Gastrointestinal Perforation and the Acute Abdomen

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The acute abdomen accounts for up to 40\% of all emergency-surgical hospital admissions and is considered in the differential in the more than 7 million visits to the emergency department annually for abdominal pain in the United States [1–3]. A large percentage of these cases are secondary to perforation or impending gastrointestinal perforation [1–3]. Gastrointestinal perforation causes considerable mortality and usually requires emergency surgery. Mortality of secondary peritonitis was as high as 90\% in the early Twentieth century and is still 30\% to 50\% despite advances in antibiotics, surgical technique, radiographic imaging, and resuscitation therapy [4,5]. Rapid diagnosis and treatment of these conditions is essential to reduce the high morbidity and mortality of late-stage presentation. Successful treatment requires a thorough understanding of the anatomy, microbiology, and pathophysiology of this disease process and in-depth knowledge of the therapy, including resuscitation, antibiotics, source control, and physiologic support.

The gastrointestinal tract is a continuous luminal (hollow) viscera composed of named organs based on physiologic or anatomic variations that result in functional differences in ingestion, digestion, processing, absorption, and elimination of nutritional substances. Gastrointestinal organs are traditionally categorized according to their embryonic derivation: Structures proximal to the ligament of Treitz are derived from the embryonic foregut, structures from the ligament of Treitz to the hepatic flexure of the colon from the embryonic midgut, and structures distal to the hepatic flexure from the embryonic hindgut [6]. Although this classification is useful to understand the developmental anatomy, vascular anatomy, and pain...
perception distribution of gastrointestinal organs, it is not useful to determine the severity and rapidity of disease progression in gastrointestinal perforation.

The relative proximal to distal location of an insult is useful to understand the clinical presentation and progression of disease. From proximal to distal, the gastrointestinal tract includes the stomach, duodenum, jejunum, ileum, appendix, colon, and peritonealized rectum. The gastrointestinal lumen serves as a space to contain, process, and selectively absorb from the extracorporeal environment. Breach of the gastrointestinal wall causes intra-abdominal contamination with peritonitis or abscess formation [4]. The type and degree of peritoneal contamination depends on the site, size, and duration of the perforation and on the physiologic state, including the time from the last meal, administration of a mechanical bowel preparation before the perforation, coexistent diseases, and the presence or absence of an ileus or bowel obstruction with accompanying bacterial overgrowth [7–13]. These factors affect the relative degree and type of bacterial and fungal contamination from perforation. The anatomic site of perforation significantly affects the type and burden of enteric contamination [8,9,12,13]. The degree and type of microbiologic colonization of the gastrointestinal tract varies for different gastrointestinal organs and depends upon the local microenvironment. Microbiological colonization increases from proximal to distal, with the stomach showing the lowest number of viable microorganisms per cubic centimeter of luminal contents. Despite individual variation, the microbiologic content of the normal, healthy stomach and duodenum is less than $10^3$ organisms per gram of luminal contents due to a hostile local milieu from acidic, biliary, and pancreatic secretions [12,13]. The microbiologic load progressively increases from approximately $10^4$ organisms per gram of luminal contents in the proximal jejunum to $10^7$ organisms per gram in the terminal ileum [12,13]. The colon has the highest microorganism burden, with loads as high as $10^{12}$ organisms per gram of luminal content [12,13]. The composition of the microflora of the gastrointestinal tract varies greatly. Gastric flora are composed of yeast, aerobic bacteria (predominantly *Staphylococci*, *Streptococci*, and *Hemophilus* species), and anaerobic bacteria (predominantly *Bacteroides*, *Veillonella*, and *Bifidobacterium* species). Gastric anaerobes outnumber aerobes by about 1000-fold. The relative frequency of aerobes progressively increases along the small bowel, with gram-negative aerobes becoming the predominant organisms in the terminal ileum. The microfloral load and composition dramatically and abruptly changes between the terminal ileum and the colon. Colonic anaerobes outnumber aerobes by up to 1000-fold, with the predominant genera consisting of *Bacteroides*, *Bifidobacterium*, *Eubacterium*, *Clostridium*, *Lactobacillus*, *Fusobacterium*, and a limited variety of gram-positive anaerobes [12,13]. Understanding the characteristic microflora of each gastrointestinal organ is clinically important in selecting antibiotics for secondary peritonitis and potential sepsis.
The microorganism load is inversely related to the relative toxicity of organ fluid composition. The stomach and duodenum contain acidic contents or erosive biliary and pancreatic fluid, whereas the distal small bowel and colon contain a relatively neutral environment [12]. Organ-specific fluid composition affects bacterial load and bacterial species composition, which can lead to different initial presenting symptoms from a perforated viscous that may be diagnostically helpful. Patients who have gastric and duodenal perforation tend to present with highly acute pain due to a rapid chemical peritonitis, often followed by a systemic inflammatory response syndrome (SIRS), which can lead to rapid clinical deterioration [5,14,15]. Patients often recall the exact time of symptom onset. The perforation may progress to an infected peritonitis and sepsis in untreated patients or in patients who have late-stage presentations [5]. Colonic perforations may present without immediate perforation-associated pain and tend to have a slower clinical progression, with the development of a secondary bacterial peritonitis or localized abscess formation [16,17] partly due to the relatively neutral and nonerosive nature of the chemical environment within the colon [12]. With some exceptions, peritoneal contamination from intraluminal colonic contents progressively leads to purulent or fecal peritonitis or to the development of an intra-abdominal abscess. Few of the more than 500 different microfloral species in the colon can survive outside their normal environment [12,18]. In fact, only 22 genera of bacteria were isolated from cultures of intra-abdominal fluid collected among 255 patients who had secondary bacterial peritonitis [5,19]. Over 70% of isolates were from the genera *Bacteroides, Streptococcus, Escherichia, Peptostreptococcus*, and *Fusobacterium* [5,18,19].

**Mechanisms of gastrointestinal perforation**

All causes of gastrointestinal tract perforation involve loss of gastrointestinal wall integrity and the release of intraluminal contents into the normally sterile peritoneal cavity. The numerous underlying causes are categorized according to the initial pathophysiology as (1) penetrating foreign body perforation, (2) extrinsic bowel obstruction, (3) intrinsic bowel obstruction, (4) direct loss of bowel wall integrity without foreign body perforation, (5) gastrointestinal ischemia, and (6) infection (Table 1).

