PERFORATED VISCUS AND THE ACUTE ABDOMEN

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OBJECTIVES

Presumed Knowledge:

1. The student is able to describe the structure and relationships of the GI Tract and peritoneum.
2. They are able to describe the nerve pathways and the features of visceral and parietal [somatic] pain.
3. They can describe the secretory function of the segments of the GI tract and the digestive organs.
4. They have a working knowledge of the natural history and gross pathology of inflammatory and neoplastic diseases of the GI tract.

Knowledge to be Acquired:

1. The student should be able to develop a differential diagnosis based on the patient's initial presentation.
2. They should be able to take then a full history, asking indirect questions and direct questions when necessary, which reflect knowledge of the most likely diagnoses..
3. The student should be able to conduct a physical examination which accurately portrays the patient's general status and identifies ventilatory and circulatory problems quickly. The student then should be able to complete the examination of the abdomen, recognizing the physical signs of peritoneal irritation, and be able to localize the process to an abdominal quadrant when this situation exists.
4. The student should be able to initiate monitoring and appropriate early diagnostic and resuscitative measures.
5. The student should be able to arrive quickly at a provisional diagnosis.
6. The student should know those conditions which may require very early operative management.
7. The student should be able to describe in basic terms the operative strategies for management of perforation of the intestinal tract based on the underlying pathology and the site of the perforation.
8. The student should be able to write appropriate post-operative orders.

Approach to the Patient with an Acute Abdomen

Although this presentation will eventually have a focus on the problem of perforated viscus, it is important initially to bring a general knowledge of the conditions which cause acute abdominal pain and their relative risks, to the bedside. A grouping into general categories is seen below.

1. Intra-abdominal or retroperitoneal hemorrhage.
2. Ischemia or infarction of the intestinal tract.
3. Perforated viscus.
4. Obstructed hollow viscus.
5. Acute intra-abdominal or retroperitoneal inflammation.
6. Extra-abdominal or non-GI causes of abdominal pain.
7. Factitious causes of abdominal pain.
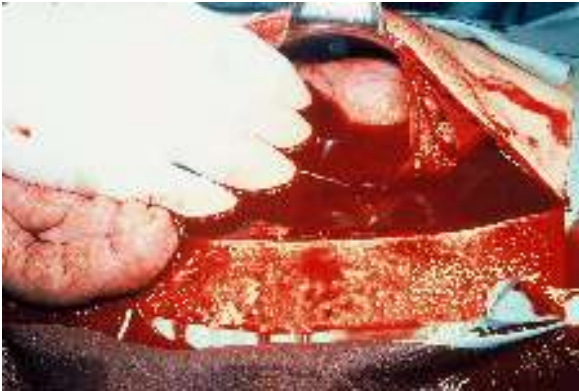
Blood in the peritoneal or retroperitoneal space appears to be a signifigant irritant. Although an uncommon cause of acute abdomen, the unpredictable course and critical nature of blood volume contraction makes early diagnosis mandatory.

Sepsis and ECF contraction may also eventually result in shock, but will not do so within minutes of the onset of pain, as in this case of ruptured, pathologic spleen [infectious mono.]

As in any acutely ill patient, the issues of Airway, Breathing, and Circulation need to be addressed. Airway obstruction seldom accompanies complaints of acute abdominal pain but needs to be quickly excluded. Usually a few seconds of observation, noting the absence of stridor and presence of a normal quality voice, is sufficient.

Ventilatory compromise may occasionally be caused by severe abdominal distention and pain, aggravated by pre-existing pulmonary diseases and narcotics, severe acidosis and hypotension caused by the intra-abdominal process.

Many of the processes causing acute abdominal pain will result in contraction of the circulating blood volume. Hemorrhage, as in rupture of an abdominal aortic aneurysm, may critically reduce blood pressure and result in a recognizable picture of decompensated hypovolemic shock, with pallor, diaphoresis, and cool and mottled extremities. Severe acute pancreatitis, or mesenteric arterial occlusion with mid-gut infarction, may also result in shock, but require longer periods in which to evolve. Small bowel obstructions cause contraction of the effective volume of the extracellular fluid by third spacing into the lumen of the obstructed gut. Again, the contraction of the plasma volume requires time, compensation occurs and the reduced blood volume and cardiac output may not be obvious.

Acute pancreatitis with positive Ranson's major criteria is a potent cause of rapid and massive "third spacing" The plasma volume contracts quickly in the first hours after onset. Hemo concentration, hypotension, anuria may be established by the time the patient presents.
  This patient underwent an early laparotmy with the mistaken diagnosis of perforated viscus .A hemorrhagic ascites, pancreatic phlegmon, and extensive fat necrosis was found. Useful surgical options are few.

The obstructed small bowel sequesters litres of fluid with electrolyte concentrations similar to ECF .Plasma volume contracts. Adults seldom develop hypovolemic shock, but a compensated picture evolves with postural drop in BP, low cardiac filling pressures[CVP] and pre-renal failure.If strangulation occurs[as in this case] toxemia and/or bacteremia will be superimposed on the volume deficit.
  If not properly resuscitated, these individuals tolerate narcotics and anesthetic agents poorly.

Patients with acute abdominal pain should be examined carefully for compensated loss of blood volume, looking for a postural drop in blood pressure, collapsed peripheral veins, absence of jugular filling, poor quality peripheral pulses, cool skin, and slow capillary refill.

Patients with an acute abdomen presenting to emergency rooms are usually assessed in a triage area. Young patients who are ambulatory and have stable vital signs, for example, a possible appendicitis, may then be assessed in a fast track or short stay area.

   [Refer to the flow chart at the end of this section for a quick overview]

Older individuals and all those exhibiting significant distress or displaying objective signs of a potentially dangerous condition (rapid pulse, temperature over 38, low blood pressure) would be then directed to an acute bed where monitoring should be established. Monitoring would consist of a 3-lead electrocardiogram display, O2-sat monitoring, and intermittent display of blood pressure and pulse. A peripheral intravenous should be quickly established and venous blood taken for laboratory tests. Specific tests to be requested may be decided later as the differential diagnosis is developed.

The patient with an acute abdomen needs to be seen by a physician as soon as the early nursing assessment is completed and monitoring is set up.

Hemorrhage as a cause of abdominal pain needs to be given first consideration. The course of arterial bleeding into the peritoneum or retroperitoneum in these conditions is unpredictable and exanguination can occur without warning. The most common cause of sudden collapse, abdominal pain and shock is a ruptured abdominal aortic aneurysm. A free rupture into the peritoneal cavity results in exsanguination and death so rapidly that no intervention could be taken; even if the patient were in an emergency room at the time it occurred. Most abdominal aortic aneurysms, however, leak initially and then are contained by the retroperitoneal tissues. During this timeframe, which may be only measured in minutes or hours, there is time for a diagnosis and transport to an operating room.   As blood dissects through the retroperitoneum pain is experienced in the back and flank. A sensation similar to tenesmus may be described by the patient as blood dissects into the pelvis.

Pallor, diaphoresis and hypotension are present almost immediately and should alert the examiner.

Many of these individuals are obese and the characteristic pulsatile mass in the upper abdomen may be difficult to feel. Although an intravenous should be started and blood taken for other baseline values and cross-match, attempts at resuscitation are fruitless and waste time. Trying to insert nasogastric tubes and place Foley catheters in the emergency room cause further delay. A trip to the x-ray department for a CT scan or ultrasound may prove fatal. Often the patient is best

taken, by a vascular surgeon, quickly to the operating room, diagnosis based only on a strong clinical suspicion.

Other causes of intra-abdominal hemorrhage, although also threatening, are at least amenable to resuscitation and may stabilize enough to permit a more thorough assessment, including imaging. These include diagnosis such as ectopic pregnancy, rupture of a corpus luteum cyst, ruptured hepatic tumors, rupture of a previously diseased spleen, or rupture of a visceral artery aneurysm.

Once hemorrhage has been considered and ruled out as a cause of the abdominal pain, attention may then be focused on other causes. During the development of the history and the physical exam, it is appropriate to initiate fluid replacement if there are indications that a deficit exists. Patients who are suffering from nausea and vomiting are best managed by insertion of a nasogastric tube and placed on continuous suction. A Foley catheter is necessary to monitor the effectiveness of IV infusion. Analgesics may be given, usually as small, frequent, intravenous doses. Patients who are stabilizing, with indications of reversal of hypovolemia and have a functioning NG tube in place may *then* be sent for imaging (three views of abdomen, CT abdomen or ultrasound of abdomen.)

Aspiration is a continuing threat to patients with an acute abdomen. The darkness and sometimes remote area of the Xray suite is poor location to suffer a massive emesis.

Obstruction of the small bowel is particularly dangerous in this respect. The NG tube will not confuse the xray findings, and its proper position can be confirmed on the films.

CT of the abdomen has emerged as a definitive imaging in the investigation of SBO. Look for *complete* obstruction. If present, the correlation with strangulation is very high. Complete the resuscitation and proceed to OR.

Laboratory test results should soon be available. A differential and provisional diagnosis can then be developed based on the evidence from the history, physical exam, imaging and laboratory results.

Ischemia of the gut, although not as likely to cause sudden death as a ruptured abdominal aortic aneurysm, is associated with a very high mortality rate. Mesenteric vascular occlusion usually occurs proximally in the superior mesenteric artery leading to ischemia or infarction of the entire mid-gut. Ten to 20 cm of proximal jejunum may be spared, as is the left colon. Most of these patients are elderly, with significant collateral diseases (diabetes, coronary artery disease, peripheral vascular occlusive disease, renal failure). If a laparotomy is performed and infarction of the mid-gut is found, it would not be an appropriate decision to resect the infarcted bowel, leaving these frail patients only ten inches of jejunum and the left colon. This length of bowel is insufficient gut to allow maintenance of even fluid balance, much less provide adequate nutrition.

Unfortunately, very few of these patients with proximal occlusion of the SMA are diagnosed and have an appropriate intervention quickly enough to save the ischemic bowel. Results have been best in centers where there is an interest in this specific condition and management protocols are in place. Given the relatively short warm ischemia time for irreversible hypoxic injury to the bowel, many patients do not present until after the bowel is infarcted.

An all to common scenario is that of a frail,elderly vasculopath who suddenly developes agonizing general abdominal pain .Distention may not be signifigant, and the degree of rigidity or guarding may not impress the examiner.

Vital signs tend to deteriorate earlier than in cases of mechanical SBO,. The patient may prove difficult to stabilize, remaining acidotic and hypotensive, despite concerted efforts.

Some cases of bowel ischemia can be salvaged, however, and it is disappointing to miss these opportunities. Push quickly through the imaging and to the operating room.
This is a *vascular* complication and a direct approach to the occlusion of the mesenteric vessel will increase the chances of a favourable outcome; i.e. get the opinion of a vascular surgeon early and do the correct imaging; a clear demonstration of the anatomy of the occlusion will be very helpful in the OR

Ischemic bowel may appear congested, as above, or it may exhibit diffuse pallor.The bowel in the two cases to the left was ischemic but viable; the most likely etiologies of this picture are a stenosis of the proximal SMA or a low flow state.

Restoration of blood flow is the operative treatment of proximal stenosis. Resection alone, leaving the vascular occlusion in place, is a poor option, even if a signifigant length of gut seems to sustain perfusion..

Another salvageable situation presents when a shorter segment of intestine is ischemic or infarcted. Often the explanation for the the loss of perfusion to the affected region is obscure [? Small, peripheral embolus, thrombotic disorder, low flow state]

Diagnosis may be difficult. Obstruction may not be complete and peritonitis is slow to evolve as the infarcted bowel retains mechanical integrity for some time, often for several days, before frank necrosis and perforation occurs.

