Management of massive gastrointestinal haemorrhage

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Abstract

Gastrointestinal haemorrhage (GI bleeding) is a common medical emergency, with one patient presenting every 6 minutes in the UK, or 85,000 cases per annum. It is associated with a significant mortality rate that has remained relatively static at 10% for more than two decades. Haemorrhage is commonly categorized as upper or lower gastrointestinal in origin, but for organization of care, both groups should be regarded as one clinical entity. Rapid assessment, resuscitation and correction of coagulopathy should be undertaken, and investigation or definitive management urgently arranged. For upper GI haemorrhage, endoscopy remains the cornerstone of investigation and treatment. In lower GI haemorrhage, a more nuanced algorithm utilizing CT angiography and endoscopic evaluation is recommended. Clinicians may utilize a range of treatment modalities including endoscopic and interventional techniques to diagnose and control the source of haemorrhage, which should be tailored to the site of bleeding and pathology. Where control is not achieved the clinician should consider either repeat intervention, use of alternative haemostatic techniques or a different modality to achieve haemostasis. Surgery is rarely used as a treatment and should only be undertaken where all other measures to control haemorrhage have failed.

Keywords Emergency surgery; endoscopy; gastrointestinal haemorrhage; haematemesis; haematochezia; interventional radiology; lower GI bleed; management; melaena; upper GI bleed

Introduction

Gastrointestinal (GI) haemorrhage is a common medical emergency, with an incidence of 134 per 100,000 population in the UK. This is equal to one patient presenting every 6 minutes, or 85,000 cases per annum.[1] GI haemorrhage (GI bleeding) can originate from anywhere along the length of the gastrointestinal tract from mouth to anus, or per stoma in operated patients. Haemorrhage is classified as upper or lower gastrointestinal in origin.

- Upper GI haemorrhage – occurring proximal to the ligament of Treitz (a band of smooth muscle which extends from the duodenojejunal flexure to the left crus of the diaphragm). In practice this means bleeding from the oesophagus, stomach, or duodenum, which can be accessed with a standard fibre-optic endoscope. Upper GI haemorrhage is subclassified as non-variceal (89%) or variceal (associated with liver disease) (11%). Overall mortality is approximately 10% and has remained relatively unchanged since the 1990s.[1,2]
- Lower GI haemorrhage – occurring distal to the ligament of Treitz, including bleeding of jejunal, ileal, colonic, rectal or anal origin. Lower GI haemorrhage is three times less common than upper GI haemorrhage, but in-hospital mortality is as high as 3.4%.[3]

Classifying haemorrhage as upper or lower gastrointestinal in origin can aid diagnosis and management, but guidelines suggest that for delivery of care, both groups should be regarded as one clinical entity and clinical governance structured as such. Studies have quantified massive (or major) gastrointestinal haemorrhage as requiring transfusion of at least four units of packed red blood cells.[1] The NHS transfusion service define it as loss of one blood volume in 24 hours (70 ml/kg), 50% of total blood volume within 3 hours, or blood loss more than 150 ml/minute. A clinical aide includes systolic blood pressure less than 90 mmHg or heart rate more than 110 beats per minute.[4,5] Landmark UK publications in the last decade include the 2015 NCEPOD Massive GI Haemorrhage report, and the 2018 UK Lower GI Bleeding Collaborative audit.[1,3,6]

