

GI BLEEDING

Photography and text by D.B. Allardyce MD, FRCS.

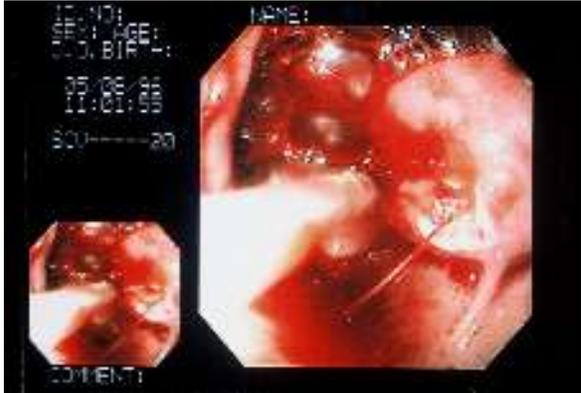
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GI Bleeding

Gastrointestinal Hemorrhage

Acute blood loss into the intestinal tract presents a management challenge and a grave threat to the patient. The lesion which is bleeding cannot be easily visualized at the bedside, nor can it be controlled in this setting. Transport of the patient to an endoscopy clinic, angiography suite or to the operating room is necessary for definitive diagnosis and management.



Large penetrating ulcers in the stomach or duodenum are unpredictable and threatening. If they bleed once vigorously they are very likely to bleed again in a similar manner. An attempt was made to coagulate the visible vessel in the base of this ulcer, the result being that a brisk hemorrhage was provoked. Blood was not cross-matched, IV access was poor, the only available OR had a case in progress [2300 hours], and the surgeon on call was unaware and 40 min. away. By the time an OR staff had been assembled and the patient reached the OR suite, 3 litres of NS and 2 units of unmatched blood had been given but the patient was still in shock, hematocrit was 20 and the ECG monitor displayed ischemic changes.

The course of intestinal bleeding is unpredictable. A peptic ulcer which has bled and then stopped, and seems to be responding to conservative treatment, may suddenly erode a large vessel and exanguinate the patient. The physical remoteness of the patient from an accessible operating room can be a determining factor. Hematemesis with profound hypovolemic shock, occurring on a medical floor at 0300, 2 corridors and an elevator ride away from the operating room, is a scenario to anticipate and avoid at all costs.

Intestinal bleeding takes many forms, and details of the history have some predictive value, but surprising changes in the course of events occur with some regularity.

If the patient has bled from the intestinal tract, whether this a hematemesis, emesis of "coffee-ground" material, melena, clots and bright blood per rectum, an aggressive approach should be taken to visualize the source, or at least localize the source. The best time to do this is when the patient is bleeding. Unfortunately ulcers, varices, diverticuli and vascular malformations have no respect for the time of day, and 50% of serious acute bleeds will occur at night, after hospital staff have gone home, GI clinics close, OR's close and attendings go home. Nurse/patient ratios increase and med students are first on call. The chemistry of the situation, if a massive GI bleed occurs, yields delay, confusion and stress for managers, and a bad outcome for the patient.

Some basic principles:

- 1) Upper GI bleeding is the most threatening. If there has been a hematemesis the patient must have gastroduodenoscopy as soon as it can be arranged.
- 2) If there is a continuing lower GI bleed, the localization of this bleeding site, by colonoscopy, RBC scans, or angiography should be tenaciously pursued.
- 3) Surgical "input" should be sought early.
- 4) Patients whose course is unpredictable should be managed in a setting where there is good monitoring and access to the OR is easy.

Upper Gastrointestinal hemorrhage

The word “upper” implies that the source of bleeding is in the esophagus, stomach or duodenum. Often, the location of the bleeding lesion in the upper GI tract is signaled by the patient’s presentation with hematemesis. Rapid filling of the stomach with blood will usually result in emesis. The occurrence of hematemesis is a function of the volume and rate of blood loss. Bleeding in the esophagus and stomach, which does not exceed the rate at which the stomach can empty, may not be associated with hematemesis. Should rapid blood loss occur in the duodenum, reflux of blood through the pylorus may not occur in sufficient quantities to result in hematemesis.



Chronic peptic ulcers penetrate deeply, often completely through the gastric or duodenal wall, until they erode into adjacent organs. This may be the pancreas, when the ulcer is in the first part of the duodenum, or in the stomach, the base of the ulcer may be the body of pancreas or the under surface of the liver. Very large vessels may eventually be eroded by this process. These include the gastroduodenal artery behind the first part of the duodenum, the splenic artery posterior to the stomach or the left gastric artery on the lesser curve. Unlike a malignant ulcer, chronic peptic ulcer base is a dense fibrous tissue, which encases the involved artery, preventing its subsequent contraction should a hemorrhage commence. The eventual erosion into the vessel wall is usually tangential or “flute hole” like. This further contributes to the inability of the vessel to contract and staunch a serious hemorrhage. This ulceration was in the antrum of the stomach and its location permitted a definitive resection by distal gastrectomy. The ulceration, although large, is very localized and all of the surrounding gastric mucosa and the duodenum is entirely normal and devoid of any other peptic ulcers. A good explanation for the localized nature of large peptic ulcers is still lacking.



This ulceration is in the duodenum. It has been exposed by an incision in the anterior wall of the duodenum. The edges of the duodenotomy are held by Babcock forceps. The duodenum was very edematous and scarred anteriorly. There was a penetrating ulcer on the posterior wall of the duodenum, which is the usual location of lesions that lead to massive hemorrhage. In the central part of this ulceration one can see a thrombus protruding from the eroded vessel, likely the gastroduodenal. The patient’s admission to hospital had been precipitated by a massive hemorrhage, passing fresh blood from the

rectum and experiencing repeated hematemesis.. On arrival in the ER, the patient was in profound hypovolemic shock. It is likely the patient was saved by thrombosis of the vessel during the period of hypotension

Although features of the history and physical exam may provide an “educated guess” as to the site of bleeding (upper or lower GI tract), unless the patient vomits blood, there will be an element of uncertainty. This concern can often be resolved by passing a nasogastric tube – if aspiration reveals bile, the bleeding site is below the duodeno-jejunal junction.

With the exception of esophageal and gastric varices, all bleeding in the upper GI tract is from the arterial system. The eroded or ruptured vessel may be small (as in the submucosa) or it may be a major visceral artery (gastroduodenal, L. gastric). The size of the vessel, and hence its blood flow, will determine the rate of blood loss.

In a general sense, bleeding in the upper GI tract is more unpredictable and dangerous than lower GI bleeding. The explanation for this observation lies in the size of vessels which can be eroded in the gastroduodenal axis.

Diverticular disease and angiodysplasia cause a high percentage of lower GI bleeds, and the vessels involved are often microscopic in size, with blood loss rates of 1-3 ml/min. In contrast, a large peptic ulcer, eroding the gastroduodenal artery, can lose 500-1000cc's in minutes.

Peptic ulceration

Peptic ulcers in the stomach and duodenum are the commonest cause of upper GI bleeding. Bleeding is a more frequent complication of ulcer disease than perforation or pyloric stenosis.

Hemorrhage from peptic ulcer continues as a frequently encountered problem in spite of the widespread display of proton pump inhibitors, H₂ receptor antagonists, and treatment of helicobacter infection.

Many ulcerations are acute in onset and their course may be self-limited, and without specific symptoms. These superficial ulcers, penetrating only to the submucosa, may erode small vessels and cause upper GI bleeding, passage of melena stools and/or coffee ground emesis.. Anemia may evolve. Abdominal pain may not be an issue. Examination of the abdomen is usually unrevealing.

Unless the bleeding is very transient, the problem usually comes to medical attention and gastroduodenoscopy is performed. Ulcers may be in the stomach or duodenum, and may be multiple. The first part of duodenum is the commonest site. Endoscopy will reveal the superficial nature of these ulcers. If active bleeding is seen, it is from small vessels which can be localized with flushing. Control is frequently possible by endoscopic techniques (injection of adrenaline, heater probes, laser.) These ulcers then heal rapidly on medical treatment. History often identifies the expected “abuses” leading to acute peptic ulcer formation. These include medication, alcohol, environmental “stress,” and poor, inconsistent diet. The role of caffeine and nicotine is unclear. Provoking medications are salicylates, non-steroidal anti-inflammatory agents and corticosteroids. Gastric and duodenal “erosions” and bleeding often complicate the course of hospitalized patients, particularly those with critical illnesses (burns, head injuries, sepsis and multiple organ failure).

These lesions appear distinct from the chronic, large, deeply penetrating, and solitary ulcer sometimes identified as the cause of upper GI bleed in patients presenting to an emergency room. These latter individuals may give a history of upper abdominal pain with “ulcer type” features (worse during fasting, relieved by food/antacids). The history may span many years, with periods of unexplained respite and “flare ups” requiring medication.



A deeply penetrating ulcer in the posterior wall of the duodenum has been exposed by a longitudinal pyloromyotomy. The bleeding vessel has already been secured. Now, the suction can be brought into play and the site of the bleeding vessel should be "agitated" with the suction in an attempt to provoke a recurrent hemorrhage. In this way, the security of the suturing of the vessel above and below the vascular erosion can be put to the test. If the hemorrhage is going to be restarted it is better that it happens at this point, in the operating room, rather than in the recovery room.

The gastroduodenal artery runs from the 6 O'clock to the 12 O'clock position across the ulceration. The vessel is imbedded in the scar tissue in the base of the ulcer. The usual strategy is to "under run", and ligate the vessel at the superior and inferior margin of the ulcer, isolating the eroded portion between the two ligatures. Ideally a strong synthetic collagen suture at least "0" caliber and mounted on a stout tapered needle with a short curve should be used. The temptation to use a suction on protruding thrombus from the vessel before the sutures are in place should be resisted, as a spurting hemorrhage will result which will make placement of the sutures more difficult to perform.

Bleeding when it occurs, may be torrential, causing repeated hematemesis and bright blood from the rectum. Often there is rapid deterioration to profound hypovolemic shock. The patient may be obtunded or even unconscious. Only weak femoral and carotid pulses may be felt. Paramedics attending at the site where bleeding began will describe large amounts of fresh blood in the toilet, on the floor, soaking bedding, etc. Although cardiac arrest or cerebral thrombosis may occur, these individuals often reach an ER, although with severe hypotension, and do respond to rapid fluid infusion.

Frequently, bleeding seems to have stopped and there is a period of “stability” when endoscopy can be undertaken and the lesion identified. The ulceration that gives rise to this pattern of bleeding needs to be deeply penetrating and display a ‘visible vessel” in its base.

If this situation does prevail, then a definitive surgical intervention should be undertaken as soon as the patient has been fully resuscitated and assessed.

A second shocking hemorrhage is unlikely to be tolerated, and a fatal outcome would be predicted if this is allowed to occur.



Endoscopy provides invaluable information in a case of upper GI bleeding. Although a small vessel in the margin of this ulcer could open and bleed, it is very unlikely to bleed rapidly enough to outpace boluses of IV saline or periodic transfusion. Medical treatment with IV proton pump inhibitors or repeat endoscopy with adrenalin injection or coagulation should be successful in stopping a bleed from this ulcer.



When clot covers the base of an ulcer, there may be hesitation to flush it away, for fear of reactivating a bleed which might be difficult to stop with endoscopic techniques. Washing the clot off the ulcer will not usually start bleeding; the maneuver may reveal the orifice of an eroded vessel; if it is a large artery, this finding may provide the necessary impetus to proceed to operative control. This vessel will usually be filled with a small thrombus; *the endoscopist should not disturb this thrombus*. Clot adherent to the posterior wall of the first part of the duodenum or lesser curve, posterior wall of stomach is more likely to cover a large vessel.