Opportunities to successfully manage ischemic bowel are often lost when the diagnosis is not considered and patients are identified as having "small bowel obstruction" and treated expectantly with intravenous fluids and analgesics. By the time hypotension, acidosis and anuria have evolved, the situation is irretrievable.

As in the instances of intra-abdominal or retroperitoneal hemorrhage, ischemic bowel needs to be included in the early differential diagnosis and then excluded by timely diagnostic imaging if there is sufficient suspicion.

Once the threatening conditions of hemorrhage and bowel ischemia have been considered and excluded, management of the other causes of the acute abdomen may be proceeded with. Obstruction of a hollow viscus, perforation of a viscus and localized inflammatory processes should now be given sequential consideration. Although many of these conditions are also life threatening and will require a timely operative intervention, they do not present the same potential for sudden death or mortal loss of an organ system.

Obstruction of a hollow viscus usually presents with pain suggestive of colic. The patient is restless and even agitated and the intensity of the pain varies..

Acute cholecystitis begins as biliary colic. The patient is restless and complains of severe epigastric pain, usually radiating to the RUQ and scapular region.
They are afebrile with stable BP and little elevation of pulse rate. There is no evidence of volume depletion.
If the stone remains impacted, inflammatory changes slowly evolve. A low-grade fever develops and the patient has RUQ tenderness. A mass may eventually be palpable
Because of the pressure in the lumen, patchy necrosis of the GB wall occurs and a localized perforation will eventually follow.
US confirms the diagnosis Early OR [48 hrs] is preferable to conservative strategies.

Mechanical small bowel obstruction is usually incomplete. For this situation to occur, the compressing band needs to be softer [omentum, in this case] There is no internal hernia, hence the mesenteric vessels are not compromised.

Proximal dilation is not as marked, as bowel content continues to get by the compression point. Bowel sounds may be audible without the stethoscope as peristalsis is quite violent[note the hemorrhage on the antemesenteric border]

Continuous NG suction is a *critically* important part of conservative management. Approx. 60% of incomplete obstructions will resolve with "suck and drip"

"It's a bowel obstruction. See the fluid level"

If the intestine contains a mix of air and fluid,. "fluid levels" may be seen on an upright abdominal film. A fluid level will usually be evident in the stomach but gas and liquid normally will pass through the small bowel so rapidly, and gas bubbles are so small, that they cannot be discriminated radiologically. Gas is always seen in the colon but fluid levels are rarely seen.

Fluid levels seen on upright radiographs *are not pathognomonic of obstruction Alternative* causes include ileus of any cause, gastroenteritis, purgatives and ischemia. Paradoxically, a complete SBO may display no fluid levels if the intestine is completely filled with fluid.

The signifigance of fluid levels on upright films should be assigned only after the "total" clinical picture has been elucidated.

Obstruction of a hollow viscus (biliary system, small or large bowel or ureter) will include a subset of situations which do not immediately and seriously threaten the patient. For example,

1. Stone in the cystic duct causing biliary colic and acute cholecystitis.
2. A simple obstruction without strangulation of the small bowel.
3. A neoplastic or diverticular stricture with obstruction of the colon. Obstruction is incomplete, or the ileocecal valve is incompetent.
4. The passage of a ureteral calculus.

However, there are also scenarios which evolve which increase the risk and need to be identified as separate from the above.

1. A stone in the cystic duct with empyemia of the gallbladder or emphysematous cholecystitis.
2. A stone in the common bile duct with ascending cholangitis and septicemia.
3. A complete strangulating small bowel obstruction.
4. A complete colonic obstruction with competent ileocecal valve and impending cecal rupture.
5. A colonic volvulus with ischemia.
6. A complete ureteral obstruction with pyonephrosis.

A stone in the CBD is a serious complication of cholelithiasis Pancreatitis and cholangitis are much more threatening problems than acute cholecystitis. The epigastric pain of biliary colic is similar for a stone in the gallbladder neck and in the ampulla, but rigors, high fever and jaundice indicate that the patient has CBD stones and cholangitis. Bacteremia is the rule and septic shock should be anticipated.. Effective volume expansion and antibiotics may result in some improvement, but decompression of the CBD is mandatory.
  There is nothing wrong with an open surgery and choledochotomy; The CBD may be filled with pus. Just place a T-tube and close.-The patient is often too ill for formal duct exploration and cholangiograms.

A small bowel obstruction which has an acute onset, with no flatus passed since the onset of pain, should be considered to be complete. When volume expansion is well underway, and the NG tube is positioned, imaging should happen-start with 3 views-if there is still doubt about the completeness of the obstruction, obtain A CT of the abd/pelvis
  If the obstruction is complete, .proceed to the OR *Do not "sit" on this problem. The mortality is 6-10 times higher if the strangulated bowel is gangrenous..*

Large bowel obstruction, often gradual in onset, may suddenly become complete if fecal material plugs the remaining passage. Should the ileal cecal valve be competent, the cecum can become so distended that it bursts,. releasing enormous amounts of gas into the peritoneal cavity. Massive fecal peritonitis is also the rule;, survivorship is unlikely.

   This patient had an obstructing cancer of the hepatic flexure. The cecum ruptured, but fortunately for this frail individual, fecal soiling was minimal.

   In large bowel obstructions with marked cecal dilation and no dilation of the small bowel [competent valve] the situation is emergent.

Sigmoid volvulus often presents in a recurrent and seemingly manageable way [endoscopic decompression]. Occasionally a critical situation evolves when distention and compromise of the mesenteric vessels leads to patchy necrosis of the colon. Ventilation may be inadequate in the presence of high intra abdominal pressures. Attempts to sigmoidoscope unstable patients who may have sigmoid ischemia are ill-advised. Stat laparotomy, trans-anal intubation and decompression of the loop, resection of the sigmoid and end colostomy may be the best option in this latter group of patients.…

Amongst patients with perforated viscus there are also subsets of variable degrees of risk and therefore a variation in the urgency with which diagnosis and intervention must occur. The outcome in these individuals varies with the site of the perforation, the underlying pathology, the age of the patient, and collateral diseases. The major issue influencing the pace of early management is the degree to which the perforation has been contained or localized by peritoneal defense mechanisms.

1. Perforation of peptic ulcer; contrast via a nasogastric tube shows evidence of a duodenal ulcer but no continuing leakage.[there may be an option for conservative management]
2. Appendiceal perforation with a localized abscess.
3. A diverticular perforation with a localized pelvic abscess.

Scenarios of greater urgency include:
1. Perforation of a peptic ulcer with free air and fluid and continuing extravasation demonstrated.
2. Perforation of the small bowel suspected.
3. Perforation of the appendix with a spreading peritonitis.
4. Perforation of a colonic diverticulum with generalized peritonitis.

Peptic ulcers may perforate through a minute hole or the perforation may be several centimeters in diameter. Gastric ulcers are frequently large.

This large perforated ulcer was on the anterior wall of the gastric antrum. It was poorly contained by omentum, and then leaked freely into the peritoneal cavity. There was a huge pneumoperitoneum.

What are the surgical options here?

This elderly, diabetic patient had presented with a hugely distended and tender abdomen. She was confused and hypotensive and did not respond well to IV fluids and antibiotics Xrays demonstrated free air under both diaphragms.. She went to the OR within hours of admission. A generalized peritonitis was present originating from a perforated diverticulum in the sigmoid colon. A sigmoid colostomy with resection of the perforated segment was performed.
The patient remained hypotensive and oliguric. She developed respiratory failure. The situation was considered hopeless and care was withdrawn. Aged patients with comorbid disease may simply be overwhelmed by a generalized peritonitis originating from a colon perforation.

Appendicitis may follow an "aggressive" course, perforating within 24 hrs. of the onset of symptoms, or the process may linger over several days. The path the disease will follow is not predictable.
The "typical" history may not be elicited or rhe appendix is in a retrocecal position and guarding over McBurney's point may not be impressive.
CT or ultrasound, if available, may help with difficult cases. Laparoscopy is another option. CAREFUL observation in hospital is not a bad further alternative.
Quick dismissal from the ER with the diagnosis of constipation, 'flu" or gastroenteritis gives neither the patient or the MD a chance.

This patient had an unusual condition with multiple diverticulae scattered through the small bowel. For reasons unknown one of these diverticulae perforated causing a spreading peritonitis. Whatever the cause, acute perforations of the small bowel are poorly contained and contamination spreads rapidly. There are really no medical options. Early surgical control is necessary if a satisfactory outcome is to result.

Inflammatory processes cause acute abdominal pain. Early operation may have little to offer when these processes are well contained and they may be manageable by intravenous antibiotics or by other conservative strategies [percutaeneous drains].

Pancreatitis, usually caused by excessive alcohol ingestion or passage of a common duct stone, may be self-limited and may subside after a few days of intestinal rest, intravenous therapy, and analgesics. Surgical interventions in pancreatitis would be reserved for the erasure of calculus disease by cholecystectomy, cholangiograms and exploration of common duct, or for the complications of the more serious variants of pancreatitis. These procedures are usually delayed until the pancreatitis has settled (cholecystectomy), or are not indicated until later in the course of the disease, as in the instances of necrotizing pancreatitis.
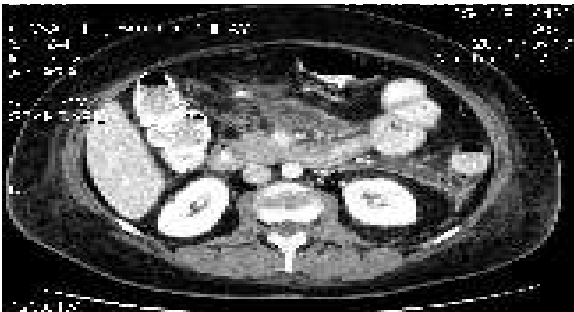
Pancreatitis is usually readily diagnosed by serum amylase elevations, usually in the order of two to three thousand. If there is still concern regarding the possible diagnoses of perforated viscus and ischemic bowel, then CT imaging of the abdomen should reveal the pancreatic inflammation and peripancreatic fluid.

Although a small fluid collection is present anterior to the pancreas, the course of the disease is unknown and a surgical intervention at this early stage may be unnecessary and increase the patient's morbidity..

The more serious pancreatitis of the necrotizing form is identified by the Ranson criteria. Very aggressive IV therapy may be required to support plasma volume and respiratory and renal failure may be quick to evolve.
 The diagnosis of pancreatitis will usually be confirmed by a marked serum amylase elevation at the time of presentation, but CT may be required as well, to identify the pancreatic phlegmon and rule out perforated viscus, volvulus, and mesenteric vascular occlusion.

More severe forms of pancreatitis, requiring a major resuscitative effort, exhibiting degrees of respiratory and renal failure, should be followed carefully for the development of retroperitoneal collections.These may be sterile initially, but often become colonized[?by the process of translocation]

Patients with a chance of survivorship sequester these "abscesses" which contain a grey-black mush and small gas bubbles generated by bacteria.

Open surgical drainage of these collections will be required, but usually at 2-3 weeks.

Pelvic inflammatory disease may spread rapidly throughout the peritoneum and cause alarming physical signs, with marked leukocytosis. A thorough gynecologic and obstetric history may raise suspicion and imaging may assist further. At most, a diagnostic laparoscopy may be necessary. These patients are usually stable, and gastrointestinal symptoms are often minimal, considering the worrisome features of the abdominal examination.