Gastrointestinal perforation secondary to a penetrating foreign body can result from the ingestion of sharp, pointed, or jagged objects [20,21], such as chicken bones, fish bones, nails, razor blades, and tooth picks [22–25]. Although these perforations can occur anywhere along the gastrointestinal tract, they more commonly occur in the small intestine and colon. More frequently, foreign body perforation involves external penetrating trauma from knife injuries, gunshot wounds, or falls onto sharp protruding objects [26]. Iatrogenic perforations have significantly increased recently due to the
greater use of invasive diagnostic and therapeutic medical procedures [7,27,28], including upper endoscopy, colonoscopy, interventional radiologic guided tissue biopsy and fluid drainage, paracentesis, nasogastric tube insertion, laparoscopy, and laparotomy [7,27,28]. The risk of gastrointestinal perforation from colonoscopy or upper endoscopy markedly increases with therapeutic procedures and with diagnostic procedures that involve tissue sampling [27].

Gastrointestinal perforation from extrinsic obstruction may be caused by mass effect from benign or malignant neoplasms, including primary gastrointestinal wall neoplasms such as gastrointestinal stromal tumors, leiomyomas, leiomyosarcomas, lymphomas, and duplication cysts [29–31]. Extrinsic compression may also occur secondary to nongastrointestinal neoplasms located adjacent to the compressed segment of bowel or metastatic to the gastrointestinal tract and associated mesentery [32,33]. Other common causes include surgical adhesions, malrotation, volvulus, and intra-abdominal or fascial wall herniation [34–36]. These conditions can cause loss of gastrointestinal wall integrity via a single point of obstruction or a closed loop obstruction. Compression at a single point can produce complete bowel obstruction. In this case, the pathophysiology involves dilation of the proximal bowel and a progressive increase in venous congestion followed by arterial stasis, ischemia, necrosis, and loss of mural integrity leading to

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Table 1
Classification of gastrointestinal perforations
perforation [37]. With a single point of distal compression this series of events is not a certainty, but it is more likely with very distal obstruction. If a point of distal fixation or compression leads to a gastrointestinal twist or volvulus, this may cause a closed loop obstruction [34,36,37]. Closed loop obstruction can abruptly block vascular inflow to the compromised segment with rapid progression of tissue ischemia, necrosis, and perforation. Alternatively, a closed loop obstruction can evolve slowly through mucosal secretion into a closed luminal space, luminal distention and pressurization, venous congestion, arteriolar stasis, and thrombosis, followed by tissue ischemia, necrosis, and perforation [38]. The pathophysiology of the latter mechanism is similar to non–closed loop obstructions but occurs with greater certainty and more rapidity.

Intrinsic gastrointestinal obstruction results from intraluminal obstruction from neoplasms, phytobezoars, ischemic or radiation-induced strictures, and Crohn’s disease–related strictures [39–41]. The most common causes of intraluminal obstruction with perforation is appendicitis and diverticulitis [38,42]. Congenital and acquired gastrointestinal diverticuli and the vermiform appendix are blind-end intestinal outpouchings [38,42–44]. The small lumens of these structures are subject to obstruction by fecal impaction, neoplastic obstruction, and lymphoid hyperplasia [38,42–44]. Intrinsic obstructions generally manifest the same pathophysiology as extrinsic nonclosed loop obstructions with progressive bowel distention, venous congestion, and arteriolar stasis followed by ischemia, necrosis, and loss of mural integrity [38]. As with nonclosed loop obstruction, perforation is unusual and more commonly occurs with distal obstructions. Obstruction of the blind loop segments of bowel with diverticulitis and appendicitis creates a closed loop bowel obstruction with secretion into a closed luminal space and a more certain and rapid onset of pathology [16,38,42].

Gastrointestinal wall integrity can be breached without distal obstruction or trauma from preexistent diseases or treatments for diseases involving the gastrointestinal wall. Common examples of the former mechanism include peptic ulcer disease and Crohn’s disease [45–49]. Since the discovery of *Helicobacter pylori*, peptic ulcer perforations can be categorized as an infectious cause of gastrointestinal perforation, but up to 50% of patients who have duodenal and gastric ulcer perforations are *H pylori* negative [48–51]. These disease processes lead to local pathologic changes of the normal mural anatomy, including loss of normal mucosal integrity, local inflammation, and eventually focal perforation [52]. Focal loss of normal mucosal integrity may also result from neoplastic growth or from adjuvant or neoadjuvant therapy [53–57]. Perforation with a primary neoplasm is usually due to local tumor infiltration, replacement or displacement of the normal histology, tumor expansion, and overgrowth of the vascular blood supply, followed by ischemia, necrosis, and eventual perforation [54–57]. Malignant perforation can result from rapid tumor lysis from adjuvant or neoadjuvant therapy with loss of integrity of the involved gastrointestinal wall [54–57].
Ischemia is a relatively common cause of gastrointestinal necrosis and perforation in elderly and critically ill patients. The major direct mechanisms of ischemia are embolic occlusion, severe arterial stenosis, systemic hypotension, or venous outflow obstruction [58,59]. Underlying pathologic states that lead to proximal arterial thrombus formation can cause release of thrombotic debris to smaller-caliber distal vessels with embolic occlusion [58]. Common sources of emboli are thrombi located in the aorta or heart chambers. The site of thromboembolic occlusion is determined by the size of the embolus and the caliber of the vessel in which it becomes lodged [58,60]. Because emboli often are small fragments of a thrombus, they commonly obstruct distal arteries and cause relatively short segmental ischemia and necrosis. Large emboli or critical stenosis may occlude the celiac, superior mesenteric, or inferior mesenteric arteries or their major proximal branches [58,60]. This may produce extensive gastrointestinal ischemia, depending on the degree of coexistent vasculopathy or development of collateral circulatory flow. Ischemia rarely occurs in the stomach because of its highly redundant blood supply. Thromboembolic events may abruptly occlude arterial inflow and rapidly produce tissue ischemia, necrosis, and perforation [58,60].