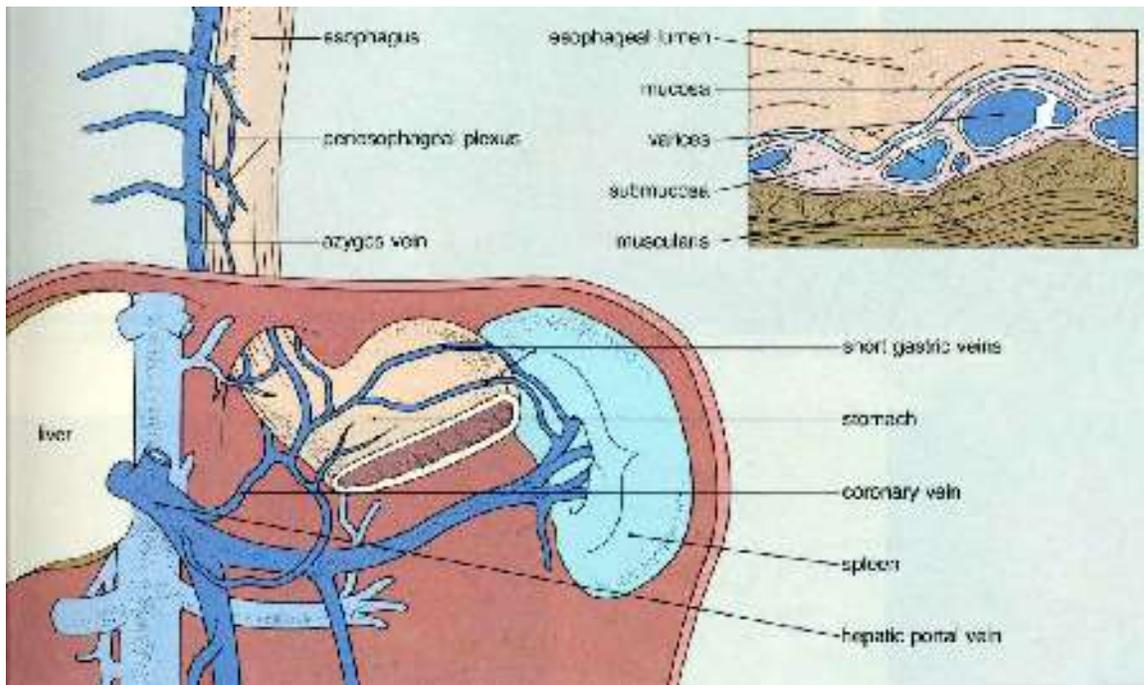


Bleeding peptic ulcers are often seen, at endoscopy, to be covered by adherent clot. It is difficult to assess the depth of penetration of the ulcer or the size of the vessel which has been eroded. Although it is tempting to leave the clot in place and hope for the best, it is necessary to wash the clot away and visualize the base of the ulcer. Small vessels in the base of superficial ulcers may be managed endoscopically by coagulation; a deep ulcer with a large vessel eroded should send a message to the surgeon. If a thrombus is seen protruding from an artery [as opposed to covering the entire ulcer] it should not be sucked away, in the endoscopy clinic, as this will result in brisk hemorrhage which will be impossible to stop in that setting.

Esophageal Varices

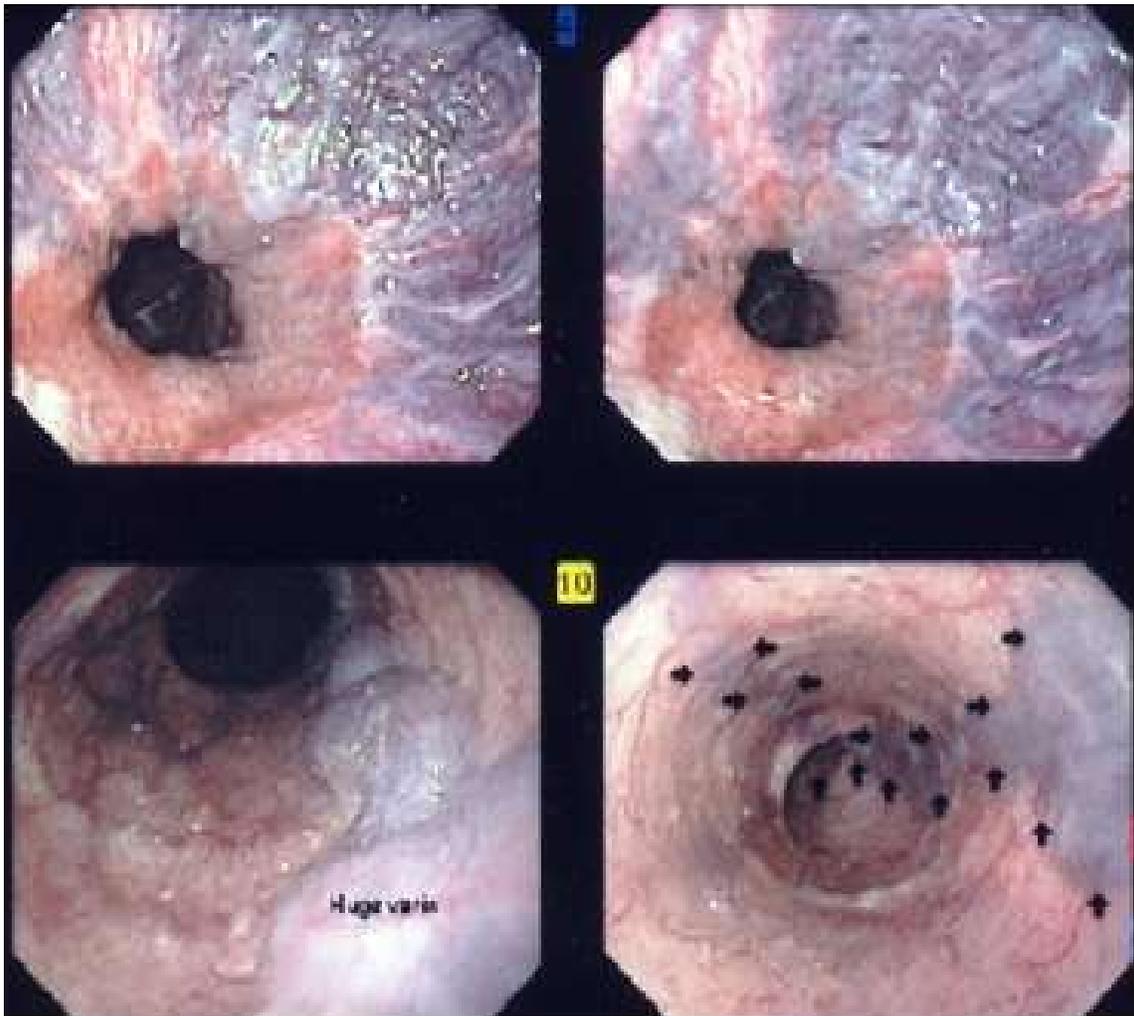
Esophageal and gastric varices develop when portal or splenic blood flow to or through the liver is obstructed.

In most instances, in North America, portal hypertension, and consequently esophageal varices, are the result of cirrhosis secondary to excessive alcohol intake. The regenerative nodules and fibrosis of the liver parenchyma obstruct sinusoidal blood flow, resulting in ascites and increased pressure throughout the portal venous system.



Venous tributaries from the lesser and greater curve of the stomach normally flow towards the

portal and splenic veins. When portal flow to or through the liver is obstructed, venous blood, seeking a pathway to the heart, flows in reverse through proximal gastric and esophageal channels to the azygous system. Vessels shown in the diagram, coursing on the surface of the stomach and esophagus, help to decompress the portal system and are harmless. Not seen in the drawing are the submucosal channels which rupture and bleed.



Extensive , superficial esophageal varices, including one “huge varix” Pictures courtesy of Hugh Chaun.

In patients with nutritional (alcoholic) cirrhosis, esophageal varices may be present throughout the latter part of these individuals lives, and never bleed. Once a hemorrhage does occur, however, and the patient survives, a second major bleed is likely to follow.

The blood flow through the gastric and esophageal varices can represent a good proportion of portal venous flow. It is not at normal venous pressure, and if a varix ruptures, bleeding may be torrential, with repeated massive hematemesis, and profound hypovolemic shock. Often, there is no recourse but to tamponade the vessels with a Minnesota tube. If properly positioned and inflated, variceal bleeding can be staunches by this device in 95% of instances.

The mortality rate of a massive, first time esophageal varix hemorrhage is about 20-30%. The patients have degrees of liver failure and the protein “load”, as blood, in the gastrointestinal tract inevitably provokes encephalopathy. In the comatose patient with the cumbersome esophagogastric tube in place, there is grave risk of aspiration. Liver function will also deteriorate from its usually poor baseline, with failure of bilirubin transfer, severe hypoalbuminemia and coagulopathy.

Since the majority of adult patients in North America with portal hypertension will have significant liver disease and liver failure, an emergent surgical intervention, to try, by direct suturing, devascularization or shunting, to control a variceal bleed has not enjoyed much success. The approach to variceal bleeding is medical and procedural. Strategies include infusion of pitressin or Octreotide to lower portal blood flow, tamponade by Minnesota tube, and subsequently endoscopic injection of sclerosants or banding of the varices.

Diagnosis is obviously pivotal, because surgical intervention is indicated for massive bleeding from peptic ulcers, Mallory Weiss tears, aorto duodenal fistula, and vascular tumors (leiomyoma). A Minnesota tube will not stop bleeding from a proximal gastric ulcer or Mallory-Weiss lesion, because the source of these bleeds is arterial.

Endoscopy may be possible, if there is a "window" where the patient is resuscitated and bleeding has slowed or stopped. Such opportunities do not always present, and the moment should not be allowed to "drift by."



Only 50% of patients with varices actually bleed from them. It is not known what factors actually lead to a variceal rupture. These large, submucosal channels look very threatening. Varices are sometimes bluish in color, but may be pale, as in this instance. Small red dots, larger red spots or red streaks on the varix correlate with an increased risk of hemorrhage.

Tortuous varices are more likely to bleed than "straight" varices.



Large varices bulge into the lumen of the esophagus and may seem to occlude the passage. Varices which extend proximally have a higher risk of bleeding than those confined to the lower one third.

Varices bleed so torrentially that it may be quite impossible to safely endoscope such a patient, much less visualize the bleeding site or do anything to control it.

In these instances it may be necessary to insert, position and inflate a Minnesota tube, the decision being based on strong clinical suspicion.

Even though peptic ulcers can occur in patients in liver failure, there is still a strong likelihood that a patient who is jaundiced, has stigmata of liver failure, ascites and splenomegaly, and then presents with a massive upper GI bleed, is bleeding from esophageal varices.



Active bleed from an esophageal varix demonstrates the “artery- like” nature of these hemorrhages. The pressure in a varix may be 20-30 mm Hg and blood is still well oxygenated, having been shunted away from the liver sinusoids.

Picture taken by Hugh Chaun.



The late phases of this selective mesenteric angiogram show opacification of the portal vein but also demonstrate a torturous, dilated, left coronary vein extending into gastric and esophageal varices

Portal venous pressure will always exceed 10 mmHg once varices have developed. The contribution of the portal vein to liver blood flow can be estimated by the extent to which portal venous arborization is seen in the liver. In this instance, some portal radicals are seen [grade 2 perfusion]



This image of the chest and upper abdomen shows a Minnesota tube in position. There is some contrast, possibly gastrografin, instilled in the esophagus and stomach. The reason for the contrast cannot be recalled by the writer. In any case, the gastric balloon of the Minnesota tube is shown to be nicely located within the stomach and properly inflated. What is required here is to put an appropriate amount of traction on the tube by some device so as to pull the inflated gastric balloon back against the fundus of the stomach. This has not been accomplished at the time this film was taken. The gastric balloon is large enough to prevent its dislocation into the esophagus. Once the gastric balloon is inflated, the esophageal balloon may be inflated as well, according to guidelines.



The late phases of this mesenteric angiogram show a normal portal vein. There is no distended or variceal coronary vein. Liver blood flow is about 1500 ml/min, and 60% is from the portal vein which also supplies about $\frac{1}{2}$ the oxygen requirements of the liver. Complete diversion of portal flow from a cirrhotic liver [as in portocaval shunt] is not tolerated, with further deterioration in liver function and encephalopathy.

If a Minnesota tube is properly positioned and inflated, and is under proper traction, pulling the gastric balloon against the fundus, and bleeding is continuing as evidenced by blood being aspirated from the gastric suction part, then the diagnosis of variceal hemorrhage needs to be immediately revisited.