Diverticulitis often presents with an acute process in the left lower quadrant. There is localized tenderness and sometimes a tender mass. The condition is manageable by intravenous antibiotics and may resolve completely or at least allow bowel preparation and a single stage resection. Imaging with CT is useful to define the extent of the process. Cases suitable for antibiotic management usually show only thickening of the colon wall, diverticulae, and mesenteric stranding or small pericolic fluid collections.

This 50 yr old male had suffered from cramping lower abdominal pain for several years. A barium study had shown extensive sigmoid diverticulosis..
He later presented to the ER with acute onset of localized pain in the LLQ. He was febrile, with impressive tenderness, guarding percussion rebound, confined to the LLQ.
The CT scan confirms the diverticular disease and demonstrates a small abscess behind the LLQ abdominal wall
The imaging is very helpful in supporting a decision for non-operative management

Appendicitis may follow a subacute course and the patient may not present for medical care, or the diagnosis may be missed. In these instances, an abscess may evolve or there may be an inflammatory mass. The distinction between these two diagnoses can usually be made by imaging, either US or CT scan. An abscess can be drained operatively or by radiologic intervention. An open operative approach to an inflammatory mass, attempting to identify and remove the appendix, may be ill-fated, resulting in injury to small bowel, ureter or major vessel. The base of the appendix may be difficult to secure. The operation may escalate, eg. to right hemicolectomy.

If imaging indicates there is a localized inflammatory mass, the best approach is usually conservative. Surprising resolution of the process usually results.A "delayed" appendectomy, otherwise called "interval" appendectomy, may be planned in 6-8 weeks.

Cecal diverticulitis is an uncommon condition, resulting from perforation of a solitary diverticulum of the cecum or ascending colon. These diverticulae do not share the same pathogenesis as the much more common diverticular disease of the colon, usually dominate in the sigmoid.

The solitary diverticulum has probably been present from birth, so the complication of inflammation occurs in younger individuals. It is virtually indistinguishable from appendicitis, other than that the pain begins in the RLQ or flank. Tenderness or mass may be higher than the usual location for the appendix. A CT scan, if done, may demonstrate the normal appendix and the localized inflammation, often lateral to the ascending colon.

Triage

Comes by ambulance appears
ill and distressed, older, pulse and temp.
elevated B.P. low

Walks in; young,
stable, afebrile

Acute area
Establish monitoring, Start IV, take venous blood,
Nursing assessment, Physician sees.

Assess A, B, C. and begin management:
- bolus with IV fluid [saline] if volume depleted.
- ABG's if indicated.
- Elicit the basic elements of the history
- Examine the abdomen. *Have a differential
diagnosis already developing.*

Consider hemorrhage as cause of the acute abdomen

Ruptured AAA → To OR with Vascular Surgeon

Other causes of hemorrhage
[ectopic. ruptured hepatoma]

Continue IV fluid boluses,
- insert foley catheter
- blood transfusion ?
Try to stabilize. Depending on possible
cause will need imaging [CT angio.
angiography,] or OR

Consider ischemic bowel as the cause of the acute abdomen
Severe abdominal pain, out of proportion to physical signs

Proceed with imaging [3 views abdomen, CT angio, selective angiography]

Proximal occlusion, severe stenosis   To OR

Low flow [non-occlusive]   ICU for optimization Cardiac Output Papaverine infusion by selective catheterization of SMA

Consider perforated viscus

Insert an NG tube-insure gastric position
Proceed with imaging [3 views abdomen, CT abdomen]

Generalized peritonitis, or retroperitoneal gas,fluid
                        To OR

Inflammatory mass.
Localized abscess.
IV antibiotics, percutaeneous drain if indicated.

Consider obstruction of a hollow viscus

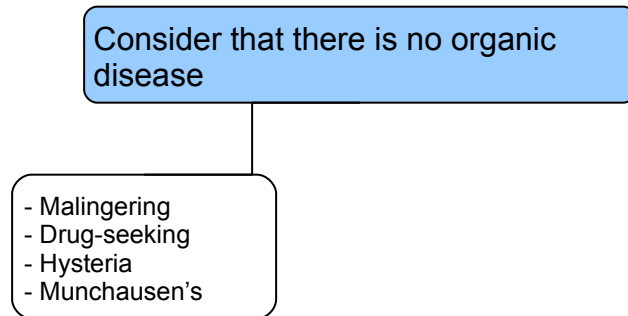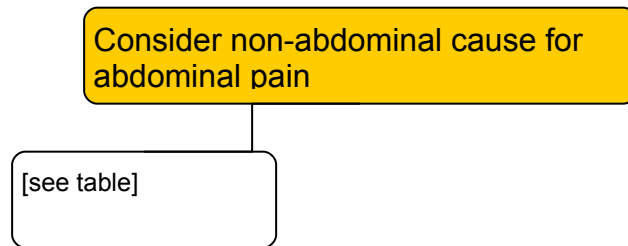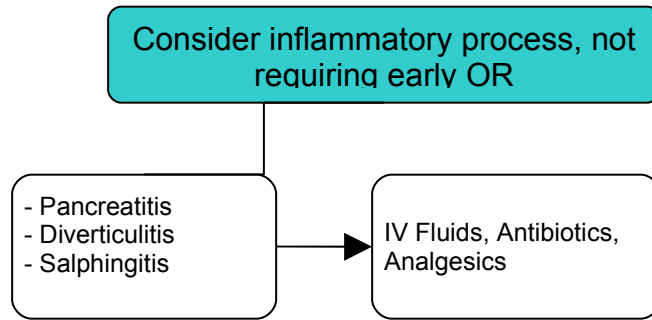Insert an NG tube [biliary conditions may not require]

Proceed with imaging [3 views abdomen, CT]

Complete SBO
L colon obstruction with competent ileal-cecal valve
Volvulus or intususception SB
CBD obstruction with cholangitis
"septic" gallbladder [empyemia, emphysematous]
ureteric stone,pyonephrosis

Cholecystitis
Incomplete SBO
Colon obstruction,ileal cecal valve incompetent
Ureteric stone,no sepsis

Conservative or expectant mgt.
                  or
Urgent, but not emergent OR

To OR

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Consider inflammatory process, not
requiring early OR
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- Pancreatitis        →    IV Fluids, Antibiotics,
- Diverticulitis            Analgesics
- Salphingitis
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Consider non-abdominal cause for
abdominal pain
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[see table]
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Consider that there is no organic
disease
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- Malingering
- Drug-seeking
- Hysteria
- Munchausen's
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For the first episode, the best approach is again conservative, with IV antibiotics directed at anerobes and coliforms.

Table 1. NONSURGICAL CAUSES OF ABDOMINAL PAIN

Cutaneous/abdominal wall
  Varicella-zoster infections
  Compressive sensory radiculopathy
  Rectus sheath hematoma
Toxicologic
  Iron
  Alcohols
  Lead
  Narcotic withdrawal
  Caustic ingestions
  Mushrooms
Metabolic/endocrine/genetic
  Hereditary Mediterranean fever
  Porphyria
  Glucocorticoid deficiency
  Hypercalcemia
  Diabetic ketoacidosis

Envenomations
  Black widow spider bites
Cardiopulmonary
  Myocardial infarction
  Pneumonia

Intra-abdominal infections/inflammation
  Mesenteric adenitis
  Spontaneous bacterial peritonitis
  Catheter-related bacterial peritonitis
  Infectious enteritis
  Abdominal syphilis
Urologic
  Epididymitis
  Prostatitis
  Nephroureterolithiasis
Intra-abdominal vasculitis
  Henoch-Schonlein purpura
  Rocky Mountain spotted fever
  Systemic lupus erythematosus
 Hematologic
  Sickle cell anemia
  Hereditary angioneurotic edema

Neurologic
  Abdominal migraine
  Abdominal epilepsy

Although the list of conditions causing abdominal pain, but having no surgical solution, is formidable, they should be discriminated without too much difficulty. These conditions are rarely encountered, but the careful gathering of the history of the present and past illnesses, using all sources[relatives, family practitioners, medical records], will be rewarded. Also key to their identification is the recognition, that in spite of what appears to be severe abdominal pain, there is a paucity of objective findings indicating a "genuine" surgically correctable disease in the abdomen.

Percussion tenderness [rebound] is absent, no mass can be felt. Normal bowel activity may be auscultated. Imaging studies fail to show abnormality.

In difficult cases, diagnostic laparoscopy will often prevent an unnecessary laparotomy.
In Table 1, rectus sheath hematoma is included. There is serious bleeding in some cases, and surgical control may be necessary [ligation of inferior epigastric artery]

No Organic Disease Present

A surprising number of individuals present to emergency rooms, even by ambulance, claiming severe abdominal pain "ten out of ten". Narcotics seem inevitably required for partial relief. Some of these patients are frequent flyers at the same hospital, but others rotate through different ERs.

Where records are available, this behavior is well documented. Many of these patients have had previous surgery; hiatus hernia repair, cholecystectomy for acalculous gallbladder, incidental appendectomy or hysterectomy for fibroids. In hindsight, the indications for these procedures may seem "shakey", but it is not fair to blame the patients for this. It is difficult to quickly dismiss these individuals without a basic workup, and indeed this should not occur. A minimum assessment would include history and physical exam, CBC, electrolytes, creatinine and amylase, plus 3 views of the abdomen. If there are no positive findings they may be treated symptomatically. Fortunately the crisis usually abates, allowing discharge "in the AM" There is no solution to this ongoing, resource consuming problem; if one patient like this appears to have a period of respite, another takes his/her place.

One can only speculate as to the cause of pain in these instances. Usual diagnoses are "partial

SBO", pancreatitis [amylase-130], or 'irritable bowel syndrome'..

There should be no apologies for a high-end investigation [such as CT scan or even a laparoscopy] Needless to say, a negative *laparotomy* would be a very unfortunate event

Another cluster of individuals actually seek medical care for gain, and will even submit to an invasive investigation if such is recommended to them. To this end, they may offer an impressive history and display convincing tenderness and guarding. The "gain" referred to above may be administration of narcotics, continuation of financial support from social agencies, a reprieve from prison life or simply the attention and sympathy of others.

A rare condition is known as" Munchausen's syndrome" Persons afflicted with this rare condition differ from malingerers in that they lack a clear understanding of the reason for their behaviour. These individuals wander the country, surfacing in hospital ER's complaining of severe abdominal pain and exhibiting a rigid abdomen, very suggestive of a perforated viscus. The abdomen is usually marked by numerous surgical scars. The patient will relate that all of the previous procedures were emergencies and for "perforated bowel". Typically, the surgeries have all been in different centers.

One should be alerted by the above elements of the history and examination, and place appropriate reliance on *objective* physical signs, *laboratory tests* and *imaging.*

If one's suspicions are aroused, a long distance phone call to one or more of the hospitals named by the patient may be very rewarding.

History Taking in Patients with an Acute Abdomen

As previously described, the taking or a history, performing a physical examination and initiating monitoring and management may all have to occur simultaneously. Obviously a prolonged interrogation of a hypotensive patient who is vomiting and in severe pain, without attempting any resuscitation or other necessary interventions may not only waste critical time, but also exposes the patient to unnecessary suffering and risk.

