Acute gastrointestinal bleeding: a review
Elroy P. Weledji, BSs, MSc (Lond), MBBCHBAO(Dublin), FRCS (Edinburgh)*

Abstract
Blood loss from the gut may be acute requiring emergency resuscitation, investigation, and management, or chronic and occult resulting in anemia. The upper gastrointestinal (GI) tract is more commonly a source of hemorrhage than the lower GI tract. Endoscopy is the mainstay of investigation and management. New endoscopic techniques and radiologic embolization have decreased the role of surgery in management, but collaboration between the endoscopist and surgeon remains. In this review the management of acute and chronic bleeding loss in the GI tract is discussed. The importance of resuscitation, clinically diagnosing, and treating the underlying problem is emphasized.

Keywords: Gastrointestinal bleeding, Acute, Resuscitation, Endoscopy, Radio-embolization, Surgery

Introduction
GI bleeding may occur from any part of the gut and remains a significant health problem. Ulcers are the most common cause of hospitalization for upper GI bleeding. The vast majority of clinical trials of therapy for upper GI bleeding focus on ulcer disease. A population-based audit of upper GI bleeding in the Western world gives the incidence of acute bleeding as 103 cases per 100,000 adults per year[1]. Of this, variceal bleeding accounted for only 4%. Overall mortality from GI bleeding was 14% (11% in emergency admissions and 3% among inpatients). Twenty-seven percent of patients were over the age of 80 years, as compared with <10% in the earlier studies were mortality was 10%-14%[2,3]. Thus, both incidence and mortality increased markedly with age. The little improvement in mortality over the years despite therapeutic advances must be related to the dramatic shift in the age of the population at risk[1,4]. As 70%-80% of upper GI bleeding stops spontaneously[1-3], there is greater importance in the simultaneous resuscitation, investigation, and active monitoring of the acutely bleeding patient than for operative intervention. The goal is to stop continuing hemorrhage and decrease the risk of rebleed. The formation of specialist GI bleeding units with dedicated properly trained endoscopists have minimized the morbidity and mortality of GI bleeding[5]. New endoscopic techniques and interventional radiology have decreased the role of surgery in management, but collaboration between the endoscopist and surgeon is still mandatory. Any trial of new therapeutic techniques must improve upon the natural hemostatic process to be of real benefit. The work has been reported in line with the SCARE 2018 criteria[5].