A proportion of adult patients with portal hypertension may not have liver disease. The most important and potentially salvageable individuals have pre-hepatic occlusion of the portal or splenic vein. If this occlusion is due to non-neoplastic process (thrombosis) the indication for a timely early surgical intervention is strengthened.

“Pre hepatic” portal hypertension will result in gastric and esophageal varices, but the patient will not have ascites, or any of the stigmata of liver failure. These individuals are rarely encountered, but should be aggressively investigated and managed by an appropriate surgical procedure. Some of these patients have metastatic or primary neoplasms, which have caused the PV or SV occlusion. These tumors may be unresectable and preclude any operative intervention. SV occlusion may result in “localized” portal hypertension involving gastroepiploic veins, short gastric veins and the spleen. A splenectomy interrupts the pathway of blood to these vessels and restores normal venous pressures to the vessels of the esophagus and gastric fundus.

Selected patients with alcoholic cirrhosis and portal hypertension who have experienced a variceal bleed, survived, and subsequently have been able to maintain reasonable liver function (Child's A), may be candidates for an elective shunt. Cessation of alcohol intake would also be a prerequisite.

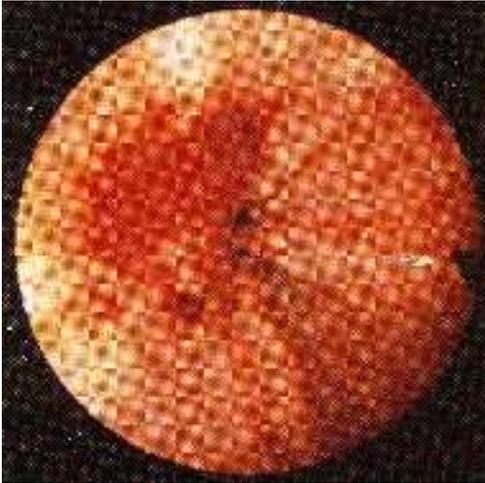
Studies have repeatedly demonstrated that the various porto-systemic shunts have reduced the frequency of bleeding, but have not prolonged life. Porto-caval shunts deprive the cirrhotic liver of portal blood flow (often completely) leading to further deterioration of liver function. The most disturbing aspect of the failure of liver function is progressive encephalopathy and all the degrading, disturbing behavior changes of a chronic brain syndrome.

Probably the best porto-systemic shunt for an individual with cirrhosis, which strikes a balance between prevention of variceal bleeding and maintenance of critical portal flow to the liver, is the distal spleno-renal shunt described by Warren.

Mallory-Weiss Syndrome

This lesion may be more common than indicated in many surveys of upper GI bleeding encountered in a major hospital. The bleeding site is a laceration close to the esophagogastric junction, presumably caused by violent wrenching or vomiting. Because of its pathogenesis, there is a common association with alcoholism. The laceration extends into the submucosa and bleeding is from small arteries torn by the rapid distention of the esophagogastric junction. In spite of the small size of the vessels, bleeding can be quite formidable, and often mistaken for variceal hemorrhage or bleeding from a proximal gastric ulcer.

The history, if it can be elicited, is one of repeated violent emesis, initially appearing as gastric contents, but suddenly becoming sanguinous, then frank blood. Bleeding may be rapid enough to cause hypovolemic shock.



A Mallory-Weiss tear which is continuing to bleed. Many of these lacerations have stopped bleeding when they are first seen at endoscopy. Injection of adrenalin submucosally, adjacent to the laceration, might be the best option here. If a Mallory-Weiss tear continues to bleed after conservative treatment, a laparotomy and proximal gastrotomy should be performed. The laceration can be oversewn, starting a continuous suture at the lower end. A useful ancillary procedure is a tube gastrostomy, for gastric decompression, avoiding an NG tube, which would cross the sutured tear.

The site of bleeding usually can be identified endoscopically, and endoscopic techniques (injection of adrenaline) may be effective in some cases. Recurring or persistent bleeding may require surgical control, usually accomplished by suture of the laceration, exposure by a proximal gastrotomy.

Duodenal Neoplasms

Duodenal Neoplasms are rarely a cause of upper GI Bleeding. A variety of lesions can be encountered including adenomas and hamartomatous polyps of Peutz-Jeghers syndrome. Bleeding may also arise from carcinomas of the ampulla of Vater and infiltrating pancreatic cancer. Leiomyomas are also found in the duodenum.



A sub mucosal tumor about two centimeters in diameter bled rapidly from a small superficial artery. When seen at endoscopy there was no active bleeding. An attempt was made to coagulate the visible vessel. It began to bleed briskly, provoking emergency surgery.

The duodenum was mobilized and a local excision was possible. The tumor was close to the ampulla; a choledochotomy was performed and the bile duct cannulated to identify and protect the ampulla.

This proved to be a carcinoid tumor.

Neoplasms of the Stomach

It is unusual for a gastric tumor to bleed actively, requiring urgent surgical intervention. Most gastric tumors are adenocarcinomas. Regardless of whether they are infiltrative or polypoid, the neoplastic vessels are of small caliber and not capable of releasing a major bleed. “Oozing” of blood from the surface of an exophytic lesion or ulcerative base of an infiltrating tumor is the rule, with gradual development of iron deficiency anemia. Occasionally, a patient may have a “coffee-ground” emesis or a melena stool.

Some gastric neoplasms may bleed vigorously. Notorious for this behavior are leiomyomas and lymphomas. A gastric lymphoma, under treatment with chemotherapy protocols, may lyse rapidly and a major vessel may be disrupted.



Malignant ulcers lead to “encasement” of vessels, but do not, as a rule, erode into an artery as acid peptic digestion will.



Gastric epithelial tumors (adenocarcinomas) may cause anemia by a slow loss of blood from neoplastic vessels on the ulcerated surface. They are very seldom responsible for an acute upper GI hemorrhage..

If a tumor is identified as the cause of an upper GI bleed, a surgical resection is usually indicated, unless the lesion is demonstrated to be inoperable or unresectable, (usually by CT scan). If the tumor is resectable, the surgery should proceed as an emergency – hoping the bleeding will stop or slow, and that the gastrectomy can be “fitted in” to an elective state, may increase the morbidity of a potentially salvageable case.

Dieulafoy Lesion

This vascular abnormality is rarely encountered. It is a vascular anomaly, with large ectatic vessels, usually arterial, close to the mucosa. The location is often in the proximal stomach. Cause is unknown. The vessel lumen may be eroded or rupture at the gastric mucosa, and blood may actually be seen to be spurting from the apparently intact gastric lining. Needless to say, they are very difficult to identify unless they are actually bleeding. If they are seen bleeding at endoscopy the site needs to be recorded as accurately as possible. Their behavior is often “perverse,” bleeding actively until the patient reaches the OR, then stopping and becoming

invisible just before the stomach is opened. Excision of the vascular lesion is better than over-sewing, as a means of control.



A classic Dieulafoy lesion, bleeding briskly, as they usually do. There is a feeding or draining vessel at 6 o'clock. They are found in the corpus or fundus of the stomach.

Histologically, a cluster of small arteries is seen in the submucosa. Careful sectioning of the excised lesion may identify where one of the vessels has opened through the mucosa. There is no ulceration or inflammatory response.

This great photo taken by Hugh Chaun.



Usually, once a Dieulafoy malformation stops bleeding, there is little hint of its precise location. In this instance, however, there is a dull red nodule which may represent a thrombus. If a gastrotomy were performed to manage the source of a recurrent upper GI bleed this would be a great discovery; endoscopic identification of the site is still helpful, as the small lesion can "hide" in the gastric rugae once the stomach is opened.

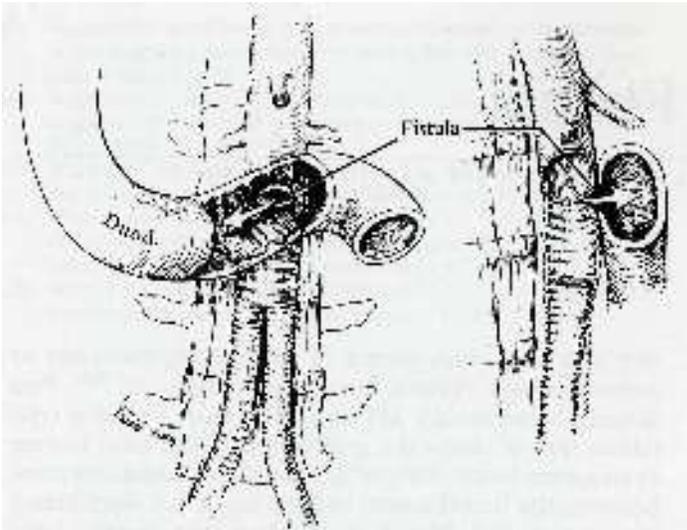
Photo by Hugh Chaun.

Aortoduodenal Fistula

This catastrophic late complication of aortic grafting still continues to present sporadically. Surprisingly, the initial bleeds may not be as massive as one would anticipate, but ultimately, a shocking hemorrhage will occur or the patient will be overwhelmed by sepsis originating in the infected graft.

If there is a history of previous grafts to the infra renal aorta, either as replacement or by-pass, and the patient experiences what appears, by history and initial vital signs, to be a sudden, brisk hemorrhage into the GI tract, this diagnosis should be immediately suspected.

The usual site of the aorto-enteric communication is the third part of the duodenum, where the duodenal wall may lie on the proximal anastomosis of the aortic graft. Other sites for the fistula can occur, but are rarely encountered.



Although an aneurysm can spontaneously erode into the GI tract, the proximal suture line of an aortic graft is the usual site of the communication. Wrapping the graft with the shell of the aneurysm, and therefore avoiding any contact of the pulsating graft with adjacent intestine, has markedly reduced the incidence of this complication.

The course of these patients also features a background of chills and febrile episodes. If bacteremia is suspected, blood cultures are usually positive.

Often the history of bleeds and pyrexia has some duration e.g. 1-2 weeks, and investigation may not be as aggressive as it should. The condition is very threatening however, and exsanguination into the gut can occur without warning.

Upper GI endoscopy will exclude a source of bleeding in the esophagus, stomach and first part of duodenum. The gastroscope should be advanced into 3rd part of duodenum where inflammation, clot and distortion of the lumen will be seen. CT scan may reveal gas bubbles and/or fluid around the aortic graft.

The only chance for survivorship lies in major surgical intervention with extra corporeal, axillofemoral bypass grafts, followed by ligation of the aorta, removal of most/all of the aortic graft and repair of the duodenum.

Hemobilia

This uncommon cause of upper GI bleeding results from a communication of a branch of the hepatic artery with the biliary system. Penetrating trauma to the liver is the usual cause, but the problem may arise after hepatobiliary surgery or rarely from disease processes in the liver (hepatic abscess, hepatoma). Transhepatic imaging, placement of stents, liver biopsy are iatrogenic causes of hemobilia [now the most common cause]

Hemorrhage fills the biliary system and exits the ampulla to the second part of duodenum. Hematemesis is uncommon, and blood is passed per rectum. The hemorrhage may be quite brisk, and the fresh appearance of blood may suggest a colonic source.

Because of the filling of the ductal system with clots the patient may experience RUQ pain suggestive of biliary colic. In the 48 hours after the bleed, an obstructive pattern of liver function tests may appear.

The diagnosis should be suspected, and the arterial-biliary connection can usually be demonstrated by selective angiography. In many instances, coil occlusion of the branch of the hepatic artery feeding the communication will erase the problem.



Percutaaneous drains were placed into branches of the biliary ducts in the left and right lobes of the liver. Brisk bleeding began several days later in the form of bleeding from around the left drain and blood and clots per rectum. When the left hepatic duct drain was removed, arterial blood spurted from the skin puncture site.

Lower GI Bleeding

By definition, the term “lower” refers to bleeding sites below the duodenojejunal flexure. Hematemesis never occurs. If an N/G tube is inserted, aspiration will yield only gastric contents. If bile is aspirated then bleeding in the duodenal loop is also excluded.