The duration of the pain and the other associated complaints need to be clearly defined. It is useful to determine the region of the abdomen where the pain was first felt. A description of the rate of onset and how the pain spread throughout the abdomen may also be helpful. If pain is felt in the flanks or in the back, this suggests a retroperitoneal process. Generalized abdominal pain or periumbilical pain suggests that there is an origin in the small bowel, colon, or appendix. Pain which is in the epigastrium and radiates towards the right upper quadrant or sometimes the scapular area will usually signal biliary origin. Pain arising very abruptly in the upper abdomen, which is severe and spreads rapidly to involve other quadrants may be experienced by patients with a perforated peptic ulcer. Perforation of a sigmoid diverticulum will result in left lower quadrant pain which may spread to a variable extent to involve other quadrants of the abdomen. Appendicitis has a characteristic pattern beginning as periumbilical colic and then shifting to the right lower quadrant where it exhibits features of localized peritonitis.

Appendicitis is often precipitated by obstruction of the base of the appendix [by a fecalith, in this case]
The patient experiences a colic type pain in the central abdomen, often associated with vomiting. As localized inflammatory changes evolve around the appendix, tenderness and guarding can be elicited over McBurney's point. Eventually, the development of peritonitis will be demonstrated by rebound tenderness [elicited by light percussion!]
Pressure in the LLQ may cause pain in the RLQ[Rosving's-another good objective sign]

Acute cholecystitis begins to evolve when a calculus impacts, either in Hartmann's pouch or in the cystic duct. The patient experiences a colicky quality of pain, which is usually felt initially in the epigastrium and then gradually migrates towards the right upper quadrant and also may be experienced in the right scapular area. As mentioned in the text, these patients are often extremely restless and uncomfortable. Gradually, with increasing pressure and changes in the chemistry of the obstructed bile, the gallbladder wall becomes progressively more edematous and thickened.

In this patient, encountered prior to the advent of laparoscopic cholecystectomy, open surgery was commenced and a tense and distended gallbladder was found.

At this stage, patients may not exhibit significant right upper quadrant tenderness. As pressure in the lumen of the gallbladder increases, then some local inflammatory changes start to develop around the gallbladder and localized tenderness in the right upper quadrant may be found. If the gallbladder becomes encased in inflamed omentum, a mass may also eventually be palpable.

It is preferable to make a diagnosis of a calculus gallbladder with acute cholecystitis early and to intervene surgically at this time, when the gallbladder is edematous and tense but before advanced inflammatory changes occur. The gallbladder does not have advanced inflammatory changes and a dissection of the critical structures should not be difficult. Should this procedure have been performed after the introduction of laparoscopic cholecystectomy, it could have been completed, sparing the patient the subcostal incision.
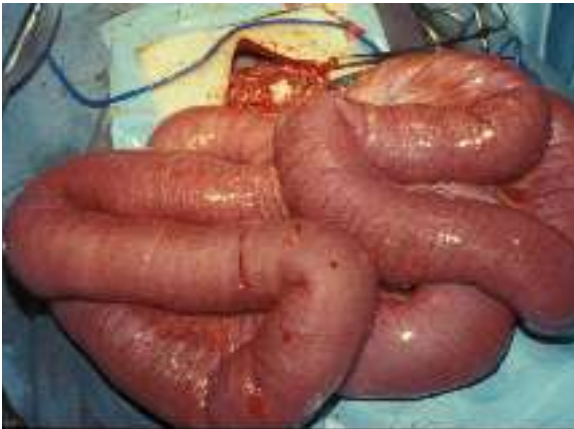
Small bowel obstruction is usually heralded by the onset of generalized abdominal cramps. While the intestine still has significant vigor and is not dilated, the cramps may be of considerable severity. Gradually the intestine dilates with its voluminous secretions and swallowed air and the intensity of the cramps may diminish somewhat. If the obstruction is in the mid-small bowel or proximal, frequent vomiting is the rule. Intestinal obstructions, as in this case, usually evolve very significant degrees of distention. If the obstruction is complete, flatus ceases to be passed soon after the onset of pain.

Characteristic physical signs are various degrees of abdominal distention with tympany. Some degree of abdominal tenderness can usually be elicited, but marked degrees of guarding and rigidity are not the rule. Caution should be used in interpreting the finding of some mild rebound tenderness, as this can be experienced when the intestine is markedly dilated.
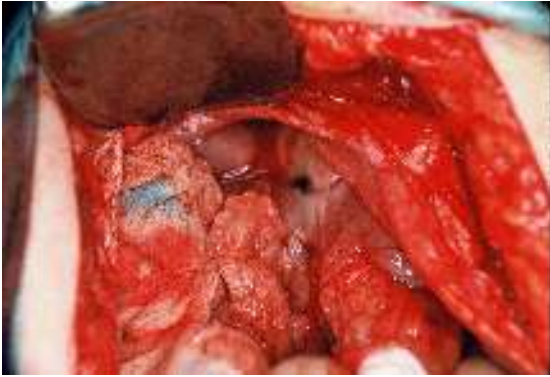
Probably the most common cause of perforated viscus is an anterior duodenal ulcer. Many of these ulcers evolve quickly, frequently under the stress of alcohol intake, salicylates, fasting, and other abuses. Sometimes a history of prior upper abdominal pain relieved by antacids can be elicited. Perforation usually occurs, as in this case, in the middle of the anterior wall of the first part of the duodenum. Bile and pancreatic juice rapidly spreads through the abdomen, inciting fierce peritoneal reaction. If the spillage reaches the remote quadrants of the abdomen, a generalized abdominal pain of severe degree evolves. The physical signs are characteristically that of a completely rigid abdomen. The degree of rigidity in the early stages is usually considerably more than that which can be elicited in some other forms of bacterial peritonitis.[key word for the student to recall on oral exams-"board-like".]

Obstruction of hollow conduits (bile ducts, ureters, and small and large bowel) incites a colic, which can be both observed and elicited in the history. These patients are characteristically restless and unable to find a comfortable position. The pain may intensify and then subside, providing brief periods of respite.

Individuals with chemical and bacterial peritonitis of spreading or generalized nature complain of sharp accentuation of their pain with any sudden movement of the abdominal wall. Coughing, walking, or even a change of position will worsen this form of abdominal pain, causing grimacing or verbalizing. Patients are often observed to adopt a fetal position and are henceforth very reluctant to move or to be moved. Assuming a completely supine position with the hips extended and the spine straight may be difficult. Individuals with peritoneal contamination reaching the diaphragmatic surfaces may complain of pain on the top of the shoulders, accentuated by deep breaths or coughing.

If peritonitis continues, whether it initially be chemical followed by bacterial or bacterial and fecal from the outset, and no interventions are taken, a generalized purulent process may evolve, with purulent membrane caking the surface of the bowel and parietal peritoneum Multiple interloop abscesses with partial bowel obstruction and ileus can evolve.

By this time the patients have usually developed degrees of volume depletion and manifest significant sepsis. The abdomen is distended and silent. Generalized tenderness and guarding can usually be elicited. If there are pelvic abscesses, they maybe felt by rectal examination.

An acute abdomen presenting in an emergency room often occurs towards the end of the natural history of GI pathology. This pre-existing problem may give rise to significant symptoms for some time and these may provide a clue as to the source of the eventual acute abdominal pain and emergency room admission. A careful inquiry may elicit this history. Some examples would be repeated short-lived episodes of biliary colic, which may precede impaction of a stone and evolution of acute cholecystitis. Epigastric pain relieved by food or antacids may eventually culminate in a free perforation of a peptic ulcer. Episodes of generalized abdominal cramps, distention and infrequent bowel motions may preceed an eventual complete colonic obstruction. Other collateral diseases are often associated with the development of a catastrophe in the abdomen. History of cardiac failure, arrhythmia, myocardial infarction or valvular disease may alert the examiner to the possibility of ischemic bowel if there is an acute abdomen. Hypertension, occlusive vascular disease and obesity may be associated with aneurysmal disease and should provide a warning of the possibility of either ischemic bowel or a leaking abdominal aortic aneurysm.

Nausea and vomiting is a very common associated complaint in persons with acute abdominal pain. Vomiting commonly occurs in patients with a small bowel obstruction and with pancreatitis. It is likely to be repeated frequently in these conditions. Other causes of acute abdomen may result in some nausea or one or two episodes of vomiting, but is unlikely to be sustained. GI bleeding is quite infrequent as an associated complaint in patients with acute abdomen. Repeated passage of liquid stools is a feature of gastroenteritis and is seldom described by patients with acute abdomens of surgical importance.

It is important to obtain or elicit an accurate record of any previous abdominal surgeries. If possible, operative and pathology reports should be obtained from medical records departments.

Current medications are important both to the management of collateral diseases, such as hypertension or cardiac failure, and also as potential contributors to the cause of an acute abdomen. NSAIDs, salicylates and corticosteroids may result in progression of peptic ulcer disease and secondary complications. Steroids also interfere with the peritoneal defenses to the spread of infection and increase the risk of any acute abdominal process.

Alcohol abuse will not only cause pancreatitis, but also may contribute to peptic ulcer perforation or result in collateral conditions, such as liver failure, which will greatly increase the risk of the primary abdominal process.

Physical Examination

Physical examination of the acute abdomen will begin when the patient is first encountered and continue while the history is being elicited and resuscitative measures are being initiated. The patient will be observed from the outset for any evidence of respiratory difficulty, confusion or delirium, or any indication of significant volume depletion. Features such as diaphoresis, pallor, mottling, tachycardia, collapsed peripheral veins, and hypotension will be noted during the initial moments of the encounter. The patient's nutritional status will be observed and other general features, such as peripheral edema or jaundice, noted.

During the initial parts of the examination considerable information can be gathered just by observing the patient's movements, or lack thereof. eg. Patients who constantly change position or alternate between periods of relative comfort and writhing about will be suffering from colic due to obstruction of a hollow conduit

Rather than making a direct approach to the abdomen, it is sometimes a useful strategy to conduct some peripheral parts of the examination first, including a quick examination of the head and neck, inspection of the throat and palpation for any cervical nodes or other masses. The trachea should be assessed as to its position in the midline. The chest can then be auscultated, as well as the heart sounds.

The patient should then be positioned for examination of the abdomen. The abdomen should be examined with the patient completely supine. Slight flexion of the hips may be allowed if the patient requires it for relief of pain. The sheets and the patient's gown can be used to keep the patient comfortable, however, the entire abdomen, including the inguinal area, genitalia and upper thighs, should be included in the inspection at some point. The patient should keep arms and hands at his or her side.

On inspection. observe the contour of the abdomen, the presence or absence of any surgical scars, visible masses, pulsations, and the movement of the abdominal wall with respiration.

Patients with very distended abdomens are usually tympanitic to percussion, suggesting distention of the gastrointestinal tract with gas, although rarely a pneumoperitoneum may explain gaseous distention of the abdomen.

Patients with left-sided colon obstruction, as in this case due to carcinoma of the rectosigmoid, may evolve a marked degree of tympanitic distention of the abdomen. This process may develop quite quickly, within 2-3 days of the presentation to hospital. Often patients have struggled with infrequent bowel motions and abdominal cramps for several weeks prior to the onset of a high-grade or more complete obstruction.

The abdomen is usually very tense, but marked degrees of guarding and rigidity are usually not demonstrable. The tumor itself is usually small and of a stricturing variety. It is seldom possible to palpate the obstructing lesion. These tumors are usually above the reach of the finger on digital rectal examination.

In cases of volvulus, as in this instance of volvulus of the right side of the colon, an asymmetric distention may be noted. The cecum in this instance was located in the left upper quadrant of the abdomen and the ascending colon lay across the upper abdomen and down towards the right lower quadrant. The onset of this problem may be sudden, but there may be a history of previous episodes of abdominal pain and intermittent distention, suggesting prior episodes which spontaneously resolved. Again tenderness and guarding are not usually a feature, but if they can also be demonstrated, this would suggest impending rupture or ischemia of the twisted segment.