Acute upper GI bleeding
Etiology
The causes of upper GI hemorrhage in the United Kingdom are peptic ulcers/erosions (45%), idiopathic (24%), esophagitis (10%), gastro-esophageal cancer (5%), varices (5%), Mallory-Weis (M-W) tear (5%), angiodysplasia, or Dieulafoy ulcer (5%)[6]. Nonvariceal upper GI hemorrhage is the most common complication of peptic ulcers occurring in 15% of ulcer patients and accounts for the commonest cause of ulcer related deaths[7]. It tends to be more common in patients aged 60 and older. Esophagitis is a form of peptic ulcer disease, but usually only causes minor acute bleeding. Occasionally, a significant vessel may be involved with consequent massive arterial hemorrhage which must be distinguished from variceal bleeding. Carcinoma and lymphoma of the stomach, when at an advanced ulcerated stage, commonly bleed. This usually results in occult blood loss, but will occasionally present with acute hemorrhage.
The Dieulafoy’s lesion is a ruptured, thick-walled artery with little or no associated ulceration. It occurs in the fundus as a round mucosal defect with a protruding artery at the base[23]. The percentage of variceal bleeders increases in inner-city areas most probably because of alcoholic liver disease[3,7]. The common causes of upper GI hemorrhage are summarized in Table 1. Upper GI hemorrhage frequently occurs in hospital patients being treated for unrelated conditions. Of these, 25% bleed from acute erosive gastritis and 50% from peptic ulcers[8]. In ulcers in the posterior wall of the duodenum or the lesser curve of the stomach the gastroduodenal or left gastric arteries can be involved, and these lesions are particularly prone to massive hemorrhage and rebleeding after initial stabilization[9]. In addition, the atherosclerotic arteries of the aged will not favor vasospasm/constriction. Peptic ulcer disease is decreasing but the number of operations for bleeding ulcers remain unchanged. This is probably because ~10%–20% of peptic ulcers bleed without any antecedent symptoms[1], and the increased ingestion of aspirin and nonsteroidal anti-inflammatory medication in the elderly[4]. Presentations may vary from melena to occult hemopositive stools, to massive hemetemesis and shock[10]. Initial management of upper GI bleeding involves resuscitation, followed by endoscopy. Initial resuscitation involves assessment of airway, breathing, and circulation (ABC), establishing large-bore intravenous (IV) access, urinary catheterization, blood transfusions as required, correction of clotting abnormalities especially for patients with liver disease, and urgent endoscopy if the patient is unstable. Nasogastric tube insertion and aspiration with or without saline lavage of retained blood greatly aids endoscopic visibility. Head down and semi-prone positioning protects against aspiration pneumonia which is the commonest cause of death in these patients. If the patient is hemodynamically unstable resuscitation is carried out in the intensive care unit, followed by urgent endoscopy or resuscitation in theater with urgent endoscopy under general anesthesia (ie, the circulation assessment “C” stage of the Advanced Trauma Life Support (ATLS) continues in theater[13,14]. However, rapid exanguination calls for an immediate operation to stop the bleeding (resuscitative laparotomy) without prior endoscopy. Emergency endoscopy is both diagnostic and therapeutic. The diagnostic yield decrease with time following the initial bleed. Performed early it identifies the cause of bleeding in 90% of cases[8–10,12]. The aim is to stop continuing hemorrhage and decrease the risk of rebleed[10]. Preendoscopic proton pump inhibitor (PPI) may be considered to decrease the need for endoscopic therapy but does not improve clinical outcomes[13].

**Risk stratification of upper GI hemorrhage**

The mainstay of investigation and management is endoscopy, and, the definitive management is indicated by the overall risk of rebleeding and morbidity[10,12–14]. Rebleeding can be predicted by the endoscopic findings and several clinical factors (Table 2). An initial Rockall scoring system[14] is an appropriate tool for assessment before endoscopy, and is predictive of death and rebleeding in patients with ulcers or varices. If the initial (preendoscopic) score is 0 (age below 60 y, no shock, no comorbidity), there is an extremely low risk of death or rebleeding and non-admission or early discharge with appropriate outpatient follow-up is considered. If initial (preendoscopic) Rockall score is above 0, there is significant mortality. Score 1 has predicted mortality of 2.4%; score 2 has predicted mortality of 5.6% and a score > 8 has a high risk of death. Upper GI endoscopy is indicated for a full assessment of bleeding risk. If full (postendoscopic) Rockall score is ≤3, there is low risk of rebleeding or death and early discharge and outpatient follow-up is considered[13,14].

**Endoscopic management**

Endoscopy allows visualization of source of bleeding and therapeutic intervention and/or biopsy. Preendoscopic erythromycin is considered to increase diagnostic yield at first endoscopy[13]. Since in the majority of cases bleeding settles spontaneously, if the patient is hemodynamically stable upper GI endoscopy can be done on the next available list. If unstable the best option is endoscopy under general anesthesia in the operating theater where the airway can be protected to prevent aspiration. An appropriate multichannel gastroscope should be available that allows for the insertion of devices such as needles, probes, clips applicators, while allowing irrigation/aspiration. Endoscopic therapy should only be delivered to actively bleeding lesions, nonbleeding visible vessels, and when technically possible to ulcers with an adherent blood clot[13]. The endoscopic stigmata of recent hemorrhage can predict the risk of rebleeding and the need for endoscopic hemostasis or surgery (Table 3)[10]. In the best