Visceral arterial stenosis, usually of the celiac, superior mesenteric, or inferior mesenteric arteries, typically develops at or near their origin from the aorta. These vessels slowly and progressively stenose; this may cause complete obstruction [58]. Because the process slowly evolves, the body has time to develop a collateral blood supply derived from the other main visceral arteries. Due to vascular collaterals and redundancy, occlusive stenosis of at least two of the three main visceral vessels is usually required to cause bowel necrosis via this process. Severe multivessel stenosis frequently predisposes to obstructive flow through the stenotic region when a second event, such as hypotension or embolic occlusion, occurs that completely blocks or further reduces the already compromised blood flow [58,61,62]. The ensuing events are the same as described for thromboembolic occlusion of relatively normal vessels but tend to produce extensive bowel necrosis due to the proximal nature of the occlusion [58].

Gastrointestinal ischemia can develop from marked and prolonged hypotension secondary to sepsis, congestive heart failure, acute myocardial infarction, and hypovolemic shock [58–62]. These diverse insults can lead to intestinal ischemia, necrosis, and perforation [58–62].

Venous outflow can be obstructed, most commonly from thrombotic occlusion of the portal or superior mesenteric veins [58–60]. This obstruction can be well tolerated due to compensatory mechanisms or can produce severe venous congestion of a bowel segment, leading to arteriolar stasis and thrombosis followed by ischemic perforation, similar to the mechanism described for distal bowel obstruction [58–60].

Numerous microorganisms have been associated with gastrointestinal perforation, including Clostridium difficile, Salmonella typhi, Mycobacterium
tuberculosis, and cytomegalovirus [63–69]. Although infection may lead to gastrointestinal perforation in any host, perforation is more common in immunocompromised patients, patients with poor nutritional states, and critically ill patients [64,65,69,70]. The injury varies in severity depending upon the virulence factors associated with the specific infectious agent. Many virulent bacterial organisms, including *C. difficile* and *S. typhi*, produce toxins that contribute to the pathologic insult to the gastrointestinal wall [63–65,69,70]. The pathophysiology of gastrointestinal perforation from infection includes a severe inflammatory reaction leading to ileus, bowel dilatation, and eventual loss of gastrointestinal wall integrity [63–70].

**Clinical evaluation**

Suspected gastrointestinal perforation should be excluded in patients who have an acute abdomen. Although the term “acute abdomen” sometimes refers to conditions requiring urgent surgery, the term most often refers to any patient who develops abdominal pain over a relatively short time interval. There are many causes of acute abdominal pain, and many of them do not require surgery. It is important to rapidly differentiate between disease processes that require urgent or emergent surgery versus those that may be treated conservatively. A thorough history and physical examination is critical [14,71,72]. Although the history and physical examination may fail to identify the specific anatomic source of pathology with gastrointestinal perforation, they can rapidly determine the patient’s acuity, differentiate likely surgical emergencies from benign disease processes, evaluate surgical risk, guide supplemental diagnostic testing and interventions, and tailor the therapeutic preoperative optimization to the individual patient. The history and physical examination may have to be abridged in patients who are obtunded or in extremis, but a limited history of key information should be obtained from the patient, family, or bystanders when possible.

The history should be detailed and complete and should focus on the history of the present condition, particularly the presenting symptoms, time course of events, and associated symptoms. When evaluating acute abdominal pain, inquiry into the antecedent events; the location, quality, duration, nature, and migration or extension of the pain; and modifiers of the pain is important for determining the diagnosis [71–73]. Other important points include symptoms of nausea, vomiting, anorexia, change in bowel habits, weight loss, syncope, dizziness, and dysuria. A complete medical history to include prior surgery, current medications and allergies, a family history, and a review of systems should be obtained to refine the differential diagnosis and guide diagnostic testing. Aspects of the history pertinent to the acute abdominal pain should be emphasized.

Pain is universal in patients who have an acute abdomen. It usually represents the reason for seeking medical attention [72,74]. Descriptions of the pain
can help diagnose the etiology of the abdominal pain. Important descriptors include the time course of the pain (e.g., When did it start? Is it recurrent? Has it progressed, diminished, or changed from the time of onset?). Hyperacute and immediately severe pain is consistent with gastrointestinal perforation but may occur with ureteral obstruction or biliary colic. What is the quality of the pain? Is it sharp, stabbing, dull, or colicky? Intermittent colicky pain is unusual in patients who have hollow viscous perforation or impending perforation [72,74]. Determine the severity of the pain based on a scale from 1 to 10. Determine radiation or migration of the pain. Where the pain started may help in determining the etiology. Foregut pathology tends to present with epigastric pain, midgut pathology tends to present with periumbilical pain, and hindgut pathology tends to present with infraumbilical pain. With time, the pain often migrates and localizes to the abdominal wall location juxtaposed to the offending organ due to localized peritoneal inflammation. Frank perforation usually causes severe, diffuse abdominal pain. Inquire as to what relieves or aggravates the pain [72,74]. Ask whether the pain is modified by position, eating, or movement. Pain that is exacerbated by movement is consistent with peritonitis, whereas pain that improves with meals is consistent with peptic ulceration [14,71,72,74].

The past medical history helps assess the surgical risk preoperatively. If the patient has undergone appendectomy, cholecystectomy, or total colectomy, then diseases of these organs would be excluded. Patients who have had prior abdominal operations are prone to bowel obstruction secondary to postsurgical adhesions [32,36]. Patients who have peptic ulcer disease, cancer, diverticulosis, or inflammatory bowel disease are at increased risk of gastrointestinal perforation from these diseases [16,47,50,75–77]. A review of recent medications is important. Patients on chronic high-dose nonsteroidal anti-inflammatory drugs or corticosteroids are at increased risk of gastrointestinal perforation. Patients who have had an acute onset of abdominal pain and diarrhea after recent antibiotic use should be suspected of having *C difficile* colitis [65].

The physical examination is important to help secure the diagnosis and to detect signs of coexistent diseases that can affect treatment options or can require specific preoperative interventions [71,72,74]. The initial focus should be on the overall appearance of the patient. Patients who are obtunded or in extremis should, like trauma patients, have an initial assessment of their airway, breathing, and circulatory status. These parameters can be assessed by conversation with the awake and alert patient. The vital signs provide clues regarding disease severity, volume status, pain severity, SIRS, and sepsis. Tachycardia, hypoxia, tachypnea, and hypotension suggest a compromised physiologic state consistent with shock [72,78]. Tachycardia with hypertension in an otherwise stable patient may be secondary to pain or anxiety.