In some cases of sigmoid volvulus an enormous degree of distention of the colon may evolve. This patient was not an aged individual, being about 45-50 years of age. He took psychotropic drugs for many years for mood disorders and it is possible that these affected the motility of his colon. In any event, the volvulus precipitating his final admission was acute in onset and with such a marked degree of tympanitic abdominal distention that his ventilation was compromised. An emergency open surgery with decompression of the volvulus through the rectum was required.The sigmoid was resected easily as it collapsed like a sack [90 mm TA stapler needed] A temporary colostomy was established.

Asymmetric tympanitic distention of the abdomen may be related to volvulus. Visible masses which are tender may be inflammatory in origin or can represent a loop of incarcerated and strangulated intestine. Incarcerated hernias are usually obvious if the region is exposed. Palpation or digital examination of the inguinal canal may be required to identify a small incarcerated hernia in an obese individual

   Bruising or ecchymosis.may be seen in the flanks or near the umbilicus in pancreatitis.Rectus sheath hematoma may result in ecchymosis in the suprapubic region.

   In slender individuals, distended, peristalsing loops of bowel may be seen in intestinal obstruction.

On palpation of the abdomen, the intent is to assess the degree of guarding by the abdominal muscles in response to downward pressure and if possible, to try to localize this resistance to one region of the abdomen. The initial palpation should be very gentle, carried out with the flats of the fingers with pressure exerted by flexing the metacarpal joints. Rest the hand on the skin for a few seconds before exerting any downward pressure. Move slowly around the abdomen from quadrant to quadrant. Start in the area of the abdomen most remote from the patient's initial description of the worst pain. Be careful on releasing the pressure of the fingers, so as not to "rebound" the patient.

It needs to be recalled that the resistance of the abdominal wall is a subjective finding and can vary significantly between different patients. It can be created voluntarily by the individual, if they wish for some reason to simulate severe abdominal tenderness. The distinction between voluntary and involuntary guarding is not a valid concept. The finding of marked abdominal wall guarding and even rigidity needs to be correlated with other general and more objective physical signs, such as pulse rate, temperature and blood pressure.

Sudden onset of severe abdominal pain accompanied by the finding of a mass in the lower abdomen may suggest torsion of a pre-existing ovarian tumor.

Although the pain is severe, it is usually confined to the lower quadrants of the abdomen and is not associated with other significant gastrointestinal symptoms.

This diagnosis might be suspected in women who have been aware of a lower abdominal swelling or have periodically had spontaneously resolving episodes of lower abdominal pain. Usually the upper quadrants of the abdomen are spared and the process is confined to the lower quadrants, where examination may demonstrate a mass. A rectal examination and/or bimanual pelvic examination may further discriminate this large mass, which would certainly also be tender

Infarction of the mid-gut with evolving small bowel gangrene results in agonizing pain, but often surprisingly little in the way of peritonitis. Actual mechanical breakdown of the intestinal wall is usually delayed for some time. The absence of peritonitis explains the difficulty in eliciting the anticipated guarding and rebound that one might expect with such a catastrophic process. When the complaint of agonizing abdominal pain is not accompanied by impressive physical signs in the form of rigidity and rebound, evolving ischemia or infarction of the gut should be suspected.

Appendicitis in older people may sometimes be provoked by tumors in the cecum or ascending colon. In patients who seem to have a history very suggestive of appendicitis, are in older age groups, and have a suspicious anemia, this diagnosis should be suspected. On physical examination, although there may be guarding and tenderness in the right iliac fossa, with care, it may sometimes be possible to demonstrate the cecal mass. In this patient there is a tumor right at the pole of the cecum, obstructing the base of the appendix. It was sufficient to cause anemia, but was probably too small to be detected by palpation.

It is important in the course of the palpation to try to localize the intra-abdominal process, if possible, to at least one quadrant. Guarding in the right lower quadrant will usually involve the

ileocecal area and appendix , or adnexa in females. Similar findings in the left lower quadrant may signal the presence of pathology in the sigmoid colon, or again, pelvic pathology in a female. Right upper quadrant guarding and tenderness may be related to the duodenum or gallbladder. Localized tenderness in the left upper quadrant is quite uncommon.

Even in patients, who by the time of presentation, have a generalized peritonitis, there may still be increased degree of guarding in the area where the process originated. For example, a generalized peritonitis due to a perforation of a sigmoid diverticulum, although there may be guarding and rebound throughout the abdomen, this may be still more intense in the left lower quadrant.

"Rebound"

Patients who have exhibited great difficulties in changing positions or moving and have generalized guarding and pain increased by even careful release of abdominal pressure, will almost certainly have significant rebound tenderness. Repeated and aggressive attempts to elicit this physical sign elicits anger, apprehension or outright terror; further examination may be refused or rendered invalid.

If generalized peritonitis is suspected and the feature of rebound demands to be elicited, this can be done by light percussion, rather than attempting to depress the abdominal wall and then allowing it to snap up, causing the patient an agonizing flare of pain.

In patients with marked intestinal distention, depression of the abdominal wall and sudden release will cause a sudden increase in pain; "Heavy-handed" demonstration of rebound tenderness in these individuals is misleading and is discouraged.

If properly elicited, however, the finding of true rebound tenderness is valuable, as the finding has a degree of objectivity over tenderness, guarding and even rigidity..

Mention is often made that the patient has a "surgical abdomen". Rebound tenderness is usually weighed in as a major contributor to the evidence for a "surgical abdomen". It needs to be recalled that all patients exhibiting rebound, and presumably having a degree of peritonitis, do not require an immediate operation. Patients with salphingitis may have florid rebound. Acute

pancreatitis may cause enough peritoneal irritation to result in rebound. Early surgery does not benefit these individuals.
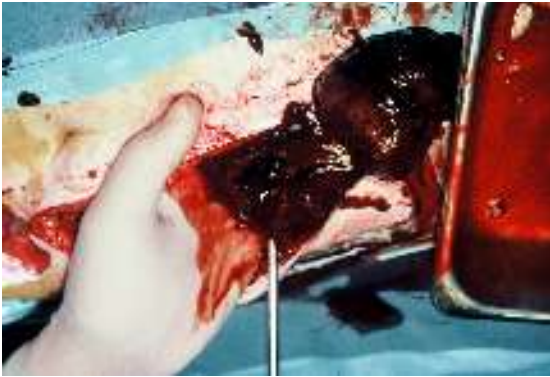
Pain and a mass in the lower abdomen will develop in patients with rupture and hemorrhage from the inferior epigastric vessels. This unusual diagnosis might be suspected in individuals who are anticoagulated and hypertensive. Often straining or coughing may precipitate the actual hemorrhage. A sizable mass may develop in the abdomen, the feature of which is that it does not cross the midline. On inspection, some bruising may also appear at the base of the genitalia where the hemorrhage seeps to the surface. There may be sufficient blood loss to actually cause hypotension and anemia.

In evolving acute cholecystitis the gallbladder may be encased in edematous and inflamed omentum. The combination of the omental mass, plus the distended gallbladder, will give rise in many patients to a tender mass in the right upper quadrant emerging from under the costal margin. It is a very good objective sign of acute cholecystitis.

In patients who present with crampy abdominal pain and appear to have a small intestinal obstruction, the abdomen should be searched carefully for a palpable mass. This would be particularly true in those individuals who have not had any prior surgical procedures on the abdomen and have no identifiable external hernias. In these individuals unusual causes such as small bowel tumors and intussusception, such as in this patient, may be the cause. In this patient a cylindrical mass could be appreciated in the central abdomen, confirmed to be intussusception by imaging.

Patients who guard to the point of rigidity throughout the abdomen but do not have other objective signs of an acute illness, may simply be uncomfortable with the examination or anxious. The rare patient is also manipulating, drug-seeking or psychotic.. In these individuals continued gentle downward pressure in the abdomen may gradually overcome the voluntary tensing of the abdominal wall. Coming back to the same area again may demonstrate inconsistency. Another strategy is to attempt to distract the patient by further questioning, on elements of the functional inquiry, for example. .

In some patients with an acute abdomen it may be possible to identify an abdominal or pelvic mass. Distended bowel loops, as in a closed loop obstruction or volvulus, may be palpable as a globular, smooth structure, providing the degree of distention is not too marked. Other masses felt in the acute abdomen include the inflammatory masses often developing around acute appendicitis, diverticulitis and pancreatitis.The distended gallbladder and surrounding omentum often becomes palpable in acute cholecystitis.

 A leaking abdominal aortic aneurysm may be seen and/or felt as a pulsatile mass above and slightly to the left of the umbilicus.

 Rectus sheath hematoma from rupture of the inferior epigastric artery may result in an impressive mass in the lower quadrants, difficult to distinguish from an intraabdominal process.

 Pelvic abscesses, originating from salphingitis, appendicitis or diverticulitis, are often felt on digital or pelvic exam. Torsion of an ovarian tumor will result in an acute abdomen and a pelvic mass.

 If the differential diagnosis includes conditions where it is known that a mass may be present or evolve, it is worthwhile to make a careful effort to demonstrate it, as a mass is one of few objective signs in the examination of the acute abdomen.

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Acute appendicitis in adults, which does not receive early surgical attention, will eventually perforate, but is often confined in the right iliac fossa by a combination of adherent omentum and adjacent loops of small intestine. Either an inflammatory mass or phlegmon may develop or one or more localized abscesses in the iliac fossa or pelvis. This mass is frequently palpable by careful overcoming of the abdominal wall guarding. Imaging techniques will be helpful in discriminating a phlegmon from an abscess.
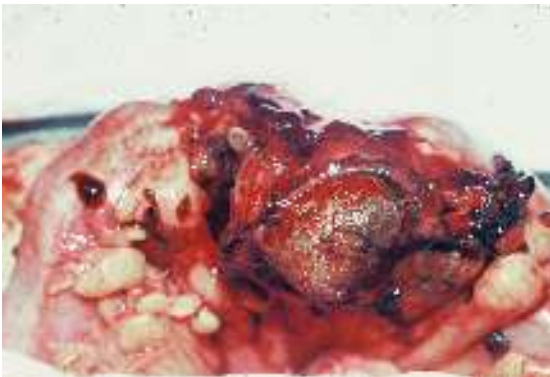
Perforation of the sigmoid colon usually results from diverticular disease and occasionally from ischemic colitis or inflammatory bowel disease. Rarely massive fecal impaction, stercoral ulceration and perforation can occur, as in this individual who was habituated to narcotics. A large perforation of the sigmoid exits and, as one would anticipate, the patient was febrile and tachycardic and exhibited marked degrees of tenderness and guarding in the left iliac fossa. The perforation was poorly contained and generalized peritonitis evolved, with diffuse rebound tenderness, again elicited by light percussion.

The inflammatory process in acute cholecystitis evolves more slowly than in appendicitis. The inflammation initially is usually sterile and mediated by chemical and pressure factors, rather than bacterial proliferation. Eventually, however, after several days of conservative management the gallbladder will begin to slowly deteriorate, with focal areas of necrosis. Eventually these will perforate. Often the abscess is localized to the right subhepatic space. In this case there was a large, right upper quadrant, tender mass.