### Table 1

**Causes of hemetemesis and melaena.**

| Esophagus | Stomach | Duodenum |
|-----------|---------|----------|
| Severe esophagitis | Gastric ulcer | Duodenal ulcer |
| Mallory-Weiss tear | Gastric erosion | Duodenitis |
| Esophageal varices | Gastric varices | |
| Esophageal carcinoma | Gastric carcinoma | |

### Table 2

**Rockall risk score.**

| Score | Variable | Age (y) | Shock | Comorbidity | Diagnosis | Major SH |
|-------|---------|---------|-------|------------|----------|---------|
| 0     | < 60    | No shock | No major comorbidity | Mallory-Weiss tear, no lesion identified and no SRH | None, or dark spot only | Blood in upper GI tract, adherent clot, Visible or spurring vessel |
| 1     | 60–79   | “Tachycardia” | CCF, IHD, major comorbidity | All other diagnoses | Blood in upper GI tract, adherent clot, Visible or spurring vessel |
| 2     | > 80    | “Hypotension” | Renal failure, liver failure, disseminated malignancy |
| 3     |        |        |       |            |         |

**Table 3**

**Esophagus:** Gastric ulcer, Duodenal ulcer

**Stomach:** Gastric erosion, Gastric varices, Gastric carcinoma

**Duodenum:** Duodenitis, Gastric varices, Gastric carcinoma
hands, initial hemostasis can be achieved in over 90% of ulcers with active bleeding or visible vessels. A nonbleeding visible vessel (which is really a protruding clot overlying a rent in a nonprotruding vessel) indicates an early rebleeding rate of over 50%. It is not known whether therapeutic endoscopy will be a true substitute for operation because visible vessels seen in 45% of cases of ulcer hemorrhage, is present in 85% of deaths from that condition. Initial results from a prospective randomized study suggest that where endoscopic expertise is available, firmly adherent clots on ulcers should be forcibly removed and the underlying ulcer treated endoscopically. Nonmajor stigmata of recent hemorrhage such as flat/red black spots have the lowest risk of rebleeding (0%–3%) and endoscopic treatment is not necessary. Oral PPI will suffice. Many bleeding duodenal ulcers can be treated successfully with a combination of endoscopic therapy (such as adrenalin injection, bipolar diathermy, a heat probe, injection sclerotherapy with various sclerosants (alcohol, 1% polidocanol, 3% sodium tetradecyl sulfate, and 5% ethanolamine), hemoclips/endoclips and laser photocoagulation). Definitive hemostasis is higher with clipping (86.3%) than injection (75.4%) and clips significantly reduce bleeding (9.5%) compared with injection (19.5%). Clipping and thermocoagulation have comparable efficacy, and there is no difference in mortality between any intervention. The enhancing benefit of combination endoscopic treatment is superior to single-modality therapy, and combination treatment does not increase complications. The most recommended and popular method is a combination of endoscopic injection of at least 13 mL of 1:10,000 adrenaline which induces vasospasm, local tamponade, and platelet activation plus either a thermal (bipolar coagulation) or mechanical treatment. New endoscopic techniques include the use of fibrin glue or the simultaneous injection of thrombin and fibrinogen around the base of the ulcer and endoscopic ultrasonography-guided angiotherapy. Endoscopy and endotherapy are repeated within 24 hours when initial endoscopic treatment is considered suboptimal. Severe bleeding despite initial successful endoscopic therapy, are considered for either repeat endoscopic therapy, selective arterial embolization or surgery. About 20%–40% require repeat injections for rebleeding and 5%–28% require emergency surgery. The factors that predict failure of endoscopic treatment are hemodynamic instability, significant co-morbidity, > 4–6 U blood transfusion in 24 hours and the following endoscopic findings of the ulcer: actively bleeding vessel, visible vessel, adherent clot, and ulcer size > 2 cm. These predictors of failure are indications for surgery. The GI surgeon should be alerted of the possibility of surgery and should not be far away.