For a suspected acute abdomen, thoroughly examine the abdomen, groin, and perineum and perform a digital rectal examination on all patients and
a pelvic examination on female patients. Many practitioners find it useful to slightly bump the patient’s bed or ask the patient to cough [79]. Patients who have peritonitis often wince in discomfort with these maneuvers. The rapid diaphragmatic excursion and resultant increased intra-abdominal pressure or the forced movement of the patient elicits peritoneal pain. The examination includes visual assessment, auscultation, palpation, and percussion of the abdominal wall. Inspect for previous surgical scars, open wounds, ostomies, masses, hernias, rashes, and ecchymoses indicative of trauma, past surgeries, or current disease. An ostomy provides a unique opportunity to partially evaluate bowel viability. Inspect the mucosa for edema, pallor, and necrosis and inspect the ostomy appliance for output quality and quantity.

Abdominal palpation is gently initiated in an area where the patient has reported the least pain, and then the entire abdomen is progressively and thoroughly examined. It is helpful to engage the patient in conversation during the examination to distract the patient from the palpation to more accurately assess the abdomen [72]. Percuss the abdomen and listen for tympany, which may be secondary to a diffuse pneumoperitoneum or bowel obstruction.

Diffuse peritonitis is a relatively straightforward clinical diagnosis. Aside from a history of severe pain exacerbated by movement, abdominal examination demonstrates exquisite tenderness and involuntary guarding manifested as a rigid abdomen [72,79] due to inflammation of the abdominal wall muscles. Patients who have gastrointestinal perforation do not necessarily present with diffuse peritonitis. They may exhibit local peritoneal irritation due to perforation contained by intra-abdominal defense mechanisms that results in abscess or phlegmon formation. Patients who have iatrogenic perforation may have extensive pneumoperitoneum with a distended and tympanic abdomen without overt peritonitis. Classic signs of localized peritonitis include the obturator sign, Fothergill’s sign, Rovsing’s sign, psoas sign, and Murphy’s sign [72].

Examination of the groin and perineum may reveal hernias; testicular, vulvar, anal, or perianal pathology; or trauma. In female patients, a bimanual pelvic examination may reveal adnexal tenderness or masses indicative of gynecologic pathology. A digital rectal examination may reveal rectal masses, perirectal abscesses, or prostatic pathology.

Laboratory and radiographic testing

Supplemental laboratory studies are selectively ordered based on the pretest differential diagnoses to help secure the diagnosis. Laboratory testing for comorbid conditions is initiated if the results are needed to guide therapeutic intervention, for further diagnostic studies, or for preoperative optimization. Typically ordered tests include a complete blood cell count with differential, coagulation panel, electrolyte panel, liver function tests, urinalysis, lipase or amylase, arterial blood gas, and serum lactate level.
Not all of these tests are indicated in every patient. The complete blood cell count is useful to evaluate the patient’s white blood cell (WBC) count, which is a marker of inflammation or infection. Although the WBC may occasionally be normal in these patients, an elevation or significant depression helps in forming the differential diagnosis. The hematocrit is used to detect anemia due to concurrent disease or the presenting problem. It can help gauge the patient’s volume status; an elevated hematocrit may be secondary to hypovolemia. Furthermore, assessing the hematocrit level and platelet count can help guide the type of fluid resuscitation (crystalloid versus blood transfusions) and the need for blood products for surgery.

An electrolyte panel, which usually includes serum sodium, potassium, chloride, bicarbonate, creatinine, blood urea nitrogen, and glucose and often includes a calcium level, is helpful. Although electrolyte panels are not necessarily diagnostically useful, they provide important data regarding the physiologic status and volume status that guide resuscitation efforts. The patient’s renal function may affect the radiologic evaluation (eg, whether to use intravenous contrast agents). Liver function tests, amylase, and lipase are useful in patients who have suspected hepatobiliary or pancreatic disorders. Performing an arterial blood gas in patients who have some distress or with known pulmonary disease provides information regarding their respiratory status and acid–base balance. These data are essential for initiating preoperative therapy, planning postoperative care, and evaluating for the onset of metabolic acidosis, which is a harbinger of shock. When ischemic bowel is suspected, a serum lactate level is useful. It is fairly sensitive but is not very specific for this diagnosis. The urinalysis, a relatively inexpensive test, helps diagnose urinary tract infection. Other potential tests include a beta-human chorionic gonadotropin for women of childbearing age and C difficile toxin analysis for patients suspected of having that form of infectious colitis.

Radiographic evaluation of the acute abdomen traditionally began with a three-view acute abdominal series consisting of an upright chest radiograph and upright and supine abdominal radiographs. Recent clinical studies have found this abdominal series to be a relatively poor instrument to evaluate the acute abdomen. In a study by MacKersie and colleagues, this three-view abdominal series had a sensitivity of 30%, an accuracy of 56%, and a negative predictive value of 51% in patients who had nontraumatic acute abdominal pain. Despite this poor performance, some authorities argue that it is an inexpensive and rapid test of patients who have a high pretest suspicion for hollow viscus perforation. Patients who have acute abdominal pain and pneumoperitoneum on a plain abdominal radiograph traditionally underwent urgent laparotomy without further diagnostic evaluation. This argument has been challenged by recent studies that have found plain radiographs to be insufficiently sensitive to evaluate pneumoperitoneum.
high as 100% for large-volume pneumoperitoneum if the patient is placed in
an upright or decubitus position for 5 to 10 minutes before an upright chest
or lateral decubitus radiograph is performed [86]. However, nearly 50% of
patients who had hollow viscus perforation at laparotomy failed to demon-
strate pneumoperitoneum on plain abdominal radiographs [87]. Moreover,
the finding of pneumoperitoneum on plain radiographs in patients who have
acute abdominal pain is not always associated with the need for surgery for
hollow viscus perforation. Roh and colleagues [88] reported that 14% of
their patients who had abdominal pain and pneumoperitoneum on plain ab-
dominal radiographs underwent an unnecessary laparotomy because the
pneumoperitoneum was from sources other than a perforated viscus. They
noted that the clinical presentation of their patients who had surgical versus
nonsurgical causes of pneumoperitoneum was indistinguishable [88]. Others
have reported similar conclusions, with about 10% of cases of pneumoper-
itoneum secondary to nonsurgical causes [89–92]. Numerous case series of
nonsurgical causes of pneumoperitoneum have been reported [89–95]. Box
1 lists commonly reported causations.