Auscultating the abdomen for the presence or absence of bowel activity is often given considerable weighting. It is important to realize that bowel sounds may persist, even though a critical problem is evolving in the abdomen. Similarly, a silent abdomen does not dictate that a critical surgical problem is at hand and a laparotomy is required. Most patients with generalized peritonitis will soon develop a paralytic ileus and bowel activity will cease. Mechanical obstruction of the intestinal tract may at first be accompanied by increased bowel activity and audible bowel sounds, but if the obstruction progresses the bowel shortly becomes quiet and no further bowel activity can be heard. Localized processes in the abdomen which are contained, such as appendicitis and diverticulitis, will usually permit continued normal activity of the intestine. Critically ischemic, but still viable small bowel, may continue peristaltic activity.

DO NOT BE REASSURED JUST BY THE PRESENCE OF BOWEL SOUNDS.

"He's got bowel sounds."

A digital rectal examination should almost always be carried out. The pelvic peritoneal tenderness, pelvic mass, and the presence of blood in the stool, either gross or occult, may be found. A complete pelvic examination with inspection of the cervix by speculum and bimanual palpation of the pelvic viscera may not be warranted in all cases of acute abdomens. In many women with lower abdominal pain or any suspicion of gynecologic problems, obviously this component of the examination should not be omitted.

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As one would imagine, this patient with a strangulating complete small bowel obstruction was markedly distended. The abdomen was tense and tender throughout, but the degree of tenderness and guarding was distinctly increased on the right side of the abdomen where the strangulated loop existed. The degree of distention and the tension in the abdominal wall made it impossible to discriminate the strangulated loop on palpation. Bowel sounds in this individual were completely absent.

Courvoisier's law dictated that patients presenting with obstructive jaundice due to tumors distal to the cystic dust and hepatic duct confluence will have a nontender, globular, distended gallbladder. In this instance, however, the patient is not jaundiced. A globular mass was palpable in the right side of the abdomen, suggestive of a distended gallbladder. This resulted from a mucocele of the gallbladder, caused by obstruction of the cystic duct by a very small stone.

Strangulation may be quite easy to detect when there is a very long segment of intestine involved. The evolving necrosis of such a long segment of intestine results in acute volume depletion and secondary toxemia with hypotension and tachycardia. There is so much ischemic and necrotic intestine in this volvulus that the patient's abdomen was extremely tense, with diffuse guarding and marked tenderness in the lower quadrants. There are no adhesions in the abdomen and the catastrophe is due to a volvulus of the small bowel mesentery.

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PERFORATED VISCUS

To follow is a more detailed discussion of perforation of the gastrointestinal tract. Conditions presented are those encountered in adults, between the esophagogastric junction and the extraperitoneal rectum. A perforation may be contained to a variable extent by omentum, other loops of adjacent intestine and mesenteries, and its spread throughout the abdomen may be prevented by these physiologic barriers. Other perforations may be of a volume or rapidity which quickly overwhelm the peritoneal defenses and allow the rapid spread of intestinal content throughout the abdomen. Some portions of the gastrointestinal tract have a part of the intestinal wall in the retroperitoneum. These would include the ascending and descending colon and the duodenal loop. Perforations through this part of the intestinal wall from diverticula, for example, may lead to spreading infection within the retroperitoneum.

PERFORATIONS IN THE ESOPHAGUS, STOMACH AND DUODENUM

Perforation of the esophago gastric junction

   Perforations of the esophagus usually occur into the mediastinum and pleural space. Occasionally the perforation or rupture is at the esophagogastric junction, and both pleural and peritoneal cavities are involved. Rarely, the contamination is entirely below the diaphragm.
   Attempts to dilate strictures. pass feeding tubes with stylets, or even endoscopy may result in perforation; violent emesis is implicated in some.

This individual presented to the emergency room after a drinking bout with an upper GI bleed. In the emergency room setting he proceeded to wretch violently and uncontrollably for a period of time. He then became septic and tachycardic. Chest x-rays revealed air in the mediastinum. His CT scan shows extensive air dissecting around the aorta and esophagus. There are bilateral pleural effusions and evolving pulmonary changes, as well.

In the same patient a Gastrografin swallow was carried out, which promptly revealed leakage from the distal esophagus just above the diaphragm. This problem would be approached by thoracotomy. If the rupture, however, were very close to the esophagogastric junction, an abdominal approach would also be satisfactory and would provide opportunity to insert feeding tubes and gastrostomy for decompression.

Peptic Ulcers

Peptic ulcers are the most common cause of perforations in the stomach and duodenum and perforations of duodenal ulcers are more common than gastric ulcers. Perforating duodenal ulcers are usually on the anterior wall of the first part of the duodenum. Ulcers posteriorly penetrate into the retroperitoneal tissues, in particular the pancreas. They incite an adjacent inflammatory reaction, which usually prevents diffuse peritoneal spread of infection.

Ulcers on the first part of the duodenum anteriorly which perforate are often small. Containment initially is frequently poor, however, and acid gastric contents and duodenal contents frequently spread widely throughout the peritoneal cavity. This chemical irritation of the peritoneum triggers the classic board like rigidity often described in perforated peptic ulcers. Rebound is hard to elicit, in spite of the wide spread peritoneal irritation, because of the extreme rigidity of the abdominal wall. Percussion will demonstrate the peritoneal contamination, however. After some period of time, there may be some softening of the abdominal wall, but diffuse tenderness and considerable guarding usually persists.
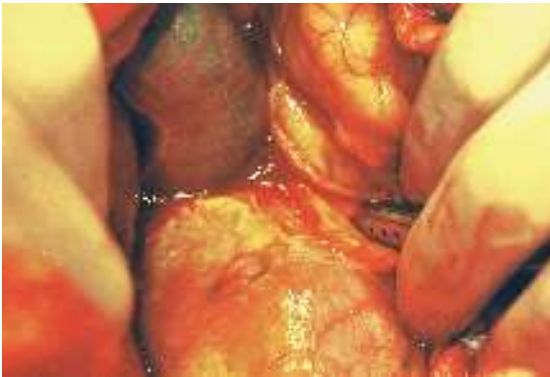
Clinical evidence would suggest that the last barrier to free perforation in an acute duodenal ulcer breaks down quickly and the spillage develops in a matter of minutes or, at the most, hours. Individuals with peptic ulcers usually have high gastric acid secretion, with sustained low pH, unfavourable for bacterial colonization. What few organisms can be cultured from the stomach are usually of low pathogenicity. The initial peritonitis is therefore largely chemical in nature, If the ulceration is contained or sealed by adjacent omentum, the peritoneum may be able to defend and clean up the acid and the pancreatic juice spillage. Sealed over ulcers demonstrated to be so by contrast studies may possibly be managed conservatively with NG suction and antibiotics. Small anterior duodenal ulcers that perforate, once they are patched, seldom go on to any further compromise of the duodenal lumen. Hemorrhage associated with a perforation is extremely rare, contrary to some beliefs. Controversy still exists as to whether or not definitive acid reducing procedures should be carried out or not, but at the present time, antimicrobial treatment for helicobacter pylori infection of the stomach and H2 blockers or proton pump inhibitors seem to be very satisfactory medical treatment if the patient is compliant. Vagotomies are frequently associated with significant late problems.

Peptic ulcers in the stomach tend to be considerably larger. They may perforate into the lesser sac. This may to some extent contain the degree of contamination of the peritoneum. Vomiting is not common. Evidence of GI bleeding is usually absent. A combination of hematemesis and perforation is very unusual. As a rule penetrating ulcers posteriorly bleed and ulcers on the anterior wall of the duodenum perforate.

Perforation of a large proximal gastric ulcer may pose a much more difficult problem. This large benign ulcer perforated through the posterior wall of the proximal stomach into the lesser sac. On opening the abdomen, peritoneal contamination was minimal and the ulcer was not palpable or evident until the gastrocolic omentum was taken down and the stomach turned outwards, as in the photograph. The margin of this ulceration is quite hemorrhagic, for some reason.

Surgical management of this large lesion is difficult. Local excision may significantly distort or constrict the stomach. Proximal gastrectomy leaving a very small gastric remnant may also be undesirable in the elderly, frail patient

The patient characteristically experiences sudden onset of pain, which is often located in the right upper quadrant of the abdomen. This usually, however, rapidly spreads. Physical examination is characterized by an individual who usually has stable vital signs, but is in extreme discomfort, particularly if he is moved in any way. Breathing is shallow and the speech is monotone. The abdomen does not move with respiration. On palpation it is rigid and on auscultation the abdomen is usually silent. Occasionally where there is significant pneumoperitoneum liver dullness may be absent.

Readily detectable free air on an upright film of the abdomen showing the diaphragms is present in 75% of instances. In other cases the free air may be shown by more discriminating imaging, such as a CT of the abdomen. Small bubbles of air may be clustered around the duodenum, under the undersurface of the liver, or around the falciform ligament. These characteristically usually would not be shown on an upright view of the abdomen.

Free air in the peritoneal cavity usually detected on an upright chest x-ray, under the right or both hemidiaphragms, and is the "looked for" radiologic sign in patients in whom peptic ulcer perforation is suspected. About 75% of peptic ulcer perforations in the stomach and duodenum will release enough air that it can be detected by a simple, upright chest x-ray. If a pneumoperitoneum is seen, this is usually taken as sufficient indication for exploratory laparotomy. Gastric and colonic perforations often release the greatest amount of air. The duodenum will commonly release air and produce a pneumoperitoneum. Small bowel perforations and appendiceal perforations seldom allow the escape of enough air to be detected on plain films.

Following the detection of a pneumoperitoneum by plain films, further investigations, such as contrast studies under fluoroscopy, are not usually found to be useful. A contrast study through an NG tube, of the stomach and duodenum, may be performed if conservative management of a duodenal perforation is being contemplated. Demonstration that there is no further leakage of gastric content would encourage continued conservative efforts.

Other contrast studies, particularly those administered by rectum, have the risk of further opening the perforation and disseminating the contamination. CT scans with contrast may frequently have the benefit of indicating the area of pathology. This avoids the surgical problem of making a long midline incision and looking for the site of the perforation if it is not found in the anticipated area.

A perforated duodenal diverticulum is a rare event, but when it occurs it has a very high morbidity rate. Air in the retroperitoneal tissues around the duodenum may not be picked up on plain films of the abdomen. Infection of the retroperitoneal tissues is poorly contained and a necrotizing mixed bacterial infection quickly spreads through the loose areolar tissues. Patients with upper abdominal pain and unexplained sepsis, fortunately, frequently go to CT scanning, where retroperitoneal gas usually can be readily appreciated, as in this scan. The surgical approach usually involves decompression of the stomach and feeding tube insertion in the small intestine, plus wide drainage of the retroduodenal area. Occasionally repair of the base of the diverticulum can be fashioned, but in delayed cases this may be technically very difficult, in view of the associated necrosis and inflammatory reaction.

The management, once the diagnosis is suspected, will usually involve performance of an exploratory laparotomy. If perforation in the anterior wall of the duodenum is found, the best strategy is to seal the perforation with an omental patch by the technique first described by Roscoe Graham. Attempts to sew the ulcer shut or to imbricate it usually fail because of the cartilaginous and rigid nature of the tissue immediately adjacent to the perforation. The omental patch should be secured carefully over the perforation, with sutures around the periphery of the patch and to the muscular coats of the duodenum and truly should appear as a "patch". In ulcers that are very large in which a patch is not a feasible solution, difficulties may be encountered. Gastrectomy is followed by considerable morbidity and the risk of leakage of the duodenal stump is considerable. Attempts to attain controlled drainage of these large ulcer perforations may not be entirely successful.