The caliber of artery which ruptures or is eroded in the gut distal to the duodenum is smaller than the vessels supplying stomach and duodenum. Lower GI bleeding is, in very general terms, less threatening than upper GI hemorrhage in that a large amount of blood (1 litre) cannot be lost in a few minutes. These bleeds are more difficult to follow, in that the small bowel and colon have a “capacity” to contain a significant amount of blood before it is passed per rectum. The slower rate of bleeding does not stimulate peristalsis, which would propel evidence of a faster hemorrhage to the rectum. A common clinical scenario in lower GI bleeding is the patient passing fresh blood and clots per rectum, e.g. 500-1000mls and then being found to have dropped his/her Hgb level 20-30gms. No change in vital signs had been detected in the prior 12 hours.

A bleeding rate of 2ml/min is slow, but it can be relentless and “adds up” as the hours pass. (2ml/min = 120ml/hr = 720ml in 6 hours = 1440ml in 12 hours).

Since the patient can protect their vital signs by fluid shifts and increases in peripheral vascular resistance, the recurring drops in Hgb can be restored easily by transfusion – e.g. 1-2 units/day. The trap is that the number of units of transfused blood “sneaks” up, sometimes reaching 15-20 units. The patient becomes less resilient, and specific transfusion related complications (febrile reactions, leucoagglutinin responses, thrombocytopenia and coagulopathy) take their toll. The individual becomes, gradually, a less and less favorable candidate for surgical intervention, should that be necessary.

It is incumbent on the physician to keep a “watchful eye” on the transfusion requirements, search tenaciously for the source, and be prepared to intervene surgically. A “threshold” number of transfused units, often quoted, is eight

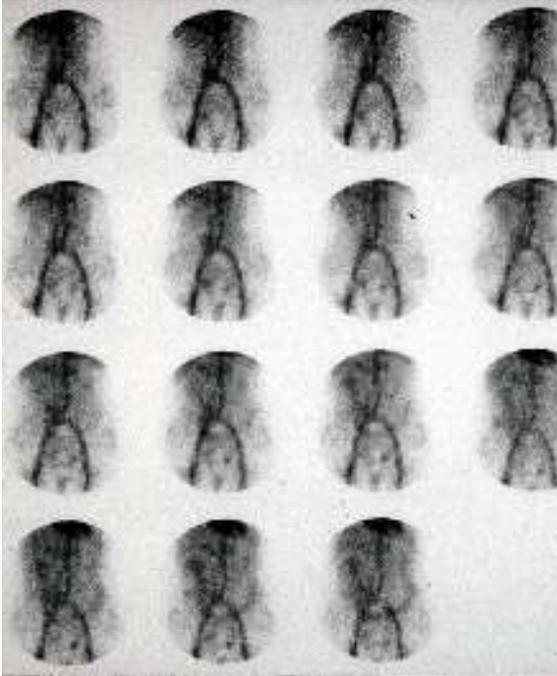
Diverticulosis

This condition can be demonstrated with great frequency in the age group 50-90 years in a western population. It is the commonest cause of lower GI bleeding.

Uncomplicated diverticulosis can result in significant bleeding. Small arteries penetrate the muscular layers of the colon, usually at the site of each acquired diverticulum. Ulceration of the mucosa at the “neck” of a diverticulum may result in erosion of this small vessel. Bleeding rates are slow, but may be relentless and eventually a very significant blood loss accumulates. It has been documented that a disproportionate number of diverticular bleeds originate in the proximal colon; bleeding cannot be assumed to be originating in the segment of the colon with the greatest number of diverticulae (usually the sigmoid).



Fecaliths impacted in the neck of diverticula; in this case of lower GI bleeding, a fecalith in the diverticulum just to right of center in the photo, eroded a small vessel which bled relentlessly. After 10 units of packed cells were transfused over 48 hrs., a resection of the sigmoid colon was performed. Localization of the bleeding site was by angiography.



Labeled red blood cell scans have been criticized in the literature for providing inaccurate or misleading information regarding the site of a GI bleed. Interpreted carefully, in the light of clinical information, however, they can be helpful. They have a great advantage, in the first instance, being relatively non-invasive in comparison to a selective angiogram. They also provide surveillance over a considerable period of time, up to two hours rather than just a few minutes, as in the case of an angiogram. In cases that are bleeding slowly or intermittently, a red blood cell scan may be the examination of choice.

In this instance, the first four panels show the labeled red cells in the aorto iliac vessels with some increased uptake over the liver and also over the background skeleton. In the third



Arterial bleeding in the colon is rarely rapid enough to pose problems in resuscitation. This selective angiogram [IMA] performed in an attempt to locate a lower GI bleed, does not demonstrate the source but does reveal the small caliber of the marginal artery and the tiny vessels penetrating the muscular layers of the colon. Barium pellets from a previous contrast study are seen impacted in diverticula.

row of four panels and in the final three images in the bottom panel, accumulation of labeled red cells is seen in the transverse colon. This can be identified as the transverse colon unmistakably because of its characteristic U- shape imposed on the abdominal wall. The density of the labeled red cells in the colon progressively increases through the bottom two rows, making the identification of the bleeding site in the colon unmistakable. It is clear also that the bleeding does not begin in the splenic flexure or left colon and pass backwards to the hepatic flexure. The identification of the site of bleeding near the hepatic flexure is extremely helpful in the event that surgical intervention is ultimately required.



The actual diverticulum which is the source of a lower GI bleed is seldom seen on colonoscopy. The usual finding is fresh blood in the colonic lumen, in a segment where there are multiple diverticulae.

In this instance, however, the orifice of a diverticulum containing a fecalith is seen to have a thrombus accumulating on its inferior rim.

Angiodysplasia

Once considered to be a rare lesion and unusual cause of lower GI bleeding, these acquired vascular anomalies are now recognized to cause a significant proportion of colonic bleeds. Again they are commonly in the proximal colon. The lesion can be visualized as a red "spot" on the mucosa. They are usually a few mm's in diameter, with an irregular border. Occasionally, there are "clusters" of these anomalies.

The ectatic vessels are in the submucosa, and in bleeding lesions, the mucosa may be eroded. If the bleeding site can be seen at colonoscopy, control by coagulation is often possible.

If significant lower GI bleeding is "in progress" it is critical to aggressively pursue the localization of this bleeding. The nature of the bleeding site (angiodysplasia, diverticula, tumor, ulceration in IBD) is of less importance than locating the bleeding site. Even distinguishing R from L colon as the source is of great importance. Elderly people tolerate total colectomy poorly, if the site is not identified and a surgical intervention becomes essential.



If a bleeding angiodysplastic lesion is surgically resected, the ectatic vessels may collapse, and be very difficult to find on histologic examination. Should the site be located, a cluster of dilated vascular channels, scattered through the submucosa and lamina propria is revealed.



An angiodysplastic lesion may be shown by selective angiography, even though it is not bleeding. The cluster of contrast in the cecum does not disperse in the bowel lumen as a bleeding site would. A prominent vein can be seen draining the collection of ectatic vessels.



This labeled red blood cell scan shows the accumulation of labeled red cells in the ascending colon and more in the distal transverse colon. The dense uptake in the lower midline is free isotope, which has not labeled red cells, has passed through the urinary tract and accumulated in the bladder. Although there was a time gap in the scanning process between the first images and the last two, during which the bleeding began again, and this was unfortunately missed, it can still be said with relative certainty that the bleeding is in the right colon and passes distally towards the splenic flexure.

If the patient has had a significant lower GI bleed, but appears to have stopped, the colon should be prepped and a colonoscopy carried out. An angiodysplastic lesion may be seen and erased, inflammatory bowel disease and ischemic colitis will be recognized.

If there is a continuing brisk bleed, selective angiography may be the best system. Slower bleeding, which tends to be "off and on" may be localized with RBC scan.



Efforts have been made to identify angiodysplasias by the perfusion of surgical specimens [by cannulation of mesenteric vessels]. Here, a suspension of microopaque was injected and reveals the aggregation of channels. These techniques are too laborious for routine use.



An extensive angiodysplastic lesion, not bleeding, found in the ascending colon at colonoscopy. Such a fragile, submucosal malformation could bleed recurrently and require a segmental colon resection.



It is considered that tension or hypertrophy in the colonic muscular layers obstructs venous drainage from the submucosa, eventually leading to arteriovenous communications. Upper left panel shows a cluster of angiodysplastic lesions; in the lower panels the largest malformation has begun to bleed actively.

Ischemic colitis

Hypoxic injury to the colon mucosa may lead to edema, congestion, hemorrhage and infarction of the mucosa/submucosa. The injury may not be transmural, so the integrity of bowel wall is preserved. Bleeding may occur from the “sloughed” ulcerated areas. Because of the small caliber of the vessels, the blood loss is not usually threatening, but may require transfusion. Ischemic ulcers may heal rapidly, so “expectant” management is the usual approach. The setting/primary condition of the patient often will suggest gut ischemia as a source of bleeding. The vascular supply of the colon is much less robust than the small bowel. Colonic perfusion deficits are likely to occur during low flow states or after sacrifice of vessels during emergency surgery. (E.g. resection of a ruptured abdominal aortic aneurysm-ligation of the inferior mesenteric artery.) The marginal artery in the colon is a variable structure and may not sustain a “watershed” area like the splenic flexure, during a period of reduced cardiac output. Ischemic colitis is often seen after myocardial infarction, after cardiac surgery, and in patients with occlusive vascular disease.



The colon is vulnerable to ischemia of a segmental nature because of its fragile blood supply and dependence upon the marginal vessel in certain watershed areas, namely the splenic flexure. Surgical interruption of a feeding vessel such as the inferior mesenteric artery during aneurysm surgery may cause ischemia of the sigmoid if the collateral circulation is not adequate. The mucosa is most significantly affected in cases where the ischemia is not transmural. Hemorrhage into the ischemic mucosa and subsequent necrosis and sloughing may lead to lower GI bleeding. This is seldom rapid and patients are usually easily resuscitated. The lesion can be readily identified by flex sigmoidoscopy. In cases of ischemic colitis, necrosis and perforation is a greater risk, in the acute phase, than bleeding.

Extensive Mucosal Injury of the Colon

A variety of insults, most poorly understood, cause inflammation and ulceration of the colon mucosa. Ulcerative colitis and Crohn's disease are most often encountered, but specific infections, autoimmune reactions and chemotherapy complications may necrose and ulcerate the mucosa. Diarrhea is usually a dominant symptom, accompanied by cramping pain. If bleeding begins, it is usually bright but mixed with liquid stool. Rarely, what is passed appears to be blood, with minimal stool.

The patient may display features of sepsis and toxemia consistent with extensive loss of the mucosal barrier.

Most of these conditions involve the rectum, so a simple proctoscopy, performed at the bedside will at least establish that the bleeding is from a diffuse mucosal injury.



In cases of ulcerative colitis a clinical picture which mixes elements of toxemia, with bloody stools may present. This patient had repeated passage of liquid stools which contained significant blood, but as well as manifesting low grade fever, tachycardia and marked leukocytosis. There is extensive destruction of the colonic mucosa with loss of its normal hairy texture. The colonic mucosa appears as a flat, smooth surface. The pseudopolyps of the remaining mucosa of the intervening areas are ulcerated.



Patients on chemotherapy regimes are sometimes vulnerable to extensive mucosal ulceration, necrosis and hemorrhage. The pathogenesis of this lesion is unclear but it seems more likely to occur in the proximal colon. Systemic sepsis and toxemia as well as GI bleeding may result.

Carcinoma of the Colon



Infiltrating carcinomas in the colon very seldom bleed acutely. The neoplastic vasculature is small and tortuous. An erosion of the tumor leads to oozing of blood from these small vessels, which mixes with the stool and may not ever be noticed by the patient. In other instances traces or smudges of blood are seen on the stool or in the toilet water intermittently. A continuous and significant GI bleed is unusual. This patient, however, had a small infiltrating carcinoma of the colon, and for some reason it did invade a vessel which caused it to bleed very acutely with the passage of some bright red blood and clots. Colonoscopy led to the identification of the lesion.