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Helical CT has become the radiographic test of choice to evaluate the acute abdomen based on numerous retrospective and prospective studies. These studies have reported a diagnostic sensitivity of 95% to 98%, a specificity of 95.1% to 97%, and an accuracy of 95.6% [82–84,96–99]. It is also a rapid and cost-effective imaging modality [96–99]. Recent advances in helical CT scanners, improvements in contrast agents, and refinement of CT protocols have further enhanced the utility of this test. Its accuracy highly depends upon the CT protocol [99]. Communication between the ordering physician and the radiologist is vital, as is selection of the CT protocol based on the pretest differential diagnosis. The protocol variables include the use of contrast agents, acquisition phases, collimation parameters, pitch, and the reconstruction interval [99]. These variables can greatly affect the diagnostic accuracy of the CT scan for specific disease processes. Urban and Fishman [99] have published an excellent CT protocol for nonlocalized acute abdominal pain. They recommend 750 to 1000 mL of an oral contrast agent and 110 to 120 mL of a nonionic intravenous contrast agent delivered at 2 mL/s. A single portal venous phase CT is performed 70 to 90 seconds after intravenous contrast infusion from the diaphragm to the pubic symphysis with a 5-mm collimation, a pitch of 1.6, and a 5-mm reconstruction interval [99].

Helical CT is an excellent tool to assess suspected gastrointestinal perforation [98]. When feasible, oral and intravenous contrast agents are used to facilitate identification of perforation, peritonitis, ischemic bowel, and abscess formation [98,99]. Oral contrast helps accurately localize the site of perforation by visualization of extravasation of contrast outside the gastrointestinal lumen [98,99]. A water-soluble contrast agent is important in patients who have suspected hollow viscus perforation because extravasation of barium can cause severe peritonitis that carries a mortality of up to 50% [100,101]. CT is also excellent at detecting free intraperitoneal air, but the location of the free air does not necessarily correlate with the site of the perforation [98,99]. Other localized CT findings include inflammation and focal fluid or air collections [99].

Other helpful radiographic studies include ultrasonography and MRI [102–104]. Ultrasonography is an inexpensive, rapid, and nonradioactive test. Although it is only a fair diagnostic test, it is an excellent tool to rapidly evaluate pneumoperitoneum, free intra-abdominal fluid, and gynecologic pathology. It is frequently used to evaluate trauma patients. In prospective studies of suspected nontraumatic hollow viscus perforations, ultrasonography had a 93% to 100% sensitivity and 64% to 99% specificity for the detection of pneumoperitoneum [102,103]. It is not the diagnostic study of choice in stable patients because it fails to identify the source of perforation, perforation without pneumoperitoneum, or nonsurgical causes of pneumoperitoneum. In preliminary studies of MRI for acute abdominal pain, it has shown no significant benefit over CT [104]. MRI is also slow and frequently unavailable.
Therapeutic intervention

The treatment of gastrointestinal perforation includes fluid resuscitation, antibiotics, source control, organ system support, and nutrition [4,5,15,105,106]. The overall clinical state of the patient dictates the treatment. Patients in extremis from gastrointestinal perforation and diffuse peritonitis require rapid resuscitation, initiation of antibiotics, and emergency surgery. Relatively stable patients undergo a similar therapeutic approach but with a greater focus on preoperative optimization of the patient’s physiologic status with vigorous resuscitation and correction of any concurrent coagulopathy, acidosis, and electrolyte disturbances [5,105,106]. Concurrent comorbid conditions should be treated or prophylaxed as necessary, such as preoperative beta-blockade for normotensive and hypertensive patients at high risk for myocardial infarction or preoperative beta-agonists, ipratropium, or corticosteroids for patients who have significant pulmonary disease [107,108]. Patients who have documented adrenocortical insufficiency or chronic corticosteroid use should receive intravenous corticosteroids, whereas diabetic and septic patients should have serum glucose levels monitored and insulin therapy administered as needed [109]. Antibiotics are standard treatment for gastrointestinal perforation [4,5,15,105,106,110]. The antibiotic is selected according to the specific site of perforation [5,105,110]. Broad-spectrum antimicrobial coverage is instituted in the preoperative period, and the therapy is then tailored based on operative findings of source and degree of contamination. Excellent broad-spectrum coverage is provided by various agents or a combination of agents [5,105,106,110,111]. The choice of antimicrobials is based on adequacy of microbial coverage, patient allergies, hospital-specific resistance patterns, patient comorbidities, cost-effectiveness, and practitioner comfort with the drug. Many efficacious regimens have been described, and no single agent or combination of agents has been found to be superior to the others [110,112,113]. A recent Cochrane database meta-analysis of 40 randomized and quasi-randomized controlled studies including 5094 patients treated for secondary peritonitis of gastrointestinal origin found no significant difference in patient outcomes based on the antibiotic coverage [112]. The antibiotic regimens included 16 different single or combination antimicrobial agents, including aminoglycosides with anaerobes or with broad-spectrum penicillins coupled with a beta-lactamase inhibitor, broad-spectrum penicillins alone or with a beta-lactamase inhibitor, aminoglycosides plus a broad-spectrum penicillin and an anti-anaerobe, carbapenems, cephalosporins with or without a beta-lactamase inhibitor alone or with an anti-anaerobe, clindamycin, fluoroquinolones with or with out an anti-anaerobe, monobactams plus an anti-anaerobe, or imipenem/cilastatin [112]. Most authorities recommend initiation of a regimen that broadly covers gram-negative, gram-positive, and anaerobic organisms [4,5,15,105,106,110]. An important issue is coverage for Candidal or Enterococcal infections [105].
The current data suggest that Enterococcal coverage is unnecessary for community-acquired peritonitis but should be considered for hospital-acquired peritonitis, immunosuppressed patients, patients who have prosthetic heart valves, or patients who have recurrent intra-abdominal infections accompanied by septic shock [105]. The need for anti-Candidal coverage in secondary peritonitis is debatable due to the variably reported frequency of Candidal species in the peritoneal fluid of patients who have gastrointestinal perforations. Some authors reported Candida to be the first or second most frequently isolated species from peritoneal fluid cultures, whereas others reported isolation of Candida in only 12% of patients [13]. In a placebo-controlled trial of patients who had gastrointestinal perforations, Eggimann and colleagues [114] showed that administration of fluconazole significantly reduced the rate of intra-abdominal candidiasis in high-risk patients. Most authorities consider the addition of anti-Candidal therapy only for immunosuppressed patients, septic patients, and patients who have positive fungal cultures. When used, anti-Candidal therapy should be continued for 2 to 3 weeks [105,115].