If the perforation has occurred in the distal stomach and is due to a large gastric ulcer, a distal gastrectomy is probably the best solution. More proximal large gastric ulcers which perforate pose difficult surgical problems, since they would require a very significant gastrectomy to resect them and often they occur in debilitated and elderly patients. Some of these can be excised and the stomach repaired. There is also a risk that these large proximal gastric ulcers may be malignant.

Duodenal Diverticulum

 Duodenal diverticulae seldom perforate. They are seen incidentally in many contrast studies and endoscopies of the upper GI tract, but elective intervention is rarely taken. Rarely, they may perforate into the retroperitoneum and the condition is quite lethal, with spreading retroperitoneal sepsis. Delay in diagnosis can often be identified as adding to morbidity or contributing to a fatality.

Perforations in the Small Bowel

Perforation in the small bowel may be the result of ischemia, foreign bodies, Meckel's diverticulum, primary or metastatic neoplasms, inflammatory bowel disease, or certain specific infections. Ischemia or infarction of a segment of the small bowel may be the most common reason for perforation. A short segment of ischemia may be missed for some period of time and may present as a partial small bowel obstruction. Eventually the gangrenous segment begins to leak and peritonitis evolves. Meckel's diverticulum is a rare condition in adults, but occasionally presents with bleeding or Meckel's diverticulitis. Usually the presentation simulates a perforated appendix. Foreign bodies may occasionally perforate the small bowel if they become trapped and cannot pass.


Perforation from a Meckel's diverticulum secondary to Meckel's diverticulitis may in many ways simulate the same progression as appendicitis, which is much more common, especially in adults. A perforated Meckel's is usually poorly contained and widespread contamination of the lower abdomen evolves quickly. Management will usually require a short small intestinal resection, although in some instances the base of the diverticulum can be closed without compromising the adjacent ileum.
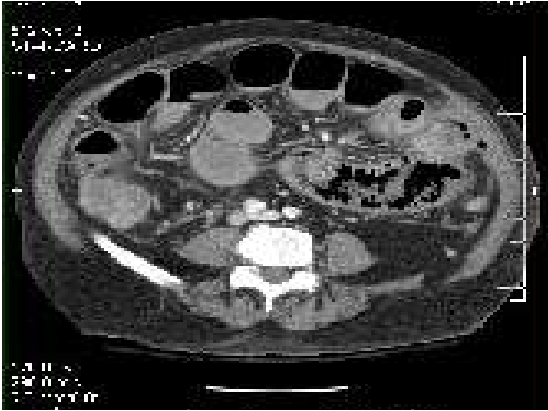
Ischemic necrosis is one of the commonest causes of perforations in the small bowel. Those coming to laparotomy and with any degree of success in management are usually segmental infarctions, which allow preservation of significant lengths of the small intestine. In this patient two segmental infarctions of intestine occurred in the small bowel following a prolonged cardiac procedure on bypass. A high degree of suspicion for this diagnosis needs to be sustained in cardiac patients with unexplained abdominal pain and partial bowel obstruction.

A CT scan shows pneumatosis in the involved segments, small amounts of free air and fluid around it, and a thick walled loop of bowel on the left side of the abdomen. Laparotomy revealed that this segment and a section of terminal ileum were completely necrotic and perforated.
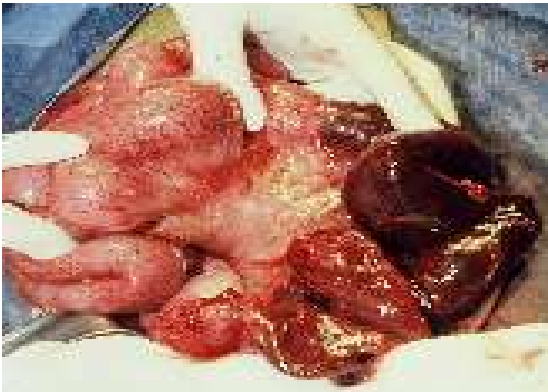
In addition to the associated problems of cardiac arrhythmias and vascular disease, segmental infarctions of the small bowel are also seen in a number of miscellaneous circumstances, including IV drug abuse, severe hypothermia, and compression syndromes. The explanation is not always clear. The diagnosis is sometimes difficult to make, although CT scanning is again extremely useful.

Gangrenous segments will be resected. If there are other questionable areas of uncertain viability, an IV bag closure of the abdomen and a second look may be employed usefully.

Neoplasms in the small bowel, either metastatic tumors, such as melanoma or undifferentiated lung cancer, or in some instances primary tumors, such as lymphomas, may perforate directly through the lesion. This, in particular may occur in lymphomas when they are being treated with chemotherapy and undergo rapid lysis. Resection of these perforations may have a reasonable outcome, particularly if there are significant alternative treatments for the provoking tumor.

Neoplasms, particularly secondary neoplasms such as undifferentiated lung cancer, may grow so rapidly that the central portion of the tumor necroses and perforation occurs through the tumor.

Inflammatory bowel disease (Crohn's disease) rarely causes free perforation. A tendency of Crohn's disease is to adhere to adjacent hollow viscera and to the abdominal wall or to penetrate the wall of the adjacent gut by its deep fissuring ulcerations. Fistulae result or occasionally localized abscesses.

Perforation of the Appendix

Perforation of the appendix usually evolves fairly slowly and it is common in adults for the perforation to be contained in the form of a pelvic or right iliac fossa abscess. Usually the characteristic history of appendicitis is not present, leading to a delay in diagnosis. The patient may not present in a timely manner. The history usually dates for several days, or even in some cases weeks. It is infrequent that free air is seen in the abdomen in cases of appendicitis, as in most cases the proximal appendix is obstructed either by lymphatic tissue or fecalith.

Appendicitis is still a serious condition if the diagnosis is delayed Morbidity and even death still occur in vulnerable individuals. The failure to diagnose appendicitis was calamitous in this patient; wide spread contamination of the peritoneal cavity occurred, with formation of large abscesses in the pelvis and right iliac fossa and also between loops of small intestine in other areas of the abdomen. The morbidity of this situation is significant, with partial small bowel obstruction, recurrent abscesses, malnutrition, and other further secondary complications.
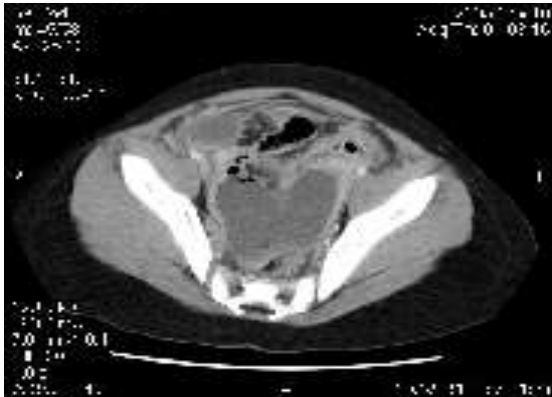
This young patient, aged 8, had an atypical history of appendicitis and went on to perforate and develop intra-abdominal abscesses while under out-patient medical observation. She presented toxic and ill, with a tense, distended abdomen. CT scan reveals multiple intra-abdominal abscesses. The image to the left shows a very large pelvic abscess and a smaller abscess in the right iliac fossa. The appendix is not seen on any of the other images with certainty.
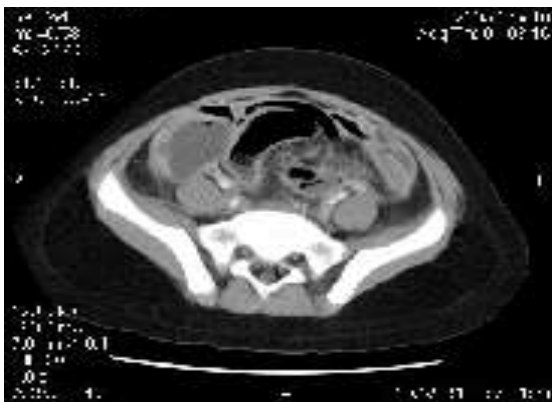
Images at a higher level of the abdomen show larger right iliac fossa abscesses and other interloop intra-abdominal abscesses, as well.

The patient required laparotomy. The appendix was successfully removed and the multiple abscesses drained, but the large pelvic abscess reformed and required readmission to the hospital. The morbidity extended for a month, although the patient eventually, fortunately, recovered.

Perforation in the Colon

Perforation of the colon may result from diverticular disease,. neoplasms of the colon, either directly through the tumor, or by rupture due to distal obstruction. Occasionally foreign bodies, ischemia, or inflammatory bowel disease may lead to perforation. Many diverticular perforations are contained in the left lower quadrant. They may present with left lower quadrant pain, possibly an inflammatory mass, and localized peritonitis. Many of these patients can be managed with intravenous antibiotics. If an abscess forms which is localized this may sometimes be drained by radiologic intervention. In this manner the patient may be guided to a stage where it may be possible to do a single stage resection of the disease.

Large or massive pneumoperitoneums are most likely to result when the perforation is either in the stomach or in the colon, where there is frequently a large amount of gas just prior to the perforation. This will be particularly true in cases of left-sided colon obstruction with massive distention of the proximal colon in the presence of a competent ileal cecal valve. This CT scan shows a huge amount of air under the anterior abdominal wall and fluid and contained debris can be seen in the peritoneal cavity lateral to the liver.

This young patient somehow managed to swallow a grocery clip. The plastic clip with a sharp hook margin managed to negotiate the intestinal tract, but became lodged in the sigmoid colon where it eventually caused a localized perforation.

Unfortunately, the patient was treated with a saline enema, which disrupted the containment of the perforation and caused a generalized peritonitis. Patients who are being managed for a confined perforation of the sigmoid colon due to diverticulitis or other causes should not be subjected to therapeutic or diagnostic tests during the acute phase, which may disrupt the containment. Endoscopy is contraindicated as a diagnostic tool in these circumstances.

Where the perforation of the colon is large and the escape of contents overwhelms the local defenses, a wide spread peritonitis may occur. Individuals who are taking corticosteroids or immunosuppressive drugs may be more vulnerable to widespread peritonitis in cases of diverticular perforation.

Neoplasms in the left colon will occasionally perforate through the tumor, but this is rare. These lesions usually have a constricting pattern of growth leading to obstruction. If the patient has a competent ileocecal valve, the proximal bowel may become markedly distended once complete obstruction evolves. This distension may lead to patchy ischemia and perforation. Because of the massive fecal and gas content in the obstructed colon, cecal rupture may occasionally produce an overwhelming fecal peritonitis and septic shock.

Ischemic colon may occasionally perforate, although the process is usually slowly evolving. The perforation may develop so slowly that the colon is completely necrotic before the condition is discovered.

Toxic megacolon, evolving in the course of inflammatory bowel disease or Clostridium Difficile colitis, may lead to perforation, or at the least, a very impressive degree of tenderness and distention. Fever, tachycardia, hypotension and secondary organ failure are the rule.

Because of the very high bacterial count and the pathogenicity of the organisms in the colon, perforations in the colon carry a significantly higher morbidity and mortality rate than those in the small bowel, duodenum, or stomach. Perforations occurring in the left side of the colon, usually in the rectosigmoid area, will usually have to be managed by resection with end colostomy. If the perforation is found in the right side of the colon, which is unusual, it may be acceptable to do an ileocolic primary anastomosis after resecting the perforated area. The cause of this perforation in the sigmoid is not entirely clear. The everted mucosal edges can be clearly seen. It does not look like a diverticular perforation and possibly occurred secondary to ischemia or stercoral ulceration.