Sites of Bleeding in the Small Bowel

Bleeding from within the small bowel may be a very challenging problem. The lesion may be small, and the 9-10 feet of jejunum-ileum is not as easy to visualize endoscopically as the colon or upper GI tract. The small bowel as the site of bleeding may first be suspected by the process of

“exclusion,” if the upper GI tract and colon are normal. In difficult cases, when conventional imaging studies have failed, M2A capsule endoscopy may be successful.



This young woman had repeated lower GI Bleeds over a span of twenty years. She was transfused with almost 100 units of packed cells. Miraculously, there was no complication of these transfusions. She was endoscoped countless times without finding a source in the upper GI tract or the colon. Finally a laparotomy was performed and clusters of ectatic vessels were seen throughout the small bowel but dominant in the jejunum. A random resection of three feet of jejunum was performed. Pathologists found dilated vessels in the sub mucosa but no bleeding site. The patient did not bleed again for ten years. Bleeding finally recurred again. A capsule study was performed showing recurrent jejunal bleeding. A further resection was performed leaving only three feet of ileum. There has been no further bleeding for five years.

Meckel's Diverticulum

GI bleed due to ulceration of the ileal mucosa by ectopic gastric mucosa in the diverticulum occurs most often in childhood. This complication may however be delayed until the person is a young adult. Other causes of lower GI bleed are rare in the 10-30 year age group. If a brisk lower GI bleed occurs in a young adult, this diagnosis should be suspected. If a colonoscopy shows no mucosal lesion, and blood is seen issuing from the ileocecal valve, the diagnosis of a Meckel's diverticulum is highest on the list. The bleeding site can be confirmed to be in the RLQ or terminal ileum on a RBC scan or selective angiography, providing it is "active" during the scanning period or contrast injection. Visualization of active bleeding can be frustrating (in all lower GI bleeds) as the lesions are often small or multiple, and always seems to stop bleeding before the scan or contrast injection begins. In the instance of a Meckel's diverticulum with ectopic gastric mucosa, however, a Meckel's scan with ^{99m}Tc Pertechnetate will identify the Meckel's. The demonstration of the diverticulum with ectopic gastric mucosa, with no obvious source in the colon, is sufficient evidence to proceed with resection of the Meckel's and adjacent ileum.



This patient bled quite significantly from an ulceration adjacent to a Meckel's diverticulum with gastric mucosa. The bleeding site was demonstrated conclusively by selective angiography after active bleeding was provoked by a combination of anticoagulant and fibrinolytic. This strategy was displayed after multiple failed attempts to demonstrate the bleeding site [bleeding would always be stopped before an angiogram could be organized]. Needless to say, have a surgical consultant aware and an operating room available before trying this.



Ulceration of the ileum close to the opening of a Meckel's. This is the lesion provoked to bleed actively in the angiogram shown above. Bleeding ulcerations caused by a Meckel's will not be in the Meckel's diverticulum but will be in the adjacent small bowel. If only the Meckel's is removed the ulceration may still bleed. A segmental resection of the ileum and the Meckel's is required.



The anomaly was first described by Johann Meckel in 1808. Bleeding is the commonest complication, followed by intestinal obstruction and Meckel's diverticulitis. Surprisingly, the lesion has a significant mortality rate of 5-10%.

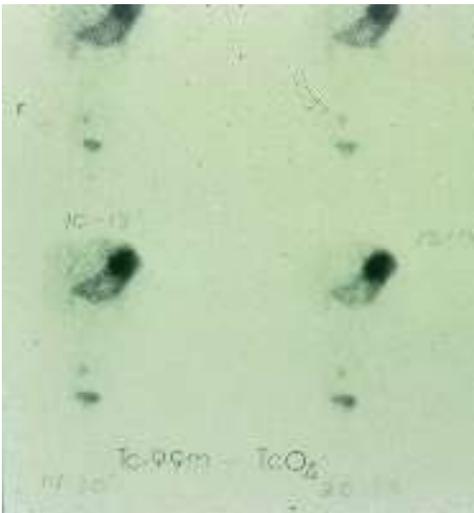


About 50% of Meckels will have ectopic gastric mucosa. The ulceration caused by the secretion of acid will be in the adjacent ileum and not in the diverticulum, so in surgery for a lower GI bleed caused by a Meckels, the diverticulum and a segment of ileum should be removed.



A Meckel's diverticulum, with ectopic gastric mucosa, causing adjacent ulceration of the ileum is a well-known cause of lower GI bleeding in infants and younger children. Occasionally a Meckels may remain quiescent in childhood and then present with complications in an adult. Like peptic ulcers in the upper GI tract, these lesions may bleed quite impressively. Depending on the rate of blood loss, it may present to the rectum as bright red blood or as a maroon colored stool.

Acid secreting gastric mucosa can be labeled with ^{99m}Tc -pertechnetate. The image here shows the isotope concentrated in the stomach in the fundal epithelium and also in the ectopic gastric mucosa in the Meckels in the right lower quadrant in the abdomen. This patient was thirteen years of age.



This Meckels scan with Technitium ninety-nine showed the expected dense uptake of the isotope in acid secreting proximal gastric mucosa.

In the right lower quadrant of the abdomen there is a small spot of increased activity, indicated by the arrow, which is ectopic gastric mucosa in a Meckels. A larger area of uptake just below, in the midline, is likely in urinary bladder.

Small Bowel Tumors

Primary tumors in the small bowel are rarely encountered. Some of the neoplasms (carcinoids and adenocarcinoma) seldom bleed acutely.

Stromal tumors (leiomyoma/leiomyosarcoma) can occur anywhere in the GI tract and will often bleed acutely. Examination of the lesion reveals an "umbilicated" appearance with deep central ulceration of the luminal aspect. Hemorrhage is from a disrupted vessel in this "pit" like ulceration.

The patient may also complain of cramping pain suggestive of a partial obstruction. If the lesion is large enough or the abdominal wall thin enough, the tumor may be found on physical exam as a mobile mass.

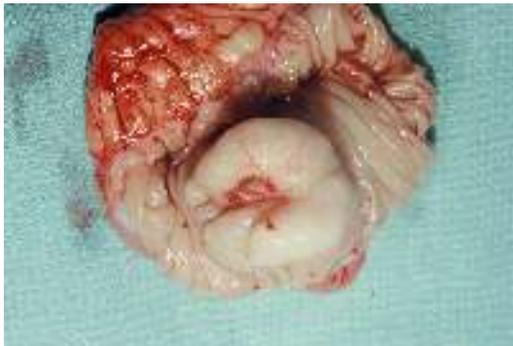
As in Meckel's diverticulum the small bowel can be implicated by exclusion if colonoscopy shows no bleeding site. An angiogram may define the lesion because of the density of vessels in the tumor, even though it is not actively bleeding. The resection of bleeding small bowel tumors should be regarded as urgent/emergent as the rate and volume of blood loss is unpredictable. Lymphomas in the small bowel may obstruct, cause intussusception, perforate or bleed. The last two complications are prone to occur during rapid lysis of tumor after chemotherapy.



This patient who had a series of brisk lower GI bleeds requiring transfusion had a selective angiogram which showed a vascularized tumor in the left lower quadrant of the abdomen in the late phases of the angiogram. There is no active bleeding demonstrated, but the density of vasculature within the tumor reveals the lesion.



Leiomyomas are notorious for their capacity to cause gastrointestinal bleeding. These stromal tumors can occur in the esophagus, stomach, duodenum or anywhere in the small intestine. This lesion was in the mid small bowel and caused periodic brisk hemorrhages. It was large enough to be detected by a physical examination as a mobile mass in the lower abdomen. A colonoscopy was negative. The lesion was demonstrated by a selective angiogram. The tumor has a typical bosselated appearance, dumbbelling out through the small bowel wall and presenting an ulcerated tumor on the mucosal side.



This photograph of a resected stromal tumor of the small bowel shows the typical central pit like ulceration of the tumor apparently caused by central necrosis. Because of the vigorous vascular supply of these lesions, this necrosis often comes to involve one of these vessels and brisk intestinal hemorrhages may result.



This patient with AIDS developed a lymphoma of the gastrointestinal tract. This lesion was intussuscepting. The lymphoma grows rapidly and is prone to central necrosis. Hemorrhage may result from vessels involved in the necrotizing process. Rapid lysis of the tumor by an effective chemotherapeutic approach, may result in either perforation or hemorrhage.

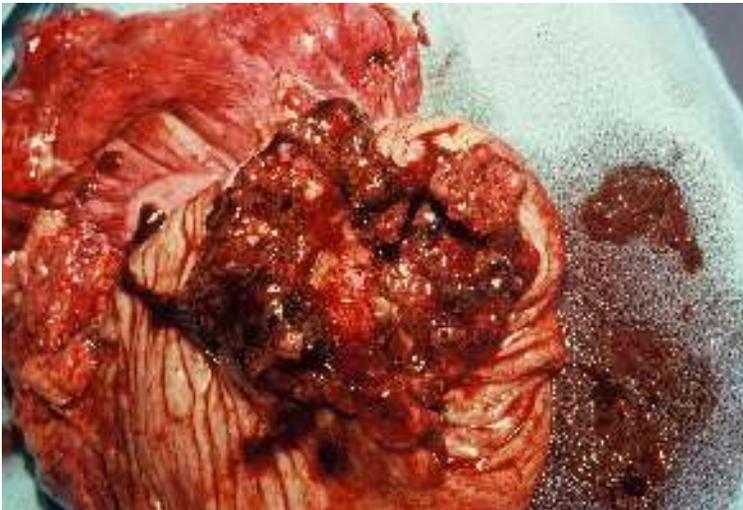
Approach to the patient with GI bleeding

GI bleeding will always declare by hematemesis or passage of blood from the rectum. Hypotensive, unstable patients who have not vomited blood and have no blood or melena on a digital rectal exam are not hypotensive as a result of GI bleeding.

Patients who present with symptomatic anemia, probably as a result of slow GI blood loss, or have noted the passage of melena stool may have a slower paced investigation than those who have vomited blood or passed blood and clots per rectum.

The decision to admit to hospital versus continuing with ambulatory care/investigation would be based on the age/vigor of the patient, provisional diagnosis, and the severity of the anemia.

Inpatient or outpatient; two cases.



Example: A 70-year-old patient, complaining of fatigue and shortness of breath, is found to have Hgb of 50gms. The morphology is microcytic, hypochromic. Provisional diagnosis is cecal cancer.

Admit to hospital and pursue investigation in this setting. The patient needs to be slowly transfused up to a safe Hgb and then colonoscoped. If colon cancer is found, proceed with the resection in the next urgent time [2-3 days]

Severely anemic, elderly patients with irreversible pathology, encountered in the ER or outpatient setting, need immediate hospital management.



Example: A 30-year-old patient with back pain self medicated with quantities of ASA . He then notes passage of black, tarry stool. Hgb is 100gms. Provisional diagnosis is gastritis or acute duodenal ulcer secondary to ASA abuse

Could be started on proton pump inhibitors, stop salicylates. Follow closely as outpatient. If an upper GI series showed evidence of the suspected duodenal ulcer, this would be sufficient investigation; otherwise, an urgent upper GI scope, as an outpatient, would be required.

Younger patients, without co-morbidities, with moderate anemia and reversible pathology can often be managed as outpatients.

Acute GI bleed, in the ER

Patients who have vomited or passed fresh blood by rectum will almost always present to an emergency room, usually by ambulance. They should be triaged into an acute care area for immediate nursing assessment and establishment of monitoring. The patient needs to be seen by a physician immediately. Assessment and management should proceed simultaneously. Patency of the airway, adequacy of ventilation and status of the circulation should be quickly evaluated, while peripheral venous access is established in the upper extremities. Base line blood work is taken and the patient should be x matched for at least 6 units. Various degrees of urgency for x matched blood may be asked for on requisitions. It may be best to stratify the request; i.e. further units can be more extensively matched.

Upper or lower GI bleed ?

If the patient has presented with hematemesis, this issue is obviously resolved. Blood will not reflux back to the stomach from below the ligament of Treitz.