Antimicrobial therapy is an adjunctive treatment of secondary peritonitis to prevent local spread of infections, bacteremia, and sepsis and to reduce late complications [4,5,15,105,106,110]. The dosage and length of antimicrobial therapy is unclear in a particular patient. Despite many years of antibiotic use for secondary peritonitis, a unified consensus statement based on scientific evidence from randomized controlled trials is unavailable. Blot and De Waele [105] recommended, based on the 2002 Surgical Infection Society guidelines, that the type and time course of antimicrobial management should be based on (1) whether the source has been adequately controlled by appropriate drainage and correction of the underlying process [105,116], (2) whether therapy is required beyond 24 hours (they advocate no more than 24 hours of antibiotics for uncomplicated gastroduodenal perforations or for traumatic or iatrogenic small and large bowel perforations operated on within 24 hours of onset), (3) whether anti-fungal or enterococcal coverage is needed [105,116], and (4) whether coverage of resistant pathogens is needed for hospital-acquired perforations. The duration of antibiotic coverage is controversial [105,116]. Some authors advocate a standard treatment of 7 to 14 days, whereas others recommend continuing antibiotics until the WBC count has normalized and the patient is afebrile [105,110]. Current general consensus advocates antimicrobial therapy for 5 to 7 days if the clinical signs of infection have resolved [105,116]. If the patient fails to improve or worsens during this period, the adequacy of source control or appropriateness of antibiotic coverage must be questioned [105].

The most important therapy for gastrointestinal perforation is source control [5,15,105,106]. Traditionally, source control is acquired by surgery, but selected conditions are increasingly treated conservatively by radiologic or endoscopic intervention. The principles of source control dictate direct containment and control of the site of perforation, evacuation of
intraperitoneal contamination, drainage of abscesses, debridement of necrotic tissue and foreign matter, and re-establishment of functional anatomy [5,15,105,106]. An exploratory laparotomy is usually performed via a midline incision and provides excellent visualization and easy access to the entire abdomen. The exploration is performed in a standard and regimented fashion to evaluate all gastrointestinal structures from the gastroesophageal junction to the peritoneal reflection of the rectum. Obvious sources of perforation are isolated and controlled. The remainder of the gastrointestinal tract is then explored. This exploration is important to find concurrent pathology or an underlying cause for the perforation, such as distal obstruction. When an obvious site of perforation is identified, exploration can be limited to an evaluation that does not unnecessarily disrupt intact anatomic structures, such as the gastrocolic ligament. Visceral exploration should include examination of the anterior stomach, the jejunum from the ligament of Treitz to the terminal ileum, the entire colon, and the peritonealized rectum. If a perforation is not identified, then the lesser sac should be entered through the gastrocolic ligament to evaluate the posterior stomach and anterior duodenum. If the source of contamination is still not evident, a Kocher maneuver can be performed to inspect the posterior and posterolateral duodenum. Most surgeons use open exploration, but a minimally invasive laparoscopic approach is an effective means of surgical exploration and source control in experienced hands [3,117–121].

Operative containment and control of the perforation is achieved by resection or repair of the anatomic site of damage. The chosen method depends on the underlying pathologic process and the size and duration of the perforation [5,15,105,106]. Small perforations can be repaired primarily by simple interrupted sutures if the following two criteria are met: (1) The lesion must not be malignant. If malignancy is possible, this question can be resolved by intraoperative biopsy with frozen section (FS) pathologic evaluation. If FS evaluation is nondiagnostic or unavailable, perforations from possible malignant processes should be resected. (2) The tissue surrounding the perforation must be healthy, wellperfused, and viable. All foreign material or necrotic debris must be removed from the perforated lumen, and only healthy, well perfused tissue must remain. The repair may be buttressed with an omental patch or with an adjacent segment of healthy bowel, such as an ileal patch, if the area is markedly inflamed [122]. Primary repair can sometimes be safely performed laparoscopically. Recently, minimally invasive repair of relatively clean colonic perforations have been successfully performed using surgical clips instead of the traditional suture repair. The initial data, consisting of scattered case reports, are promising.

Primary repair of perforated bowel is preferable to resection and anastomosis because it carries a lower complication rate [123]. This better outcome may reflect the limited tissue injury in these patients [123]. Primary repair should not be performed in patients who have malignant lesions, necrotic bowel, perforations associated with mesenteric vascular injuries, lesions
involving more than 50% of the bowel circumference, or multiple contiguous perforations. When resection is required, the entire diseased segment is resected, leaving healthy, well perfused ends for anastomosis. The technique for the enterointerostomy, whether stapled or hand-sewn, seems to have little impact on the anastomotic complication rate [123]. Primary bowel anastomosis must be considered cautiously in the setting of gross purulent or feculent peritonitis because of a high rate of serious complications [123,124]. Traditionally, a damage control operation was performed with resection of the injured bowel and peritoneal toilet [5,15,106], followed by a washout and reanastomosis if the conditions were favorable. It was recently shown that the functional anatomy may be safely restored during the first operation, but lumen patency and bowel wall viability should be monitored by planned relaparotomy [124]. The principles of colonic resection are similar to those for small bowel, with resection of the diseased segment and adequate peritoneal toilet [4,105]. Restoration of functional anatomy is approached differently in the setting of generalized peritonitis. Primary colocolonic anastomosis is avoided because of a high complication rate [16,125–127]. The standard approach for middle and distal colonic perforations in the presence of generalized peritonitis has been a Hartmann’s procedure, with creation of a proximal end-colostomy and a distal colonic pouch with or without a mucous fistula [42]. Take-down of a Hartmann’s pouch with restoration of colorectal continuity can be performed subsequently for most benign diseases, but this operation can be challenging. Some surgeons advocate a primary colocolonic anastomosis with a diverting loop ileostomy to avoid the complications and technical difficulties of a Hartmann’s take-down. Ileal diversion seems to be a safe and promising alternative to the traditional Hartmann’s procedure, but a randomized controlled trial comparing the two techniques has not been performed.

