How to avoid injury to bowel during laparoscopy

Lessons in timely recognition of laparoscopy-related bowel injury

⤵ Be diligent about inspecting the bowel in the area of surgical intervention and quick to suspect intestinal injury when the patient doesn't improve postop

Michael S. Baggish, MD

Dr. Baggish is Chairman of the Department of Obstetrics and Gynecology at Good Samaritan Hospital and Director of Obstetrics and Gynecology Residency Training for TriHealth Hospitals in Cincinnati. He is also Professor of Obstetrics and Gynecology at the University of Cincinnati College of Medicine and a Course Director of the 2008 Pelvic Anatomy and Gynecologic Symposium (PAGS), to be held December 11–13 at the Wynn Las Vegas. For more information go to: www.PAGS-cme.org

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In Part 1 of this article (page 46), I outlined circumstances in which abdominal adhesions should be anticipated and described strategies to prevent intestinal injury during operative procedures. Here, I describe ways to identify intestinal injury as soon as possible after it occurs, which is vital to prevent serious sequelae such as sepsis and even death.

During operative laparoscopy, a quick search for injury through the laparoscope cannot assure any surgeon that the intestinal wall has not been seriously denuded. A damaged muscularis—even if it is not recognized as transmural injury—may subsequently rupture if it is not appropriately repaired intraoperatively.

Following dissection of adhesions, irrigate the neighboring intestine with sterile saline, and perform a detailed inspection of the intestine to ascertain integrity of the bowel wall. The color of the intestine is important, as it can indicate whether the abundant vascular supply has been compromised. Include a detailed description of the intestines in the operative note.

Avoid stapling or vascular clips when repairing any wound; careful suturing is preferred.

Why early diagnosis is critical

The most favorable time to diagnose an iatrogenic intestinal perforation is within the intraoperative period. Prompt recognition and repair of bowel perforation offers several advantages:

- A second or third operation is less likely (<50% probability)

Effects of intestinal perforation?
Infection, fluid-electrolyte imbalance, sepsis syndrome

The principal derangements that arise as a result of bowel perforation are infection and fluid-electrolyte imbalance and their sequelae. Intestinal fluid and feces contain a variety of bacteria, such as *Escherichia coli*, *Enterococcus*, *Klebsiella*, *Proteus*, *Pseudomonas*, and *Clostridium*, to name a few. These bacteria produce toxins that facilitate entry of bacteria into the circulation and contribute to a downward spiral of events, referred to as sepsis syndrome, as well as intra-abdominal abscess:

1. Contamination of the abdominal cavity leads to inflammation of the peritoneum

2. In turn, subperitoneal blood vessels become porous, causing interstitial fluid to leak into the third space

3. Paralytic ileus and an accumulation of intra-abdominal fluid push the diaphragm upward, lowering the capacity for lung expansion within the thorax and contributing to partial lung collapse

4. Fluid of inflammatory origin may accumulate in the chest as pleural cavity effusion.

A number of progressive complications are predictable, but may occur at variable intervals after the initial perforation. The most frequent complications associated with colonic injuries are:

▶ peritonitis (98% of cases)
▶ ileus (92%)
▶ pleural effusion (84%)
▶ colostomy (80%)
▶ intra-abdominal abscess (78%).

The most common sequelae after small-bowel perforation are:

▶ peritonitis (100% of cases)
▶ ileus (89%)
▶ intra-abdominal abscess (63%)
▶ pleural effusion (59%).[1]

- The risk of abdominal sepsis is decreased.
- The volume of peripheral injury to the intestine is reduced.
- The patient can be followed for subsequent complications more precisely, permitting earlier diagnosis, more timely and effective treatment, and lower morbidity.

If the diagnosis is missed intraoperatively, then early postoperative diagnosis—less than 48 hours after the termination of surgery—is infinitely more beneficial for the patient than late diagnosis. Clearly, the longer diagnosis is delayed into the postoperative period, the greater the risk of serious morbidity and associated mortality.

The 130 intestinal injuries reported by Baggish reflect the clinical significance of timely diagnosis.[1] Seventy percent of small bowel and 51% of large bowel perforations were correctly diagnosed more than 48 hours postoperatively. Sepsis was present in a majority of these cases at the time of diagnosis.

Reasons for diagnostic delay

- The gynecologic surgeon fails to place intestinal injury at the top of the differential diagnosis.

- A surgical consultant is delayed in making a correct diagnosis. Surgeons have less experience with perforation than do gynecologists, and invariably consider the postoperative abdominal problem to be ileus or intestinal obstruction. The presence of postoperative pneumoperitoneum is incorrectly thought to be lingering CO_2 gas from the initial laparoscopy rather than air from a perforated viscus.