**Radiologic embolization**

Severe bleeding despite conservative medical treatment or endoscopic intervention occurs in 5%–10% of patients, and requires surgery or transcatheter arterial embolization. Surgery is associated with a mortality rate as high as 20%–40%. Indeed, patients who developed failed endoscopic hemostasis are likely to be poor surgical candidates with multiple co-morbidities. In the past decade, it has been considered in many institutions as the first-line intervention for massive bleeding from gastro-duodenal ulcer after failed endoscopic treatment. Radiologic embolisation is highly useful for patients with recurrent bleeding despite medical or surgical intervention, but local protocols and expertise vary greatly.

### Table 3

| Method                  | Delivery                      |
|-------------------------|-------------------------------|
| Thermally active methods| Electrocoagulation, bipolar, monopolar |
|                         | Heat probe, laser, heat probe |
| Injection               | Adrenaline 1:10 000, tissue glue |
| Mechanical methods      | Endoscopic clips, band ligation |
| Combination methods     | Injectable thermal material |

### Table 4

| Endoscopic Stigma         | Incidence (%) | Risk of Rebleeding (%) | Endoscopic Treatment |
|----------------------------|---------------|------------------------|----------------------|
| Active arterial bleeding   | 5–15          | 90–100                 | Yes                  |
| Visible vessel             | 25            | 50                     | Yes                  |
| Adherent clot              | 15            | 30                     | Yes                  |
| Flat/red black spots       | 15–20         | 5–10                   | No                   |
| No sigmata                 | 35            | 0–3                    | No                   |

### Table 5

| Predictors of failure of endoscopic treatment. |
|-----------------------------------------------|
| Hemodynamic instability                        |
| Significant comorbidity                        |
| > 4–6 U blood transfusion in 24 h              |
| Endoscopic findings of the ulcer               |
| Actively bleeding vessel                       |
| Visible vessel                                 |
| Adherent clot                                  |
| Ulcer size > 2 cm                              |
endovascular embolization is superior with high technical (95%) and clinical (72%) success rates. An important complication is the risk of significant ischemia which increases in patients with previous surgery within the same area. The disadvantage of radiologic embolization is that angiography will only detect bleeding vessels if bleeding occurs at a rate of >0.5 mL/min.

**Surgery for bleeding peptic ulcer**

Ten percent of patients still require operative treatment to arrest bleeding despite recent advances in medical, endoscopic, and interventional radio-embolization. Operative intervention is required when bleeding cannot be controlled successfully by endoscopic means or the patient is unstable during initial bleeding requiring a resuscitation laparotomy. The indications for surgery are summarized in Table 6. As patients over 60 years of age do not tolerate blood loss well the threshold to operate should be lower in these cases especially with shock or anemia on admission. The principles of surgery include an operation that is safest and quickest to arrest bleeding followed by treatment with PPI and Helicobacter pylori eradication therapy. General simple suture control of the bleeding combined with aggressive medical therapy usually proves sufficient. Historically, vagotomy and pyloroplasty with sutting of the bleeding ulcer was the operation of choice but the advent of the etiologic role of H. pylori, the role of nonsteroidal anti-inflammatory drugs (NSAIDs), and the development of PPI therapy have made these operations very rare. The special operative hazards would include (1) the risk of damaging the retroduodenal portion of the bile duct if sutures are hurriedly inserted to underrun a bleeding gastroduodenal artery. If a duodenal ulcer has caused much distortion of the tissues the supraduodenal portion of the bile duct is opened and a rubber Jacques catheter inserted to aid its identification. The duct is then closed over a “T” tube; (2) if no gastroduodenal cause for the bleeding is found, a complete small bowel laparotomy, and careful examination of the pancreas, gall bladder, bile duct and the aorta where it is crossed by the fourth part of the duodenum are necessary. If the site is still not apparent and the bleeding has stopped, the abdomen is closed. The complications of surgery would include (i) rebleeding, which may be from a gastric suture line or the regional bleeding site. If it persists despite IV PPIs and correction of any clotting abnormalities the patient would be reoperated, the anterior gastric suture line reopened, and hemostasis secured; (ii) the discovery of an unsuspected gastric cancer following partial gastrectomy. In a young fit patient, or if the excision margins appear to be involved with tumor, a more radical elective resection is considered; (iii) an incomplete vagotomy, if a definitive truncal vagotomy and drainage were performed in addition to under-running the ulcer. In this case, PPIs should be continued rather than considering another operation; (iv) leakage from a suture line or duodenal stump if a Billroth II gastrectomy (partial distal gastrectomy with gastrojejunal anastomosis) was performed.