Perforation from ischemic bowel follows the treatment principles discussed previously. A critical difference involves determination of viable versus nonviable bowel. It is important to preserve as much viable bowel as possible to prevent the short gut syndrome, especially when long segments of bowel are ischemic. At surgery all clearly nonviable bowel is resected. When nonviable bowel is flanked by segments of questionable viability, clearly necrotic bowel is removed, and areas of uncertain viability are left in situ and in discontinuity. Re-exploration is performed to resect any newly demarcated bowel followed by functional anatomic restoration [105]. Alternatively, bowel viability is determined intraoperatively by clinical judgment, ultrasonography, electromyography, fluorescein viability testing, or laser Doppler flowmetry [128–130]. Clinical judgment based on bowel color, motility, pulsation, and mucosal bleeding has a sensitivity of 91% and a specificity of 82%. It is the most used and least cumbersome approach [58]. Several papers have been published on the other techniques, with mixed results [58,128,129]. Superior Mesenteric Artery (SMA) blood flow must be assessed [58]. If SMA flow is severely compromised due to arterial
thrombosis or embolism, patency must be restored by embolectomy or by-pass grafting [58]. Once viability has been determined, resection is performed, leaving viable ends for primary anastomosis. If concern still exists, a second-look operation is performed to assess anastomotic integrity and intestinal viability.

Gastric and duodenal perforations are common causes of free intra-abdominal hollow viscus perforation [50], most commonly secondary to peptic ulcer disease (PUD) [48–51,131]. Treatment of PUD has changed dramatically over the last several decades since the identification of *H pylori* as a major etiologic factor. The mainstay of PUD therapy is *H pylori* eradication and proton pump inhibitors (PPIs) [48–51,132]. This approach has almost eliminated the need for elective surgery for PUD. However, the incidence of perforated PUD may be increasing [50]. These and other data have led to speculation that perforated PUD may have a different etiology than non-perforated PUD. Although 95% of patients who have duodenal ulcers (DU) and 60% of patients who have gastric ulcers (GU) have *H pylori*, as few as 50% of patients who have perforated GU and DU have *H pylori* [48]. Regardless of this debate, the surgical approach to perforated PUD has dramatically changed from extensive resections and acid-suppressing procedures to simple closures, with or without omental patching [48,50,57,132]. Simple closure for a perforated DU, coupled with temporary acid suppression with PPI and *H pylori* eradication, entails a less than 5% recurrence rate at 1 year, which rises to 38% when *H pylori* therapy is not instituted [50,132]. In a series of 88 consecutive patients who had perforated DU treated by this approach, Rodriguez-Sanjuan and colleagues [51] noted no ulcer relapse during a mean follow-up 22.3 months (range, 1–80 months) [50]. This study and other studies have established that simple closure, acid-suppression medication, and *H pylori* eradication for *H pylori*-positive patients is the standard therapy for perforated DU [50,132]. The treatment for perforated GU is debatable partly due to the 10% to 30% risk of cancer in these gastric ulcers [133,134]. The current practice is to resect the GU, including an adequate margin, and obtain FS analysis. If the lesion is benign, then simple closure with an omental patch coupled with PPI therapy and *H pylori* eradication for *H pylori*-positive patients is the standard approach. This approach is somewhat controversial due to a rate of GU recurrence as high as 75% and the risk of repeated perforation [51]. A dilemma arises when FS evaluation is unavailable or nondiagnostic; some authorities advocate simple closure with reoperation if the final pathologic review demonstrates cancer, whereas others argue for a distal gastric resection incorporating the perforation according to established oncologic principles, followed by lymph node dissection at a separate surgery if the final pathologic analysis indicates cancer. This latter policy is based on the relatively high incidence of gastric cancer in perforated GU and the high long-term mortality from simple closure of a perforated gastric cancer [51,135,136]. The correct approach is uncertain and is in the hands of the individual surgeon.
The approach to gastric cancer perforation, documented by the patient history or FS evaluation, is fairly well established. It was previously believed that perforation represented late-stage cancer with tumor dissemination into the peritoneal cavity [136]. Based on this belief, simple closure was believed to provide the appropriate palliation while minimizing the surgical stress in a critically ill patient [136]. Recent surgical data show significantly increased perioperative and long-term mortality with simple closure compared with gastric resection [135–138]. Therefore, patients who have perforated gastric cancer should undergo appropriate gastric resection in spite of concurrent peritonitis unless the patient is hemodynamically unstable or has unresectable cancer [135–138].

Many patients who have gastrointestinal perforations develop SIRS or sepsis with accompanying hemodynamic compromise, hypothermia, acidosis, and a coagulopathy [4,139–141]. These patients require rapid resuscitation and rapid surgery. The challenge is to provide life-saving surgery without causing further physiologic compromise from the stress of surgery in these already compromised patients. The standard approach is known as “damage control” or “salvage” surgery. The goal is to rapidly obtain source control and peritoneal toilet without prolonging the surgery to restore functional anatomy or extensively mobilize tissues [105]. This surgery may simply involve suture control of perforations or rapid resection of frankly necrotic bowel [4]. Frequently, the bowel is left in discontinuity, the abdomen is cleared of gross debris, and a temporary abdominal closure is performed. The patient is then returned to the intensive care unit for appropriate resuscitative measures [142]. After stabilization, the patient can undergo a more deliberate and definitive surgery. Management of the open abdomen is greatly facilitated by the “vacuum-pack” procedure [142–144], which involves placement of a perforated, nonadherent polyethylene sheet over the exposed visceras and under the anterior borders of the opened abdominal fascia. A layer of compressible, absorbable material (surgical sponge or towel) is placed over the polyethylene sheet along with two silastic drains. A sheet of self-adhesive polyester (or equivalent) is placed across the skin of the outer abdominal wall, fully encompassing the other components of the vacuum device. The drains are connected to a continuous suction device set at 100 to 150 mm Hg negative pressure [142]. The vacuum pack effectively isolates the intra-abdominal cavity from the environment, provides drainage of abdominal fluids, provides for internal expansion to avoid the abdominal compartment syndrome, and facilitates emergency access and reoperation of the abdomen [142–144].