If blood in any form [melena, maroon, bright], is only being passed by rectum, then the question of upper vs lower is not answered with certainty. Passing an NG tube and obtaining bilious return will rule out the upper GI tract as the source.

Making this distinction early is useful, as the answer provides direction to the special investigations which need to follow.

Upper GI bleed needs “optimal” in pace of investigation and surveillance



A deeply penetrating peptic ulcer is probably the most threatening and unpredictable lesion causing a GI hemorrhage. These individuals often have a favourable prognosis, providing they receive prompt and definitive management." Good management" in my opinion, means that the patient bled *once* [the acute bleed leading to presentation], was quickly resuscitated, had endoscopy, went to the OR for suture of the vessel or resection of the ulcer and *did not bleed again*.

Many of these individuals will display evidence of circulatory compromise, with hypotension, pallor, tachycardia, weak peripheral pulses, with cool, mottled distal extremities. Oxygen therapy is required, with rapid infusion of a balanced salt solution. The volumes, rate, and end point of crystalloid boluses will be based on the patients' response to volume expansion. Critically hypotensive individuals with a history of massive bleeding should have early consideration for administration of O-negative blood. A general rule might be that a patient not stabilizing after 2 litres of crystalloid should receive universal donor (O-negative) blood. Continued bolusing with normal saline in the face of on-going blood loss, or initial, massive (greater than 40% blood volume) hemorrhage, will result in profound hemodilution. The patient may then be "full" from standpoint of the intravascular volume, but at the expense of critical dilution of coagulation factors, platelets and serum proteins. Hgb may then be less than 50gms, compromising O₂ delivery to vital tissues.



This patient's angiogram was retrieved from the library of the radiology department of the Vancouver General hospital. I am not aware of the course of events or the survivorship of the patient concerned. The selective angiogram, shows a massive hemorrhage from the region of the gastroduodenal artery. The hemorrhage is obviously "in progress". Contrast spills into the antrum of the stomach and then outlines the pyloric channel with a halo- like shadow. Attempts to occlude the gastroduodenal vessel by angiographic techniques might have been attempted. Frequently, these fail because of the rich blood supply and collateralization. A well-placed proximal occlusion may just result in a rush of arterial flow through the gastroepiploics and into the gastroduodenal from the distal end. Extensive and repeated attempts to thrombose this vessel completely may infarct the duodenal loop. The patient would have been best served by laparotomy, duodenotomy and ligation of the gastroduodenal vessel. I was unable to trace the outcome of this precarious situation.

Fortunately, it is infrequent that a massive bleed from an eroded vessel continues relentlessly. Blood pressure often falls to critical levels as a result of the initial hemorrhage. This "bleeding" phase may continue for only a few minutes, but can result in a blood loss greater than 2 litres. This often happens outside of hospital; patients are found by paramedics with unobtainable B.P. and arrive in the ER on the verge of cardiac arrest. During this interval, the eroded vessel usually clots, and it becomes possible to resuscitate the patient. Large amounts of blood may still be in the stomach. The lower GI tract will also contain 1-2 litres of blood, which will then start to pass per rectum. At this point, a nasogastric tube (at least 18F) should be inserted, and the stomach suctioned and lavaged. The purpose of this is not to stop bleeding, but to try to clear the stomach, as much as possible, preparatory to endoscopy. Liquid blood should be aspirated; clots may pass into the duodenum. Some anxiety may be caused by the continued passage of very "bright" blood from the rectum, suggesting on-going bleeding. However, if the patient is responding to the infusion of saline/blood with better B.P. and peripheral perfusion, bleeding has likely stopped.

The interval when bleeding has stopped, and the patients' vital signs are stabilizing, *will not last forever*. The clots in the eroded vessel are "soft". As pressure in the feeding arteries is restored,

and gastric/duodenal secretions bathe the ulcer bed, the thrombus will be lysed or expelled and another shocking hemorrhage will occur

Take advantage of the window of opportunity provided by your resuscitation and proceed quickly with investigation.

The “investigation” referred to above will almost always be an upper GI endoscopy.

- 1 The vast majority of pathologies causing upper GI bleeds will be seen at gastroduodenoscopy
- 2 There are therapy options
- 3 the patient can usually be quickly moved to an OR, if necessary; endoscopy may actually be done in the OR

Do not take the patient to an investigation which has few therapy options and is remote from the OR. [see below]

Identifying patients bleeding from esophageal varices

Variceal hemorrhage, in contrast to bleeding from arterial sources, may not only be massive, but it may be more relentless, demanding an immediate intervention to prevent recurring critical falls in B.P. It may be quite impossible to organize and carry out an endoscopy. The endoscopist will also be looking at a torrent of blood, precluding any injection or banding. If these individuals can be identified by history and physical exam to have portal hypertension, then a Minnesota tube should be inserted, and the balloons inflated. With traction on the tube, gastric/esophageal varices should be tamponaded, and bleeding should stop. In the rare event that massive bleeding from the esophagogastric junction is not from varices (e.g. proximal gastric ulcer or Mallory Weiss), the Minnesota tube will not be effective. Fresh blood will continue to be aspirated from the gastric port, and an urgent laparotomy or angiographic intervention will be required.



A correct diagnosis is pivotal to the appropriate management of an upper GI bleed; even if the patient does have physical evidence of portal hypertension. It may be warranted to attempt endoscopy, even in the presence of repeated hematemesis. If varices can be “glimpsed” and blood is issuing from them, at least appropriate measures, which are medical/procedural and not operative, can be taken.

Keeping a Minnesota tube in the optimal position, with proper gastric and esophageal balloon inflation, continuous effective traction, plus protect the airway, is a considerable challenge and may require ICU management. Endotracheal intubation to protect the airway may be required. Patients with pre existing liver failure will inevitably be precipitated into worsening encephalopathy, and may become comatose. Measures to reduce the bacterial “load” in the gut, in addition to evacuating the blood from the colon should be initiated immediately. Fortunately, not all patients with esophageal varices bleed massively. When the rate of blood loss is less, pharmacologic methods may be successful in arresting bleeding. Pitressin and Octreotide infusions have both been effective. In these instances, endoscopic techniques, injecting sclerosants or banding varices may be successful.

Lower GI bleeding is often less threatening

Lower GI bleeds, especially those originating in the colon, seldom result in rapid loss of blood volume. Patients present with less threatening hemodynamics; tachycardia, postural hypotension, diminished quality of peripheral pulses are often evident, but deep shock is very unusual. They are usually easily stabilized, even if they continue to bleed.

Individuals who have not experienced hematemesis, are relatively stable or easily resuscitated, but are passing blood/clots per rectum, are very likely to be bleeding from below the duodenal jejunal flexure. However, it is still a good precaution to pass a nasogastric tube and irrigate the stomach, to conclusively rule out an esophageal or gastric source for the bleeding. If clear bile is aspirated, then the duodenum is also excluded. An aspirate of what appears to be gastric contents makes duodenal bleeding unlikely but not impossible as even major blood loss in the duodenum may be prevented from refluxing through the pylorus, if the channel is strictured or in spasm.

Monitoring the GI bleeder

All patients with GI bleeding, whether they are proceeding to operation, diagnostic and therapeutic interventions, or are to be admitted to an acute care area for medical treatment and monitoring, should have a bladder catheter for hourly urine volumes, frequent vital signs, O₂ sat. and ECG monitoring as well as frequent repeat Hgb or Hct levels.

Patients admitted to medical wards should be in a safe environment where continuous nursing surveillance is possible. Many acute care areas of larger hospitals have designated “step-down” units of 6-8 beds where good surveillance of 6 patients could be assured by 2 or 3 nurses. Hospital rooms remote from the nursing station are not an appropriate site for a patient with an acute GI bleed.



An image from a selective angiogram demonstrates the robust nature of the branches of the celiac axis. Large duodenal ulcers may penetrate and erode the gastroduodenal artery, indicated by the arrow. Deeply penetrating posterior wall gastric ulcers could conceivably either erode the left gastric artery or even the splenic artery. Once a major visceral artery “opens”, the peripheral resistance of its field of perfusion is by-passed and arterial blood rushes to the site of the erosion in much greater volumes than the normal flow rate for that specific vessel.

Decisions regarding timing of investigations

Regardless of the suspected source of an acute GI bleed, patients who have needed resuscitation should have immediate investigation. Waiting “until the morning” or until “Monday” to save resources or because of “quality of life” issues (those of on call physicians and technicians) is not acceptable.

An opportunity to identify the site of bleeding (knowledge which may be critical in later management) or visualize a threatening lesion which needs operative management immediately may be missed. The patient may be stable transiently, but suddenly rebleed and be precipitated into a critical situation made much worse by the lack of information about the site and nature of the bleeding lesion.

Taking a history will help

During the course of resuscitation and other early management, a history needs to be taken or gathered from a variety of sources, and the physical examination needs to be completed. Distressed, hypotensive patients experiencing hematemesis or voluminous bloody bowel motions are difficult to interview. Valuable information may be obtained from previous hospital records, family physician and relatives. Obtain what information you can from the patient. Previous gastrointestinal disease, surgeries, treatment for these conditions is obviously important. Medications and alcohol abuse often play a pivotal role in cases of GI bleeding. Liver disease is a frequently encountered collateral condition. Patients with a history of peptic ulcer disease, esophagitis, inflammatory bowel disease may have been experiencing an escalation of their symptoms.

It may be a good strategy to focus on determining the acuteness and severity of the blood loss. That the patient has bled massively may be self-evident from physical signs, but if bleeding has stopped and some fluids have been given, the patient may be deceptively "stable". A story of sudden onset of nausea, followed by a huge emesis of bright blood and then collapse gives a clear signal that the patient is at grave risk; establishing the site of this type of bleeding is emergent.

Patients who have had previous GI bleeds and investigation/operations, should they re-present, will likely be bleeding from the same region, same condition. A "track record" of heavy alcohol consumption may suggest cirrhosis, portal hypertension and varices, but many of these patients do not have portal hypertension and are bleeding from other causes.

Patients with peptic ulcer disease are often asymptomatic with respect to the ulcer. Some will have an increase in ulcer-type symptomatology in the weeks prior to developing a complication such as perforation or bleeding. As a rule, perforated ulcers do not bleed (anterior ulcer perforates, posterior ulcer bleeds.)

Abdominal pain is rarely a dominant complaint in patients presenting with GI bleeding. Pain is not a feature of superficial gastric and duodenal ulcers, diverticulosis or angiodysplasia, which cause over 80% of all acute GI bleeds.

Ulcerative and ischemic colitis may lead to significant lower GI bleeding. Diarrhea and lower abdominal cramps suggest these diagnoses.

The personal habits and medications of patients with GI bleeding may be revealing. Since self-abuse in various forms is often identified in patients with upper GI hemorrhage, a specific diagnosis cannot be established by finding that the patient consumes a very poor diet, drinks heavily, consumes salicylates, NSAIDs and other drugs, smokes 2 packs a day and is completely non-compliant. An accurate medication history needs to be obtained, and this may require a pharmacist search. Drugs which inhibit platelet function (plavix) or anticoagulate the patient are obviously important.

Physical exam; what findings help?

Specific, diagnostic physical signs, discovered on examination of the abdomen are uncommon. Since a high percentage of GI bleeds are caused by peptic ulcers, esophagitis, gastritis, angiodysplasia and diverticulosis, the examination of the abdomen is often negative, and hence not helpful.

The physical signs of portal hypertension should be sought, as they may support a decision to treat a patient for variceal hemorrhage, should endoscopy not be immediately available or not possible because of massive on-going bleeding and instability of the patient's vital signs. "Caput medusae" at the umbilicus is rarely seen, but ascites is common. Splenomegaly is also frequently demonstrable by a careful exam.

Viewed from another direction, a patient with a massive upper GI bleed who does not have ascites or splenomegaly is very likely to be bleeding from a peptic ulcer.