**Follow-up postdischarge**

The prevention of recurrent bleeding is based on the etiology of the bleeding ulcer. H. pylori is eradicated with triple therapy including a PPI and 2 antimicrobial agents (amoxicillin 1 g or clarithromycin 500 mg or metronidazole 500 mg), all given bid for 7–14 days, and, after cure is documented anti-ulcer therapy is generally not given. Cure of H. pylori should be confirmed 4 weeks after treatment with C13 urea breath test for duodenal ulcer or repeat endoscopy for gastric ulcer and if there is suspicion of malignancy. NSAIDs are stopped but if they must be resumed low-dose COX-2-selective NSAID plus PPI is used. Patients with established cardiovascular disease who require aspirin should start PPI and generally reintroduce aspirin soon after bleeding ceases (within 7 d and ideally 1–3 d). Patients with idiopathic ulcers receive long-term antiulcer therapy. Because of the side effect of platelet dysfunction with the selective serotonin reuptake inhibitor (SSRI) antidepressant, it should be used with caution, or, ideally a non-SSRI may be an appropriate choice in patients who have increased risk of GI bleeding especially in patients taking NSAID or aspirin.

**Esophageal varices**

Ninety percent of variceal bleeding occurs within 2 cm of the gastro-esophageal junction, the site of the portosystemic venous collaterals. Evidence of a recent bleed is seen by finding a transparent fibrous clot on the surface of varix. Long-term survival is dependent on the severity of liver disease (Child’s classification). The therapeutic armamentarium includes, intravariceal sclerotherapy or endoscopic band ligation (for acute bleeding control and future prophylaxis which is effective in most patients); vasopressin/somatostatin (lower portal pressure in the acute situation); Sengstaken-Blakemore tube (balloon tamponading of the varices as a temporizing maneuver before definitive sclerotherapy); portal/systemic shunts (decrease portal pressure but the progressive increase in hepatic encephalopathy would render its consideration only after 2 failed sessions of sclerotherapy); and the transjugular intrahepatic portosystemic shunt under radiologic control to lower portal venous pressure. Numerous operations have been described for portal hypertension when bleeding has not responded to medical treatment, but it is important to remember that some of these operations may impede a later liver transplant. Esophageal transection of the esophageal varices at the gastro-esophageal junction and reanastomosis with a stapler via a small gastrostomy is a relatively simple operation, but it can be difficult in those patients who have had chronic injection sclerotherapy as there may be very thickened and hemorrhagic para-esophageal tissue and a friable esophagus. It is contraindicated when there is bleeding from esophageal ulceration following injection sclerotherapy.

**Acute erosive gastritis**

Acute erosive gastritis must be distinguished from the chronic forms of gastritis as these do not bleed. Hemorrhagic gastritis is
often caused by stressful stimuli such as head injury (Cushing ulcer) from increased vagal stimulation with resulting acid hypersecretion, burns (Curling ulcer), shock, or hepatic failure from probably impaired mucosal blood flow. Drugs may also be responsible and the agents which are commonly implicated include steroids, NSAIDs and alcohol. Hemorrhage from NSAIDs injury can be arrested with a platelet transfusion but pharmacological suppression of acid secretion with IV PPIs or the use of sucralfate is the mainstay of therapy. A total gastrectomy is rarely indicated if the site of bleeding is unclear, and it is important to reduce gastric blood flow as quickly as possible. The first steps are to ligate and divide the right gastric and gastro-epiploic vessels, divide the duodenum, lift up the stomach and ligate and divide the left gastric vessels. The rest of the operation is done at relative leisure[44,56].