- Ancillary diagnosis confuses the primary physician. Pleural effusion, chest pain, and tachypnea are usually thought to indicate pulmonary embolism; as a result, the gynecologist and consulting pulmonologist focus on pulmonary embolus and deep-vein thrombosis. Only a spiral computed tomography (CT) scan, a ventilation perfusion (VQ) scan, or arteriogram quickly rules pulmonary embolus in or out. Peritonitis associated with ileus or third-space fluid leakage resulting in diaphragmatic elevation also creates pleural effusion, tachypnea, and dyspnea.

Presumptive diagnosis is critical

Definitive diagnosis of intestinal perforation happens at the operating table under direct vision and is corroborated by the pathology laboratory if bowel resection is performed. However, presumptive diagnosis helps overcome inertia and gets the patient to the operating room sooner.

The process by which the presumptive diagnosis is made is the most important issue in this article. The shorter the process, the lower the patient's morbidity, and vice versa.

Look for steady improvement. Worry when it is absent

After any laparoscopic operation, the postoperative course should be one of steady clinical improvement. When a patient deviates from this model, the foremost presumptive diagnosis should be laparoscopy-associated injury, and the intestine should top the list of organs that may be injured. Other diagnoses should be subordinate to the principal presumptive diagnosis; these include ileus, bowel obstruction, pulmonary embolus, gastroenteritis, and hematoma, to name a few.

I do not mean to imply that a potentially life-threatening complication such as pulmonary embolus should not be ruled in or out, but that the necessary imaging should be performed in a timely fashion. The abdominal-pelvic CT scan will offer clues to the presence of free air, free fluid, air-fluid levels, and foreign bodies. It also is useful in detecting intra-abdominal—specifically, subphrenic—abscess. If necessary, a VQ scan or spiral CT scan can then be performed without delaying the diagnosis of the primary intra-abdominal catastrophe responsible for the pulmonary symptoms.

In the opening case, before making an improbable presumptive diagnosis, the surgeon should have questioned why an otherwise healthy woman would coincidentally develop gastroenteritis after laparoscopic surgery. The same can be said for diagnoses of intestinal obstruction or vascular thrombosis involving the intestinal blood supply.

Typical presentation of the injured patient

An injured patient does not experience daily improvement and a return to normal activity. Instead, the postoperative period is marked by persistent and worsening pain, often compounded by nausea or vomiting, or both. The patient may complain of fever, chills, weakness, or simply not feeling normal. Breathing may be labored. As time elapses, the symptoms become worse.

Reports of more than one visit to an emergency care facility are not uncommon. When examined, the patient exhibits direct or rebound tenderness, or both. The abdomen may or may not be distended, but usually is increased in girth. Bowel sounds are diminished or absent.

Vital signs initially reveal normal, low-grade, or subnormal temperature, and tachycardia, tachypnea, and normal blood pressure are typical. As time and sepsis progress, however, fever and hypotension develop. Most other symptoms and signs become progressively more abnormal in direct proportion to the length of time the diagnosis is delayed.

Seminal laboratory values for sepsis include a lower than normal white blood cell (WBC) count, elevated immature white-cell elements (e.g., "bandemia"), elevated liver chemistries, and an elevated serum creatinine level.

Mortality is most often the result of overwhelming and prolonged sepsis, leading to multiorgan failure, bleeding diathesis, and adult respiratory distress syndrome.

Among 130 laparoscopic surgical cases complicated by bowel injury and reported by Baggish, sepsis was diagnosed in 100% of colonic perforations and 50% of small-bowel perforations when the diagnosis was delayed more than 48 hours after surgery.[1]

TABLE 1 lists the signs and frequency of sepsis in these 100 cases, and **TABLE 2** collates the signs and symptoms that were observed. Peritoneal cultures obtained at the time of exploratory laparotomy revealed multiple organisms (polymicrobial) in 60% of cases.

Concurrent injuries to neighboring structures

A number of collateral injuries may occur in conjunction with intestinal perforation, depending on the location of the trauma. The most dangerous combination includes indirect laceration of one of the major retro-

FAST TRACK

When a patient fails to improve after laparoscopy, the first presumptive diagnosis should be laparoscopy-associated intestinal injury

peritoneal vessels. A through-and-through perforation of the cecum can also involve one or more of the right iliac vessels. A trocar perforation of the ileum may continue directly into the presacral space or pass above it and penetrate the left common iliac vein or aorta. Similarly, perforation of the sigmoid colon may penetrate the left iliac vessels.

Careful inspection of the posterior peritoneum for tears and evidence of retroperitoneal hematoma is required to avoid missing a serious collateral injury. More likely, however, is a penetrating injury to the small bowel presenting with collateral mesenteric damage and compromise of the blood supply of an entire segment of bowel. The ureter and bladder may also be injured when dissection along the pelvic sidewall, or a trocar thrust, deviates to the right or left of midline. In thin patients, the stomach may be perforated as well as the small intestine or transverse colon.