Iatrogenic gastrointestinal perforations from endoscopic instrumentation can provide a unique opportunity for conservative management. Endoscopic perforations often occur in a prepared and relatively clean intestinal tract [7,27,28]. The perforation may also be identified immediately or within minutes of its occurrence. If the perforation is recognized quickly and the patient has a relatively clean preparation, nonsurgical management is often
successful [7,27,28]. Nonsurgical management highly depends on the time of diagnosis, location and size of the perforation, degree of contamination, and condition of the patient. Nonsurgical management can be successful in stable patients who have minimal signs and symptoms of peritonitis and who have small injuries to the stomach, duodenum, and retroperitoneal portions of the colon [7]. These locations offer possible anatomic containment of the perforation by the retroperitoneal space or omentum. Perforations of the intraperitoneal small bowel and colon usually require surgery, except for microperforations. Microperforations often cause minimal peritoneal contamination and can seal spontaneously [7,145]. Treatment of endoscopic perforations is rapidly evolving. Numerous case reports and case series have demonstrated the efficacy of endoscopic repair of injuries at the time of occurrence by approximating the two ends of a perforated segment via endoluminal clips [146–155]. Although these clinical reports are limited to small perforations, experimental studies of large endoscopic perforations in laboratory animals have produced similar results [156]. In one study, animals were randomized to open repair with interrupted sutures versus endoscopic repair with vascular clips for standardized iatrogenic injuries. The authors reported no difference in clinical outcome, repair burst strength, or hydroxyproline levels between the two groups. Iatrogenic gastric perforations have also been successfully repaired endoscopically by pulling extramural omentum into the gastric defect and tacking it to the muscularis propria with clips to endoscopically create an omental patch [7]. These endoscopic therapies are in their infancy but may become a standard therapeutic approach for iatrogenic perforations in stable patients.

Nonsurgical management can be occasionally applied to nonendoscopic perforations of the stomach, duodenum, and colon. These perforations are usually small and have been contained by local tissue, omentum, or the retroperitoneum [7]. These include small gastric and duodenal perforations caused by benign diseases, such as PUD. In these cases, the perforation may be spontaneously sealed by the greater or lesser omentum. The resultant omental patch is similar to a surgically created patch and may be sufficient therapy [145]. It is difficult to determine whether a perforation has spontaneously sealed, but a CT scan with oral contrast may be helpful [157]. Nonsurgical management of spontaneously sealed perforations should be considered only in stable patients who do not have overt signs of peritonitis [7]. These patients must undergo endoscopy after recovery from the acute process to ensure appropriate healing and to exclude cancer. Selected colonic perforations, such as certain iatrogenic injuries or perforation secondary to diverticulitis, may also be managed nonoperatively [42]. Not all diverticular perforations can be managed nonoperatively. Spontaneously sealed perforations and perforations that are contained with the development of an associated abscess cavity can often be successfully managed without surgery [42]. An excellent and clinically useful classification system for diverticular perforations was developed by Hinchey and colleagues [16]
and modified by others. In short, Hinchey stage 1 (pericolic or mesenteric abscess) and stage 2 (walled-off or pelvic abscess) disease is usually treated conservatively with antibiotics, radiologically guided abscess drainage, and parenteral nutritional support. Hinchey stage 3 (generalized purulent peritonitis without colonic communication) or stage 4 (generalized fecal peritonitis with colonic communication) requires emergency surgery, following the treatment principles for gastrointestinal perforation discussed previously [16,42].

Postoperative care

The medical management of patients treated surgically or conservatively for gastrointestinal perforation is partly based on the physiologic state of the patient and the clinician’s perceived risk of further deterioration. The level of required care is individually assessed based upon the general underlying principles of management. Emphasis is placed on continued resuscitation, antibiotics, physiologic support of compromised organ systems, nutrition, and pain management [4,5,15,72,105]. Fluid management is usually the key resuscitative measure. Peritonitis directly causes loss of intravascular fluid volume [4]. The peritoneum is composed of a basement membrane with an inner single-cell layer of mesothelial cells. This lining permits free bidirectional exchange of fluids across an average surface area of 1.7 m² [2,4]. Gastrointestinal perforation with generalized peritoneal inflammation results in hyperemia and exudation of large amounts of fluid containing WBC and opsonins into the peritoneal cavity [4]. A massive fluid shift from the intravascular compartment into the peritoneal cavity can lead to the development of hypovolemia and shock. The degree of hypovolemia is partially determined by the size, location, and duration of the perforation [4,5,105]. Fluid loss during open abdominal surgery and postoperative open abdominal management can aggravate the fluid deficit. Crystalloid fluid resuscitation is usually indicated, but transfusion of blood products should be considered in patients who are coagulopathic, have ongoing bleeding, or have profound anemia that is insufficient to meet the patient’s oxygen delivery requirements. Determination of the adequacy of resuscitation is controversial, and there are no universally accepted criteria. Restoration of normal hemodynamic parameters, normalization of urine output (0.5–1.0 mL/kg/h), and correction of acidosis are considered reasonable clinical indicators of adequate resuscitation [4], but these parameters can be complicated by acute or pre-existent organ system failures, especially of the heart, kidneys, or liver.

When gastrointestinal perforation results in SIRS or sepsis, patients often develop shock accompanied by multiorgan failure, coagulopathy, hypothermia, and acidosis [141]. Ongoing resuscitative and supportive measures focus on vigorous warming of the patient, correction of the coagulopathy,
and acid–base management through ventilator and fluid manipulation [4,5,105,141]. These patients often require mechanical ventilation, hemofiltration, and mechanical cardiac augmentation, which can provide a lifesaving bridge while the underlying disease process is treated. Invasive monitoring can provide instantaneous, accurate clinical data to guide the administration of pharmacologic hemodynamic support. The most important treatment of gastrointestinal perforation is source control. All other therapies, including antibiotics, are adjunctive or supportive measures [4,5,15,105].

Summary

Perforation of the gastrointestinal tract remains a considerable cause of morbidity and mortality. The treatment relies on a thorough understanding of the anatomy, microbiology, and pathophysiology of the underlying disease and resultant acute processes. Optimal patient care depends upon early diagnosis and rapid therapy.

The diagnosis is founded on a well conducted history and physical examination, supplemented by appropriate laboratory and radiologic studies. Once gastrointestinal perforation has been diagnosed, strict adherence to the principles of management is essential, including resuscitation, antibiotics, source control, and physiologic support. Source control is the most important step in the definitive management of these patients and must be rapidly achieved. The other supportive or adjunctive therapies help to supplement adequate source control.

References