M-W tear

The M-W tear occurs in the region of the gastro-esophageal junction on the lesser curve in 80% of cases, as a result of severe vomiting or retching, often after excessive alcohol intake. The tear is mostly on the gastric mucosa but may extend into the esophagus. Very occasionally repeated vomiting may result in full-thickness tear, “Boerhave syndrome” associated with sudden onset severe upper abdominal or chest pain. Although bleeding is profuse with M-W, it usually stops spontaneously in 90% of cases. Emergency endoscopy establishes the diagnosis and conservative management as with erosive gastritis. Endoscopic electrocoagulation or operative underrunning is indicated in resistant cases[56].

Aortoenteric fistula

Most aortoenteric fistulas are secondary to prior aortic Dacron graft surgery and most always involve the third part of the duodenum[57]. Although a “primary” aortoenteric fistula can also occur[58], the classical presentation is a “ herald” bleed that occurs and stops spontaneously hours or occasionally weeks before the major hemorrhage. A high index of suspicion is necessary and the vascular surgeon consulted after upper GI endoscopy has excluded other causes of bleeding. The endoscopist should attempt to reach the third part of the duodenum to visualize the fistula[59]. An abdominal CT may also demonstrate the fistula. Surgery is required to repair the fistula. This entails graft removal with either extra-anatomic bypass or in situ replacement with a rifampicin-bonded graft[57,59,60], or a staged endovascular repair without graft removal[60,61].

Gastric leiomyoma

This is the commonest tumor to cause a major upper GI bleed. Leiomyomas are the most common benign mesenchymal tumors of the upper GI tract and rarely cause symptoms when smaller than 5 cm in diameter. They are recognized by endoscopy as a yellowish polyp with an ulcer crater on the surface. Treatment by local excision is curative, but endoscopic local ethanol injection may be the treatment of choice in carefully selected patients with hemorrhagic leiomyomas[62]. Gastric carcinomas, in contrast, ooze slowly causing anemia[63].

| Causes of lower gastrointestinal hemorrhage in different age groups. |
|---------------------------------------------------------------|
| **Children** | **Adults** | **Elderly People** |
| Meckel's diverticulum | Inflammatory bowel disease | Diverticular disease |
| Juvenile polyp | Adenomatous polyps | Angiodysplasia |
| Inflammatory bowel disease | Carcinoma | |
| | Arteriovenous malformations | |
| | Small intestinal neoplasia | |
| | Hereditary telangiectasia | |
| | Infective colitis | |
| | Hemorrhoids | |
| | Solitary rectal ulcer | |
| | Anal fissure | |

Acute lower GI bleeding

The lower GI tract is less commonly a source of major hemorrhage than the stomach or duodenum[63]. Patients with recurrent or profuse bleeding pose a diagnostic dilemma as the source of their hemorrhage is notoriously difficult to identify. Ninety percent of them have either colonic angiodysplasia or diverticular disease (Table 7). Profuse diarrhea with rectal bleeding and mucus passage may suggest inflammatory bowel diseases such as ulcerative colitis or Crohn’s disease. There may be a history of previous rectal bleeding episodes in patients suffering from hemorrhoids or ulcerative colitis. Infective conditions such as shigella, amebic dysentery, or schistosomiasis can cause rectal bleeding associated with diarrhea are confirmed by stool culture. Acute hypovolaemic shock may cause sloughing of the intestinal (usually colonic) mucosa resulting in blood per rectum. Clinical examination is usually unremarkable in terms of localizing the source of the bleed. This is apart from the occasional torrential upper GI hemorrhage from a bleeding duodenal ulcer that may be brisk enough to cause the passage of unaltered blood per rectum, thus mimicking a colonic bleed but with signs of hemorrhagic shock[64].