In this patient, who had leaking abdominal aortic aneurysm resected, the inferior mesenteric artery was ligated. A picture of diarrhea, sepsis, and left lower quadrant pain evolved and ischemic colon was suspected. In spite of some attempts to confirm the diagnosis, no interventions were taken until finally the patient was in extremits. Laparotomy revealed that the sigmoid colon was completely necrotic and could be scooped from the abdomen as a foul smelling mass. In such circumstances, clearly, a reanastomosis of the colon is not appropriate and closure of the rectal stump and end colostomy (Hartmann) is performed. Miraculously, the aortic graft did not become infected and the patient recovered to survive a further decade.

Although uncommon in the colon, large, rapidly growing necrotic tumors in the proximal or transverse colon, in particular, may eventually destroy or invade the colonic wall to the point where perforation occurs directly through the tumor.

Ischemia of the colon may also occur in watershed areas during periods of low flow. This trauma patient had a significant period of hypotension due to hemorrhagic shock from a fractured pelvis. In later convalescence he developed sepsis and abdominal pain. Exploration revealed that the splenic flexure was necrotic. Although a perforation has not yet occurred, this would probably evolve within the next 24 to 48 hours. Again the bowel is completely unprepared, distended with gas and fecal material. Resection, colostomy, and a distal mucus fistula would be the appropriate procedure.

Clostridium Difficile colitis may be encountered in patients receiving antibiotics. Treatment of pulmonary infections with clindamycin and cephalosporins is often the 'set-up". Patients may or may not have significant diarrhea. They develop abdominal pain and a marked degree of distension. X-rays suggest a toxic megacolon. A perforation is not usually present, although the patient may have physical signs suggesting peritonitis. In cases where a toxic megacolon has evolved, an emergency total colectomy may be required as a life-saving measure. Cases with this extensive involvement are unlikely to respond to medical measures.

COMPLICATIONS OF PERFORATED VISCUS.

General "Systemic" Complications

Patients with an acute abdomen due to perforated viscus will suffer from general complications with greater frequency than those individuals presenting electively with gastro intestinal diseases. Patients presenting to emergency rooms with acute complications of end stage disease are in poorer general condition and have a higher rate of collateral medical problems. The complication of the disease process in the abdomen will result in "negative" secondary events such as blood volume contraction, toxemia, and bacteremia. Peritonitis, gut distention and fecal loading of the colon will often mandate multiple, "staged" procedures which contribute to the increased morbidity.

Adult respiratory distress syndrome may complicate the course of many acute intra-abdominal conditions.   Severe pancreatitis with necrosis, toxic megacolon due to any cause, and generalized peritonitis secondary to organ perforation may frequently lead to diffuse, noncardiac pulmonary edema and provoke ICU admission and ventilatory dependence.

As a general rule, complications and mortality will be higher the longer the delay period between onset of the acute symptoms and presentation for treatment and then to initiation of specific treatment after the patient has reached the facility. Studies have indicated that at least 60% of patients suffering from a specific condition, such as appendicitis, will present late. This data suggests that an unfavorable situation resulting in morbidity and mortality is often patient related.

It is unnecessary to completely review all possible systemic complications which could occur during the management of a perforated viscus. To be comprehensive, the list of complications which potentially could occur would include almost every known disease or complication in every system.

There are clearly high rates of aspiration pneumonia and nosocomial pneumonia in this group of patients. Cardiac events, including myocardial infraction, dangerous arrythmias and congestive heart failure occur with increased frequency.   Because of problems with sequestration and loss of body fluids, prerenal failure is very common and may be severe if it is imposed upon previous

chronic disease. The well known complications of pulmonary embolus and DVT also occur with an increased frequency.

Coagulopathy may be encountered, in the presence of acidosis, hypothermia, sepsis and liver failure. Patients may have been receiving anticoagulants for collateral cardiac and vascular disease.

States of agitation and delirium frequently develop and complicate the management of these individuals; important drains, IV lines and catheters may be pulled, patients fall, aspirate or are dangerously over-sedated.

Regional Complications (abdomen)

New disease in the GI tract

Because of the usually prolonged display of broad spectrum antibiotics, the delay in return of intestinal function, extensive use of narcotics, multiple imaging and other support strategies required, this group of patients will suffer an increased rate of these type of complications.

Clostridium difficile colitis may start insidiously. If diagnosis and treatment is delayed and the condition gains significant momentum, a critical illness may evolve, with toxic megacolon and secondary organ failure. This elderly patient was being treated with cephalosporins for pneumonia. His abdomen became distended and tender. He had a small amount of diarrhea. The condition was not initially diagnosed. A marked leukocytosis evolved, with a white cell count at 50,000. The abdomen exhibited extreme generalized tenderness and rebound, suggestive of perforation. The imaging shows the enhancing mucosa of the inflamed colon, but does not demonstrate any free air.

The patient's abdomen became so tense he developed a compartment syndrome, with respiratory distress and impaired venous return. These problems were superimposed upon the toxemia from the colitis. In desperation, urgent laparotomy was performed and the colon was removed. Unfortunately, the patient never recovered from the severe inflammatory response and deteriorated and died soon after the surgery.

Clostridium difficile colitis may occur, particularly in those individuals receiving Clindamycin or third generation Cephalosporins. Prolonged ileus, lack of oral intake and narcotic analgesics may contribute to the development of acalculus cholecystitis and pancreatitis. Stress, absence of oral

intake and sepsis will increase the rate of gastrointestinal bleeding from gastritis, acute erosions and even anastomotic sites.

Complications related to the peritoneal cavity

The frequency of serious complications in the abdominal cavity, retroperitoneum or wound will be very closely related to the delay in control of the on going contamination. Delay in presentation or in operative intervention, with extended periods of peritoneal contamination, will increase the incidence of intra abdominal abscesses. Even though these will be drained and irrigated at operation, collections may reform post operatively, either in the same or other sites. Common locations for postoperative abscesses are the subphrenic spaces, subhepatic space or in the pelvis.

In cases of perforated viscus, the longer the peritoneal contamination is in place, the more likely there are to be post-operative problems with reformation of intraperitoneal abscesses. In this patient with a perforated appendix, who delayed his presentation for medical care, an appendectomy was performed quite urgently. There were collections of pus in the iliac fossa and pelvis, which were irrigated until clear.

This was to no avail, however, and post-operatively within the first week the patient redeveloped fever and scanning demonstrates a pelvic collection. This was not approachable for percutaneous drain through the anterior abdominal wall, but was successfully entered and drained via the sciatic notch. The scan shows the drain in position within the abscess.

Severe abdominal contamination which is difficult to clear by operative means and by antibiotics will usually lead to a prolonged paralytic ileus or partial obstruction of the intestinal tract.   Failure of the intestinal tract will lead to nutritional issues. Total parenteral nutrition will be required and the patient will then be at risk of complications related to central venous access.

Complications related to the wound

When the peritoneal cavity is severely contaminated and access to the problem must be gained by an abdominal incision, the wound is also contaminated and the likelihood of wound problems, particularly infection and dehiscence, increases. Wound management strategies may play an important role in the prevention of complications in this area.

Patients with severe abdominal contamination present a serious risk of morbidity related to the incision. Risk factors include obesity, diabetes, and immunosuppressive drugs. Obesity is a major player, however. It is optimistic to close these wounds completely and expect an uneventful healing of the incision to occur. These attempts are frequently followed by fascial necrosis and wound dehiscence.

This patient was on prednisone for Crohn's disease. The Crohn's disease perforated in the ileal cecal region, leading to severe peritonitis and pelvic abscess formation. A laparotomy and resection of the disease was performed. The wound was completely closed, including closure of the skin with staples. A severe necrotizing infection occurred involving the fascial layers, which soon dehisced and the patient eviscerated.

Complications related to the specific procedure performed

The rate of complication in this category is related to the nature of the primary problem causing the acute abdomen. Perforation of a duodenal ulcer, operated on and sealed by an omental patch, after a quick presentation, usually has a good outcome with little likelihood of complication. If a complex surgery, with many suture lines [eg, gastrectomy] is performed, morbidity is very likely.
On the other hand, delayed presentation of a patient
with perforation of the distal small bowel or colon will be followed by serious morbidity. The higher bacterial and mechanical load of the more distal bowel and the frailty of its circulation are probably the major factors contributing to this observation.

Perforations in the descending colon will usually be managed by resection of the perforated segment with the diseased bowel [diverticulitis, ischemia, etc.] Anastomosis will not be attempted, so the patient will have an end-colostomy, usually in the descending colon or proximal sigmoid. Only the defunctioned rectal stump is left as a potential source of leakage.

Pathology in the terminal ileum or ascending colon, leading to perforation, often is managed by right hemicolectomy, The operator will need to make a choice between an ileostomy stoma and reanastomosis to the transverse colon; frequently, the latter course is taken.
Most small bowel perforations are resected and reanastomosed.

Problems with the wound are often associated with intra-abdominal complications, as well. During the course of secondary healing of a dehisced abdominal wound, bilious drainage was noted and a surface fistula from small bowel appeared in the middle of the wound. This complication provides great difficulties in management and attempts to deal with it very often lead to further morbidity and even death.

Perforated viscus in the form of an anastomotic leak may complicate any abdominal surgery involving anastomosis, but it may also occur post-operatively due to inadvertent injuries to the intestinal tract. This patient had an appendectomy with an inadvertent injury to the terminal ileum. Fecal peritonitis resulted and was signaled also by the appearance of fecal drainage from the appendectomy wound and cellulitis of the surrounding abdominal wall. Needless to say, this is a very threatening and morbid complication.

Wherever there is a suture or staple line, there is a risk of leakage. In the postoperative abdomen, with resolving peritonitis, delay in recognizing this complication is common. Frequently this disastrous event is finally declared by intestinal content seeping from the wound or a drain. The mortality rates for this complication are reported to be 20-60%. Variation in prognosis within a population suffering anastomotic leak is likely explained by the age of the individual, as well as risk factors, such as age, obesity, diabetes, immunosuppressive drugs [corticosteroids] and pre-existing collateral medical conditions.

Complications occur secondary to various intestinal tubes, which are often well intentioned and even well indicated. In this patient a percutaneous gastrostomy was done for the purposes of feeding after complex surgery on the head and neck. Unfortunately, the percutaneous access to the stomach is not secure and a leakage occurred in the ICU while the patient was under the affect of sedation and analgesia. The peritonitis became very widespread and severe before it was recognized.

If tubes for gastric decompression, intestinal feeding or drainage of the gall bladder, etc. are inserted, then there may be complications related to the placement of these catheters. Creating a "valve" at the site of entry into the stomach or bowel [Witzel technique] is a good precaution, as is suture of the visceral to the parietal peritoneum close to the tube These strategies would only be possible in open surgery.

If stomas are created, necrosis or retraction of the stoma.may occur. Often this evolves in obese individuals who always seem to have thick, short mesenteries. It is not an easy matter to revise a necrotic colostomy

Stomas that are under tension and appear "dusky" at the conclusion of an operation are unlikely to "pink up" later, although this hopeful statement is often made.

It is better to do whatever is necessary to obtain a clearly viable stoma than to be faced with a sick, early post op patient with a foul smelling, black stoma, the proximal extent of necrosis being uncertain.

Whenever stomas are created under emergent conditions there is an increased rate of complications related to these stomas, in particular necrosis and/or retraction. In this instance, where the patient had a Hartmann resection for perforated diverticulitis, perfusion to the stoma was lost and it became necrotic, at least down to the fascial level. The wound also became infected and all skin sutures had to be removed. The fascia was partially necrotic and gradually the wound dehisced, as well.

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