In one memorable case, a primary trocar penetrated the omentum, injuring several underlying structures. In its transit, the trocar passed through both the anterior and posterior walls of the duodenum and finally entered the superior mesenteric artery. The gynecologic surgeon performing the laparoscopic tubal ligation failed to recognize any of these injuries. The patient went into shock in the recovery room and was returned to the operating room. Fortunately, a transplant surgeon from a neighboring theater was immediately available to consult and repair the damage.

Another danger: intestinal ischemic necrosis

Abnormalities in splanchnic blood flow are sometimes caused by elevations in intra-abdominal pressure. Caldwell and Ricotta inflated the abdomens of nine dogs and reported a significant reduction of blood flow to omentum, stomach, duodenum, jejunum, ileum, colon, pancreas, liver, and spleen, but not to the adrenal glands.[2] The splanchnic flow reductions essentially shunted blood away from abdominal viscera with auto-

TABLE 1 **Frequency of signs of sepsis among 130 patients with colon or small-bowel injury**

Sign	Colon (49 patients)	Small bowel (81 patients)
Normal or subnormal temperature	30*	41*
Fever	19	40
Tachycardia	31	44
Tachypnea	30	40
Hypotension	21	15
Anemia	38	51
Depressed WBC count	20	18
Elevated WBC count	24	32
Bandemia	25	30
Elevated creatinine and blood urea nitrogen levels	12	5

*Number of patients.

Source: Baggish[1]

TABLE 2 **Watch for signs and symptoms of intestinal injury**

Symptom	Sign
Abdominal pain	Direct or rebound tenderness
Bloating	Abdominal distension
Nausea, vomiting	Diminished bowel sounds
Fever, chills	Elevated or subnormal temperature
Difficulty breathing	Tachypnea, tachycardia
Weakness	Pallor, hypotension, diminished consciousness

Source: Baggish[1]

transfusion to the heart, lungs, and systemic circulation.

Eleftheriadis and colleagues studied 16 women randomized to laparoscopic versus open cholecystectomy.[3] Significant depression of the hepatic microcirculation during the period of CO_2 gas insufflation was noted in the laparoscopy cohort but not in the control group. Gastric mucosal ischemia also was observed in the laparoscopy group.

Several case reports of catastrophic intestinal ischemia have appeared in the literature (1994–1995).[4–7] These articles have

mainly involved laparoscopic upper abdominal operations in elderly people.

Recently, however, Hasson and colleagues reported a case of possible ischemic necrosis of the small intestine following laparoscopic adhesiolysis and bipolar myolysis.[8] The authors emphasized that CO_2 pneumoperitoneum reduces splanchnic blood flow, predisposing the patient to ischemia, but that ischemia with infarction requires an underlying vasculopathy or inciting factors such as traction on a short mesentery, atherosclerosis, or thrombosis.

A high index of suspicion for bowel ischemia following laparoscopic surgery should occur when, postoperatively, a patient experiences inordinately severe abdominal pain associated with tachypnea, tachycardia, and alterations in the WBC count. A paucity of physical abdominal signs in the early phases of this disorder should alert the clinician to the possibility of bowel ischemia.

Diagnosing and treating ischemia

A CT scan with contrast can suggest ischemia, but angiography is usually required for definitive diagnosis.

Treatment begins with infusion of papaverine into the intestinal vasculature via angiography cannula. In some cases, anticoagulation may be indicated. Surgery by laparotomy is clearly indicated for patients who fail to respond to vasodilatation measures.

This condition can be ameliorated by intermittent intraoperative decompression of the abdomen. Avoiding prolonged CO_2 pneumoperitoneum and a lengthy laparoscopic operation also may diminish the risk of intestinal ischemia. ●

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would be to subtract those expenses that are eliminated at retirement—the mortgage and the costs of raising children, I would hope.

Running the numbers

Now you are armed with just about all you need. To take an example, suppose you have determined that you're spending roughly $100,000 a year after taxes. If your portfolio will return 6% (net, 4% after taxes), then simply multiply by 25 (the inverse of 4%) to come up with the portfolio you'll need: $2.5 million. If you want to be very conservative, assume a 4% return (3% net), and multiply by 33 to yield a needed retirement portfolio of $3.3 million.

This is, of course, a very simple analysis for those close to retirement. It does not consider the effect of inflation, Social Security (which I do not like to count when calculating), pensions, etc. It also does not answer the question of how much to save now.

Many financial Web sites do allow you to calculate how much to save, however. A site that covers the gamut of calculations is www.dinkytown.com. One piece of information it asks for that is hard to define is the expected return on investments during preretirement years. With a portfolio tilted toward growth, using a pretax return of 7% to 9% is a safe assumption. Running the calculation at both 7% and 9% sets the parameters for how much you need to be saving now.

Hopefully, the number you come up with will not break the bank for you. But if it does, there is always that deluxe mobile home. ●