Diverticular disease

Diverticular disease was traditionally considered to be the most common cause of major lower GI hemorrhage. Bleeding occurs when a blood vessel breaks down, as it passes through the wall of a diverticulum in a submucous plane. Diverticular disease is a common finding on barium enema examination and for this reason, has been implicated as the source of bleeding in the absence of any other discernible abnormality. Many cases of lower GI bleeding that would previously have been attributed to diverticular disease are now recognized with colonoscopy due to colonic angiodysplasia[64,65].

Angiodysplasia

Congenital angiodysplasia are acquired lesions that predominantly affect elderly people. They are small vascular swellings, usually <5 mm in diameter, comprising dilated venules located immediately beneath the mucosa. They can be shown by colonoscopy and arteriography but can easily be overlooked. Most lesions are located in the right colon and they are often multiple. Colonic angiodysplasia often coexists with other abnormalities that have hemorrhagic potential such as a Meckel’s
diverticulum, which may cause confusion in determining the source of bleeding. Many patients also have associated cardiovascular disorders.

**Management of major lower GI hemorrhage**

Generally, bleeding from the lower GI tract ceases spontaneously, and thus most cases are managed conservatively by blood transfusion and close observation of vital signs (Table 8). Proctosigmoidoscopy should be performed although it may be difficult during the bleeding as blood may obscure the field of view. Occasionally, one can get above the bleeding point suggesting that the origin of the bleed is distal to that site. Proctocolitis may be evident and biopsies confirm the diagnosis. It is rare for colonic carcinoma to result in major acute hemorrhage, though polyps may do so. Colonoscopy should be requested if proctosigmoidoscopy could not account for the blood loss. If bleeding continues, a radio-labelled red cell scan should be undertaken and the site of bleeding will be identified as a “hot spot” on the scan. If this test fails to demonstrate the source or frank bleeding continues, selective mesenteric angiography should be performed. Apart from detecting angiodysplasia the site of bleeding is accurately located if blood loss is at least 1 mL/min and surgery may be confidently undertaken.

Should bleeding be copious and with no available diagnosis, “blind” emergency surgery may be necessary. The bleeding source is rarely evident at laparotomy, and the patient may undergo a right hemicolectomy on the suspicion of cecal angiodysplasia or a sigmoid colectomy on the suspicion of diverticular bleeding or a subtotal colectomy if no clues at all are encountered intraoperatively. On-table colonoscopy should decrease the “blind” resection rate. Unfortunately, a proportion of those who undergo a “blind” resection continues to bleed. Thus referral to a specialist center for visceral angiography is preferable to a “blind” laparotomy.

**Conclusions**

The upper GI tract is more commonly a source of acute hemorrhage mostly from the bleeding complication of peptic ulcer disease, aspirin, and NSAIDs ingestion than the lower GI tract. As most GI hemorrhage cease spontaneously, there is greater importance in simultaneous resuscitation and nonoperative intervention. The definitive management of upper GI hemorrhage is indicated by the overall risk of rebleeding and morbidity (Rockaall risk score). GI hemorrhage is potentially life-threatening and failure to respond to endoscopic and medical management (ie, recurrent or continuing bleeding) is an indication for urgent surgery. Radio-embolization is a growing useful adjunct in the available armamentarium for nonoperative management of upper GI bleeding. Although new endoscopic techniques and interventional radiology have decreased the role of surgery in management, the collaboration between the endoscopist and surgeon still remains. Any trial of new therapeutic techniques to stop acute GI hemorrhage must improve on the natural hemostatic process, to demonstrate any benefit.

**Ethical approval**

No ethical approval required.

**Sources of funding**

No specific funding was required for this study.

**Author’s contribution**

E.P.W. is the sole author.

**Conflict of interest**

The authors declare that they have no financial conflict of interest with regard to the content of this report.

**Research registration unique identifying number (UIN)**

It is a retrospective research review study.

**Guarantor**

Marcelin Ngowe Ngowe act as guarantor.

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