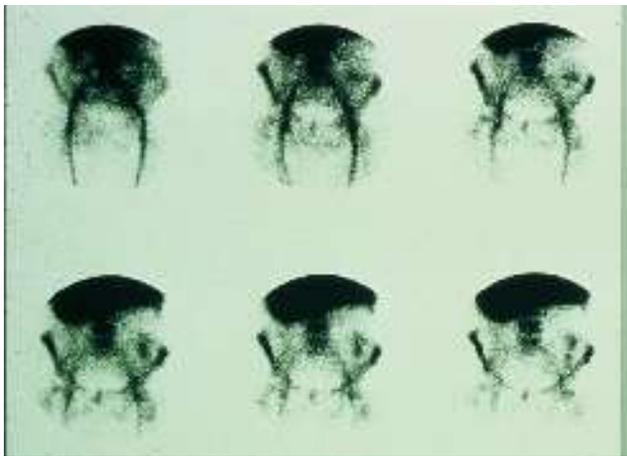
If the patient has a lower GI bleed, the abdomen should be carefully searched for a palpable mass, which would suggest a small bowel tumor.

Ischemic colitis, which may present as rectal bleeding, may be suggested by LLQ tenderness.

A rectal examination will establish exactly what is being passed, in a case of lower GI bleeding.

The "blood" passed per rectum may have occurred prior to admission, or even in hospital, it may have been disposed of. Witness's accounts of whether it was bright red, maroon, or melena may be confusing.

In a patient with a lower GI bleed, passing bright red blood per rectum (BRBPR) a proctoscopic examination to 10-15cm should be performed (at the bedside) to exclude the rectum as a source of bleeding.



A series of images from a red blood cell scan, shows the gradual appearance of labeled red cells just medial to the left iliac bone, presumably within the sigmoid colon. The scan provides adequate confirmation of the site of hemorrhage as in the lower descending or sigmoid colon.

The initial assessment, resuscitation, establishment of N/G tube and foley, gathering of history and completion of the physical examination have all proceeded sequentially but with some "overlap" so that within an hour of presentation the patient should be prepared for further investigation to localize the site of bleeding.

Laboratory work should be available for review by this time.

If the hemoglobin is normal after a recent major bleed, this is because the bleed was acute and hemodilution has not yet begun. Anemic patients have been bleeding more slowly and over a span of at least 2-3 days, often longer. This finding often correlates with a history of melena stools, "coffee-ground" emesis and an initial assessment reflecting more stable vital signs.

Leukopenia and thrombocytopenia should suggest hypersplenism, splenomegaly and portal hypertension.

Coagulation assessment (INR and PTT) is obviously important. Any prolongation of the indices should be corrected with FFP. Any anticoagulant or platelet adhesiveness inhibitor should be stopped. Even minimal elevations in INR (1.2 – 1.6) should be reversed with I.V., vitamin K and FFP.

Liver function is a determinate of survival from any adverse medical event and the indices predict and guide management of variceal hemorrhage. Elevated bilirubin, low albumin and prolonged INR separate patients into Child's group A, B, and C.

Low fibrinogen levels occur in late stages of liver failure and predict a poor outcome.

Serum ammonia level is an index of portosystemic shunting and liver failure, and relates to encephalopathy and coma.

What is the best investigation?

A decision regarding further investigation needs to be made at this stage, and there is dispute and probably several defensible courses to pursue. It would be a very rare circumstance that a GI bleed proceeded to laparotomy without benefit of any localizing study. Some suggestions, which I believe are "reasoned", follow:

1. Massive upper GI bleed, resuscitated, either slowed or stopped
 - Immediate upper GI Endoscopy
2. Upper GI bleed – melena, anemia, no change in vital signs upper GI
 - Endoscopy within 24 hours



The patient experienced hematemesis and then passed melena stool about 3 hours prior to an emergency admission. Paramedics noted hypotension, BP 80\0, at the site and during transport.

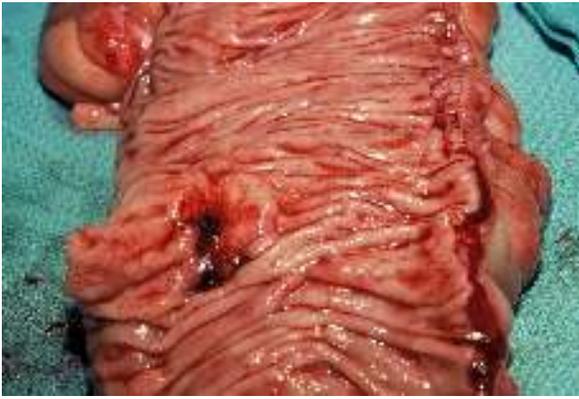
In the ER, the patient responded to a litre of NS and appeared to stop bleeding.

Endoscopy 1 hr later revealed a 2cm, benign-appearing ulcer in the pre-pyloric region. There was no active bleeding, but a thrombus was seen in the margin of the ulcer.

The decision regarding continued medical treatment vs early surgery is not easy. If the road chosen is not a success, the decision will likely be judged in hindsight by other experts, not participating at the time.

3. Lower GI bleed – appears to continue to bleed. Requiring resuscitation.
 - Angiography immediately
4. Lower GI bleed – probably stopped, stable patient.
 - Prep and colonoscopy

-



The patient saw fresh blood in the toilet water and on the surface of the stool several times over a span of 2 weeks. This was attributed to her anticoagulants [on Coumadin, with an INR of 2.8] Then the patient passed a large blood clot. Staff at the care home called an ambulance

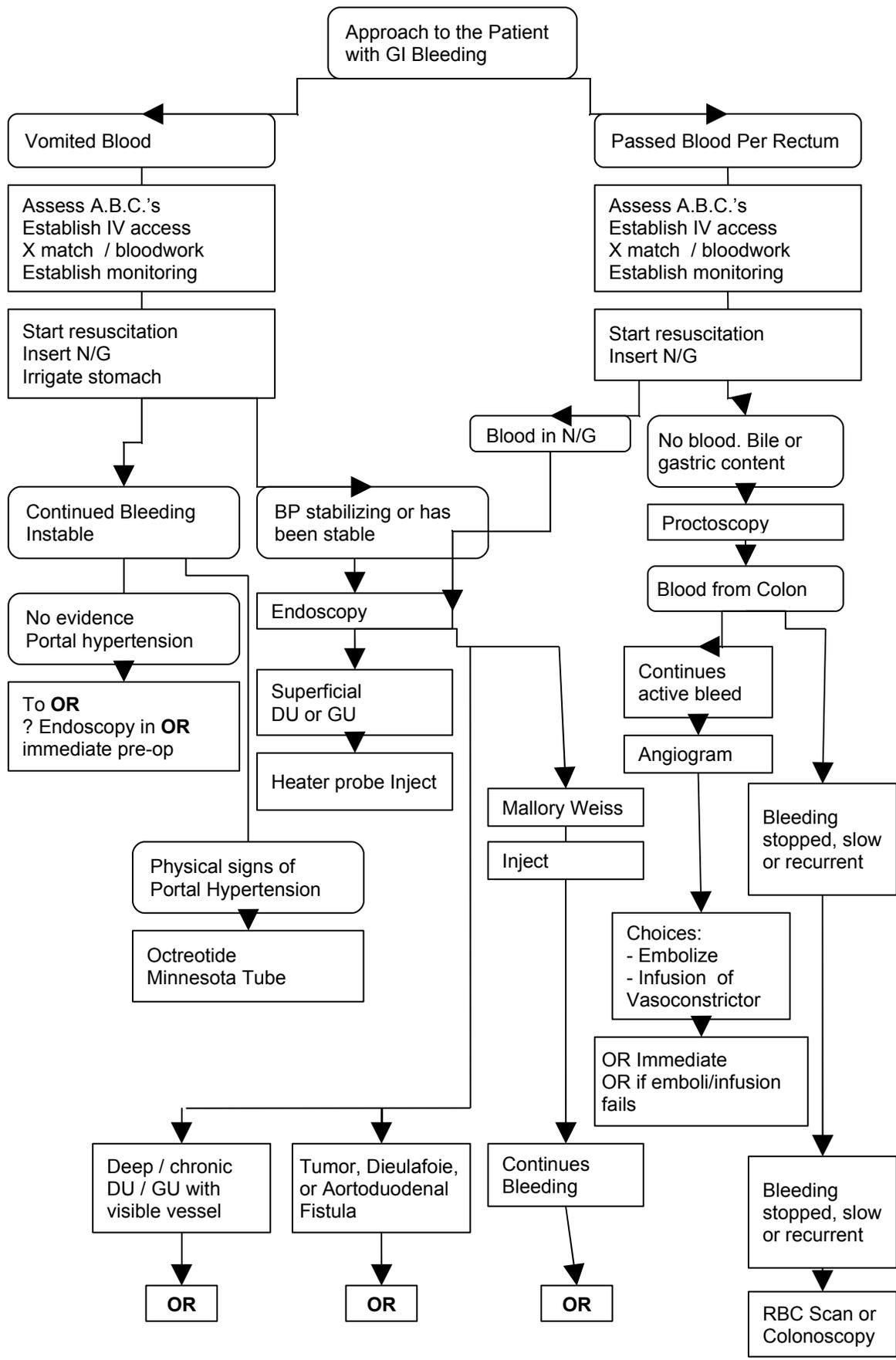
The patient was stable with Hgb of 95 gms. She was transfused and the colon prepped. Scoping revealed an ulcerating lesion in the descending colon.

Anticoagulants provoke bleeding from pre-existing lesions; they are

5. Lower GI bleed – Colonoscopy was unsuccessful in identifying site, but diverticula or other potential sources of bleeding seen in both the R and L colon.. Bleeding recurs
 - Labelled RBC scan.

6. Lower GI bleed. colonoscopy normal. Consider small bowel tumor or Meckels.
 - Meckels scan
 - Angiogram (vascular tumor)
 - M2A capsule.

FLOW CHART FOR MANAGEMENT



Other Reading:

1. From the Family Practice Notebook.

- I. **Epidemiology**
 - A. Accounts for 350,000 hospitalizations in U.S. yearly
- II. **Risk factors**
 - A. [Aspirin](#) or [NSAID](#) use (most common cause)
 - B. [Helicobacter Pylori](#) infection
 - C. Elderly (especially over age 70 years)
 - D. Acid suppression therapy does not reduce bleeding risk
- III. **Causes: Adults**
 - A. [Duodenal Ulcer](#) (30-37%)
 - B. [Gastric Ulcer](#) (19-24%)
 - C. [Esophageal Varices](#) (6-10%)
 - D. [Gastritis](#) or [Duodenitis](#) (5-10%)
 - E. Esophagitis or esophageal ulcer (5-10%)
 - F. Mallory-Weiss tear (3-7%)
 - G. Gastrointestinal malignancy (1-4%)
 - H. Dieulafoy's Lesion (1%)
 1. Artery at gastric fundus may bleed heavily
 2. Difficult to identify on endoscopy
 - I. Gastric antral vascular ectasia (0.5 to 2%)
 1. Longitudinal erythematous stripes on gastric mucosa
 2. Known as Watermelon stomach
 - J. Arteriovenous malformation
 - K. Angiodysplasia of stomach or duodenum, associated with
 1. [Chronic Renal Failure](#)
 2. [Aortic Stenosis](#)
 3. [Cirrhosis](#)
 4. [Von Willebrand's Disease](#)
- IV. **Causes: Children**
 - A. Esophagitis
 - B. [Gastritis](#)
 - C. [Peptic Ulcer Disease](#)
 - D. [Esophageal Varices](#)
 - E. Mallory-Weiss Tear
- V. **Signs**
 - A. [Hematemesis](#)
 - B. [Coffee-Ground Emesis](#)
 - C. [Melena](#)
 - D. [Hematochezia](#) (if bleeding is brisk)
 - E. Nasogastric aspirate bloody (15% false negative)
 - F. Elevated [Renal Function](#) tests (BUN, [Serum Creatinine](#))
 - G. Hyperactive Bowel Sounds
- VI. **Labs**
 - A. BUN to [Creatinine](#) ratio
 1. Does not reliably distinguish upper GI source
- VII. **Evaluation**
 - A. See [Upper GI Bleeding Score](#)
 - B. See [Upper Endoscopy Evaluation of GI Bleeding](#)
- VIII. **Management: Initial**
 - A. See [Acute Gastrointestinal Bleeding Management](#)
 - B. [Nasogastric Tube](#) with aspirate
 1. Fresh blood suggests persistent bleeding
 2. Avoid lavage due to aspiration risk
 - C. If severe bleeding and suspected variceal source

