candidates, then we can laparoscopically go in and fenestrate the cysts. This is associated with a low risk of recurrence if we are compulsive in fenestrating the cyst wall. The entire cyst does not need to be removed. The only situation in which the entire cyst must be removed is if the diagnosis is a cystadenoma, because these have a risk of malignant degeneration.

**End-Stage Liver Disease: MELD Score vs Child-Pugh Classification**

General surgeons have 2 tools available to them to help gauge surgical risk or postoperative mortality in patients with end-stage liver disease (ESLD). The Model for End-Stage Liver Disease (MELD) helps estimate mortality risk in patients with ESLD and can be used as a disease severity index to help prioritize organ allocation for transplant. The Child-Pugh classification (Child’s score) was developed to give surgeons a way to stratify surgical risk for patients undergoing portal hypertensive surgery or abdominal surgery. The Child’s score has 3 broad categories: A, B, and C. Compared to the Child’s score, MELD has less subjectivity. In the Child’s score, ascites and encephalopathy (both subjective assessments) are included in the grading system. The MELD score is being driven by bilirubin, creatinine, and INR — purely objective data. Therefore, the MELD score was developed for the risk of mortality after transjugular intrahepatic portosystemic shunt (TIPS) for patients with chronic liver disease. Although the Child’s score and MELD were developed for slightly different indications, both help with risk stratification in a complementary sense. I think many surgeons probably use MELD more than the Child-Pugh scoring system probably because the MELD app is easy to use, needing only to enter labs values. In addition, most MELD apps are free and can be found on any of the medical software apps that you have. Based on MELD scores, patients with scores <12 are relatively low-risk liver surgery patient. If the MELD score is >20, the risk of complications increase and the surgeon must balance the benefits of the needed procedure with the risks. Most hepatologists would say that it is best not to operate on somebody with a MELD score >14 if a hepatologist is not caring for the patient. These patients need to be under a hepatologist’s care in case the patient decompensates — under a hepatologist’s care, they would have already been looked at from a transplanter’s perspective.

**Liver Failure: Managing Ascites and Encephalopathy**

**Case:** A patient presents with a poor MELD score after undergoing cholecystectomy. Are beta-blockers used routinely in patients with liver failure?

**Recommendations:** The indication for beta-blockers is based on the presence of gastroesophageal varices and portal hypertension. The term “liver failure” is a very broad umbrella term — just having liver failure and the development of ascites would not be an indication to administer beta-blockers. This is a good example of why patients with poor MELD scores should be seen by a liver specialist before undergoing surgery. Hepatologists routinely perform an upper endoscopy to grade the varices to determine if the patient should be on beta-blocker therapy before any procedures are performed. When managing a patient with liver failure after surgery, the key is to balance symptom management and liver management, to ensure that infections are minimized, and to control ascites.

**Ascites:** Patients with liver failure generally present with either the development or worsening of ascites and/or the development and worsening of encephalopathy. Ascites management involves a combination of drainage and fluid management. How much saline are you giving the patient (more saline equals more ascites)? Is the patient hypoalbuminemic and in need of albumin replacement? You want to drain the ascites to a point, but if you keep draining them, they will continue forming more ascites. Therefore, these patients need to be placed on diuretic therapy to control the formation of the ascites.
In my practice, I generally do not drain new-onset postoperative ascites if the patient does not have tense ascites. Instead, I manage these patients via a “closed box” approach using diuretics.

**Encephalopathy:** Several medications are available to manage encephalopathy. Lactulose is a very effective medication. Hepatologists also use Xifaxan®, an oral agent, either alone or in combination with lactulose to help control encephalopathy.

**Portal Vein Thrombosis in Cirrhotic Livers**

**Case:** A cirrhotic patient presents with portal vein thrombosis (PVT). How should this be managed?  
**Recommendations:** Most patients with chronic liver disease who develop PVT do so in a slow indolent manner. It is not like the acute portal vein thrombosis in which sick patients present with acute venous congestion of the intestine. Usually with treatment of acute portal vein thrombosis, the portal veins go down slowly or more progressively. The challenge that we see is in trying to anticipate PVT before thrombosis and deciding whether the patient should be anticoagulated, which has its own set of challenges because these patients have portal hypertension, are thrombocytopenic, and may have varices. Your goal is to keep their portal vein open because this will reduce their incidence of encephalopathy. This also helps from a transplant standpoint, but you have to balance the use of anticoagulants with the risks of bleeding.

**Spontaneous Bacterial Peritonitis**

Spontaneous bacterial peritonitis (SBP) is an acute bacterial infection of ascitic fluid as well as a well-known complication of cirrhosis. The key to managing SBP is the microbiology. SBP is generally associated with very classic microorganisms, such as *E. coli*, *klebsiella*, and *enterococcus*. A certain percentage of SBP cases are atypical (culture-negative but cell-count-positive). In other cases, the culture is positive but atypical microbial sources are identified, such as *Strepococcus viridans*, or some of the fungal agents, such as *Clostridia*. When you identify anything that is rare in this space, then you have to question whether this is SBP or is it a secondary bacterial peritonitis, which is devastating for a patient with cirrhosis. As we have become more aggressive in managing cirrhotics, especially in a pretransplant position where they have had multiple paracenteses, I used to be relatively confident of the SBP diagnosis when a single bug was identified on culture. But now, the culture results sometimes come back with both a gram-negative and a gram-positive organism — a polymicrobial SBP. Polymicrobial SBPs are concerning for potential microperforation, which is more complex than a straightforward SBP. The fungal SBPs are also challenging because the duration of antimicrobial therapy must be extended before those patients are ready and viable for transplant.

**Hepatic Varices**

**Case:** A patient presents with variceal bleeding. What is the role of transjugular intrahepatic portosystemic shunts (TIPS) in these patients?  
**Recommendations:** If local source control can be done for a variceal bleed, the endoscopist will certainly do that with clips or injection. TIPS has definitely taken hold and plays a large role in the management of acute variceal bleeds now that most centers, certainly tertiary care centers, have strong interventional radiology support. Some patients with variceal bleeding undergo TIPS once and that TIPS stays patent and trouble-free until the patient comes up for transplant. However, other patients develop either in-stent stenoses or partial thrombosis of their TIPS stent. These cases must have their TIPS stent redilated or manipulated. At a center that does liver transplants, the interventional radiologists are generally very facile at keeping the stents within the liver rather than going all the way down the portal vein to the superior mesenteric vein, which makes transplant more difficult.