1. See [Esophageal Varices](#)
 2. Octreotide 50 ug bolus, then 50 ug/hour
 3. Avgerinos (1995) J Hepatol 22(2):247-8
- IX. **Management: General Measures**
- A. [Helicobacter Pylori](#) management
 - B. Empiric acid reduction ([Proton Pump Inhibitor](#))
 1. Not proven in-vivo to aid clotting
 2. No proven benefit in mortality and other outcomes
 3. Does not lower overall [Incidence](#) of re-bleeding
 4. [Omeprazole](#) may heal ulcer if near-achlorhydria
 5. Daneshmend (1992) BMJ 304:143-47
- X. **Management: Very low risk patients**
- A. Indications
 1. Hemodynamically stable with normal lab testing
 2. No evidence of significant bleeding in last 48 hours
 3. [Nasogastric Tube](#) aspirate without blood
 - B. Protocol
 1. Home with follow-up within days
 2. General measures as above
- XI. **Management: Low risk patients**
- A. Indications
 1. Hemodynamically stable within 1 hour of [Resuscitation](#)
 2. Minimal [Blood Products](#) required (2 PRBC or less)
 3. No evidence of active bleeding
 4. [Nasogastric Tube](#) aspirate without blood
 5. No active comorbid medical conditions
 - B. Protocol
 1. Consider for rapid protocol
 - a. Immediate [Upper Endoscopy Evaluation of GI Bleeding](#)
 - b. Discharge to home if low-risk endoscopy results
 2. Admit if rapid protocol not available
 - a. Follow moderate risk patient protocol below
 3. General measures as above
- XII. **Management: Moderate risk patients**
- A. Indications
 1. Tachycardia persists despite [Resuscitation](#)
 2. [Blood Products](#) required >2 PRBC
 3. Active comorbid condition
 - B. Protocol
 1. General measures as above
 2. Admit to regular medical bed
 3. Upper endoscopy when patient stabilized (<24 hours)
 - a. See [Upper Endoscopy Evaluation of GI Bleeding](#)
 - b. Disposition based on Upper Endoscopy results
 - i. Low risk endoscopy: Observe for 24 hours
 - ii. Moderate risk endoscopy: Observe for 48-72 hours
 - iii. High risk endoscopy
 - Initially observe in ICU for at least 24 hours
 - Observe in hospital for 72 hours total or more
- XIII. **Management: High risk patients**
- A. Indications
 1. Active ongoing bleeding
 2. Hypotension persists despite [Resuscitation](#)
 3. Severe active comorbid condition exacerbation
 4. Liver disease exacerbation
 5. [Endotracheal Intubation](#) for airway protection
 - B. Protocol
 1. General measures as above

2. Admit to intensive care unit for first 24 hours
 3. Observe in hospital for 48 to 72 hours or more
 4. Urgent upper endoscopy when stabilized
 - a. See [Upper Endoscopy Evaluation of GI Bleeding](#)
 5. Consider arteriography if source not evident
- XIV. **Management: Refractory and Recurrent Bleeding**
- A. Indications
 1. Persistent or recurrent bleeding despite EGD
 2. See [Upper Endoscopy Evaluation of GI Bleeding](#)
 - B. Protocol
 1. Surgical Intervention
 2. Consider embolization for non-surgical patient
- XV. **Prognosis: Outcomes**
- A. Overall Mortality: 2-15% (often related to comorbidity)
 - B. Bleeding stops and does not recur: 70% (<2% Mortality)
 - C. Bleeding after initially stopped: 25% (10% Mortality)
 - D. Continued active bleed: 5% (30% Mortality)
- XVI. **Prognosis: Predictors**
- A. Bleeding characteristic predictors of poor outcome
 1. See [Upper GI Bleeding Score](#)
 2. [Emesis](#) or nasogastric aspirate contains red blood
 3. Low initial [Hematocrit](#)
 4. Coagulopathy (low platelets or high INR)
 - B. Comorbid condition predictors of poor outcome
 1. Active [Coronary Artery Disease](#)
 2. [Congestive Heart Failure](#)
 3. Active lung disease
 4. [Renal Failure](#)
 5. Sepsis
 6. Metastatic cancer
 7. Advanced liver disease
 8. Advanced age
- XVII. **References**
- A.  [Gupta \(1993\) Med Clin North Am 77\(5\):973-92](#)
 - B.  [Fallah \(2000\) Med Clin North Am 84\(5\):1183-208](#)
 - C.  [Longstreth \(1995\) Am J Gastroenterol 90\(2\):206-10](#)
 - D.  [Peter \(1999\) Emerg Med Clin North Am 17\(1\):239-61](#)
 - E.  [Terdiman \(1998\) Postgrad Med 103\(6\):43-64](#)

2. Review Article

Acute lower intestinal bleeding

Part II: Etiology, therapy, and outcomes

Gary R. Zuckerman DO

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American Society for Gastrointestinal Endoscopy

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3. Retrospective Analysis For acute Lower GI Bleeding

John R. Kirckpatrick, MD and Thomas Stahl MD

Washington Hospital Center Department of Surgery, Division of Colorectal Surgery, and the Medlantic Research Institute, Washington D.C.

4. Special Investigations

Complete intraoperative small-bowel endoscopy in the evaluation of occult gastrointestinal bleeding using the sonde enteroscope

M. J. Lopez, J. S. Cooley, J. G. Petros, J. G. Sullivan and D. R. Cave
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OBJECTIVE: To review our experience with intraoperative small-bowel Sonde enteroscopy in evaluating occult bleeding in the small intestine. **DESIGN:** Retrospective study with 100% follow-up. **SETTING:** University-affiliated, tertiary-care teaching hospital. **PATIENTS:** Sixteen consecutive patients referred with occult gastrointestinal bleeding in whom esophagogastroduodenoscopy, push enteroscopy, and colonoscopy had failed to identify the source of bleeding. Fourteen of the 16 patients had required one or more transfusions. **MAIN OUTCOME MEASURE:** Completeness of visualization, diagnostic accuracy, and complications of the procedure and follow-up for recurrent bleeding. **RESULTS:** In all 16 patients, intraoperative Sonde enteroscopy allowed visualization of the entire small bowel. In 14 of the 16, it revealed the cause of bleeding, which was ileal angiodysplasia in three patients, ileal ulcers in six patients, neoplasia in two patients, and ileal ulcers caused by Crohn's disease, small-intestinal enteropathy and varices caused by portal hypertension, and radiation stricture in one patient each. Two patients had normal small bowel mucosa. The patients with mucosal disease underwent small-bowel resection or oversewing of bleeding sites. Two surgical complications occurred: prolonged postoperative ileus (one patient) and small-bowel obstruction that resolved without surgery (one patient). Two of the patients with angiodysplasia had recurrent bleeding postoperatively. **CONCLUSIONS:** Intraoperative Sonde enteroscopy is safe and effective in localizing small-intestinal bleeding sites, providing complete visualization of the small-bowel mucosa without enterotomy while avoiding the trauma that can be caused by push endoscopy. It is the diagnostic assessment of choice in selected patients with occult gastrointestinal bleeding of presumed small-bowel origin.

M2A Capsule Endoscopy

Davey R. Deal, M.D., Louis Lambiase, M.D., Jianjun Li, M.D.

The M2A (mouth-to-anus) capsule endoscopy (CE) system developed by Given Imaging, Ltd. has provided gastroenterologists with the ability to examine the small intestine as never before. It was approved by FDA in August of 2001 and it is currently available in 33 countries worldwide. CE is being performed by two medical centers in Jacksonville, with forty studies completed at our hospital in the first half of 2002.

The CE is an 11x26-mm capsule that encases a digital camera, light-emitting diodes, batteries, and a transmitter. Images are taken twice-per-second and transmitted to a recording device worn on a belt by the patient. Thousands of images are transmitted to the recording device and then evaluated after the study is completed. It takes approximately two hours for a gastroenterologist to review the series of images produced. The capsule has a gastric transit time of approximately one hour and a small intestinal transit time of three and a half to four hours. Patients swallow the capsule in the morning and wear the recording device for eight hours. They may perform their regular daily activities during the study. The only bowel preparation involved is that the patient cannot eat after midnight the day prior to the study. The capsule is discarded after one use.

The new technology has its greatest utility in the evaluation of obscure gastrointestinal bleeding. Obscure GI bleeding is defined as bleeding of unknown origin that persists or recurs (i.e., recurrent or persistent iron deficiency anemia, FOBT positivity, or visible bleeding) after a negative initial or primary endoscopy (colonoscopy and/or upper endoscopy).¹ In approximately 5% of all patients with GI bleeding, no cause for the bleeding is evident even after an extensive

workup.² Twenty-seven percent of patients with obscure GI bleeding have been shown to have lesions in the small bowel.³

Prior to the development of CE, the nonsurgical evaluation of patients with obscure gastrointestinal bleeding was limited to push enteroscopy, Sonde enteroscopy, small bowel follow-through (SBFT) x-ray series, small bowel enteroclysis, technetium 99m-labeled red blood cell scan (TRBC), and angiography. All of these modalities are limited in their sensitivity for small bowel pathology, and they have been especially disappointing in terms of eliciting the etiology of obscure gastrointestinal bleeding.

In a study comparing CE to push enteroscopy to identify beads sewn into the small bowel of canines, the sensitivity of the capsule was 64% compared with 37% for push enteroscopy.⁴ In a study in humans comparing CE and push enteroscopy for the identification of a source of obscure gastrointestinal bleeding, CE was superior to enteroscopy in locating a bleeding source. This was due to the limitation of reaching distal small intestine by enteroscopy. In addition, patients preferred the CE and the image quality were judged as good to excellent. A bleeding site was discovered by CE in 64% of patients, and CE found a bleeding source in 5 of 9 patients with normal push enteroscopic exams.⁴ Lewis B, Swain P. Capsule endoscopy in the evaluation of patients with suspected small intestinal bleeding, a blinded analysis: the results of the first clinical trial. (*Gastrointestinal Endoscopy* 2001;53:AB70).

There are several drawbacks to CE. The main drawback is the difficulty in identifying the precise location of pathology when it is found. In May of 2002 Given Imaging announced the release of its new "M2A Plus", which will allow location of small intestinal disease with greater accuracy. The M2A Plus and the accompanying new version of RAPID® software create a graphic representation of a patient's GI tract to more accurately locate the detected pathology. A validation study prepared for the FDA showed that the location determined by the M2A2 system was comparable to the actual location found by fluoroscopy.

Another drawback to CE is the lack of ability to biopsy or treat pathology when it is found. CE is quite expensive, and insurance company reimbursement is poor considering the 1-2 hours required by the physician to review the images for one patient. The M2A CE has provided gastroenterologists the ability to more accurately assess for the presence of small intestinal pathology. Studies thus far indicate that it is more sensitive than other nonsurgical procedures for diagnosing the etiology of obscure gastrointestinal bleeding. The only absolute contraindication for capsule endoscopy is intestinal obstruction. The new technology will likely prove to be of benefit in diagnosing otherwise elusive small intestinal pathology such as undiagnosed small intestinal Crohn's disease and small intestinal tumors not seen with available radiologic imaging techniques. More studies are needed to determine its role in the diagnosis of other gastrointestinal pathologies.

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1. Zuckerman G et al. AGA technical review on the evaluation and management of occult and obscure gastrointestinal bleeding. *Gastroenterology* 118-1 2000.
2. Lahoti S. The small bowel as a source of gastrointestinal blood loss. *Curr Gastroenterol Rep* 1999; 1(5): 424-30.
3. Appleyard M et al. A randomized trial comparing wireless capsule endoscopy with push enteroscopy for the detection of small-bowel lesions. *Gastroenterology* 2000; 119(6): 1431-8
4. Lewis B, Swain P. Capsule endoscopy in the evaluation of patients with suspected small intestinal bleeding, a blinded analysis: the results of the first clinical trial. *Gastrointestinal Endoscopy* 2001;53:AB70.