In 2015 NCEPOD reported a combined overall mortality of 10.4% for upper and lower GI haemorrhage, not stratified by severity of bleeding. However, the study method was structured to assess quality of care and therefore is at risk of reporting bias.[1] Both the NCEPOD report and the Lower GI Bleeding Collaborative found that mortality was associated with three factors. Requirement for at least four units of red cell transfusion (i.e. massive gastrointestinal bleeding) doubled overall mortality to 24% in the NCEPOD study (non-variceal upper GI bleeding 21%, lower GI bleeding 20%). Oakland et al. also reported the same 20% mortality in lower gastrointestinal bleeding requiring four or more units transfusion, and gastrointestinal bleeding is the second most common diagnosis resulting in blood transfusion (after haematological malignancy), accounting for 14% of all transfusions. Secondly, mortality was closely associated with degree of shock. Thirdly, onset of gastrointestinal bleeding in patients already admitted to hospital was associated with a mortality rate of 18% in the Oakland et al. study (regardless of transfusion) and 37.7% in the NCEPOD study. Comorbidity and lack of fitness for treatment appear to contribute significantly to risk of mortality following onset of severe gastrointestinal haemorrhage, as 79% of the mortalities reported by NCEPOD were in patients on a palliative care pathway at time of death.[1,6]

Presentation

The signs and symptoms of gastrointestinal haemorrhage are dependent on the exact source of the bleeding. Upper GI bleeding

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typically presents with symptoms of melaena (black, malodorous faeces caused by altered haemoglobin), or haematemesis (coffee ground—appearing blood-stained vomitus, caused by blood interacting with gastric acid). Lower GI bleeding presents with haematochezia – fresh red rectal bleeding or passage of clots per rectum. Less overt or asymptomatic signs of both upper and lower gastrointestinal bleeding include reduced haemoglobin or iron deficiency anaemia, or an abnormal faecal immunochemical test (FIT). Often the clinical picture can be mixed; brisk upper GI bleeding presenting with haematochezia can lead to the misdiagnosis of lower gastrointestinal bleeding in as many as 15% of patients; this can lead to a potentially fatal delay in commencing appropriate management. Conversely, patients with bleeding from the caecum or distal small bowel may present with melaena.[6,7]

It is important where possible to obtain an accurate and thorough history from the patient or a collateral history from a relative or carer. Focused questioning of the acutely unwell patient should consider character, frequency and volume of blood loss, associated symptoms such as pain, nausea, vomiting, change in bowel habit and indicators of sepsis such as fever. A complete past medical history must be obtained, with focus on comorbidities that indicate an increased risk of bleeding or identify the cause. For upper GI haemorrhage, this should include liver disease and presence of varices, alcohol abuse, peptic or duodenal ulcer or reflux disease and any known vascular abnormalities such as angiodysplasia. Symptoms of undiagnosed upper gastrointestinal malignancy, such as dysphagia and weight loss, must be considered. A history of acute excess vomiting of any cause must alert the clinician to the risk of a Mallory-Weiss tear. In lower GI haemorrhage, the commonest cause in the UK is diverticular disease, followed by anorectal conditions such as haemorrhoids. Symptoms of colorectal malignancy or inflammatory bowel disease, such as recent change in bowel habit, must be considered. Recent endoscopic instrumentation of the gastrointestinal tract could suggest an iatrogenic cause of bleeding.

It important to ascertain whether the patient is taking medication or undergoing treatment which may damage the gastrointestinal mucosa causing ulceration or predispose to bleeding; this includes non-steroidal anti-inflammatory drugs (NSAIDs), oral steroids, or radiotherapy. Up to a third of patients presenting with GI haemorrhage in the UK take a form of antiplatelet drug.[1] It is therefore critical to identify if the patient is taking medication which increases the risk of bleeding; this includes antiplatelets and anticoagulation such as aspirin, warfarin and direct oral anticoagulants (DOACs). Patients already admitted to hospital may be prescribed heparin as prophylaxis or treatment for thromboembolism.

It is essential to perform a full gastrointestinal examination of the patient. Be aware of any signs which may help to localize the source of the bleeding, such as the presence of an abdominal mass. Examine for the presence of stigmata of chronic liver disease and for the signs of malignancy (e.g. palpable lymphadenopathy). You must perform a digital rectal examination (which allows accurate and objective assessment of melaena versus haematochezia) and if tolerated by the patient, consider proctoscopy to identify an anorectal cause.

Remember that while the source of bleeding may be inferred by symptoms and signs, it should never be assumed. National guidance suggests patients should progress to prompt formal investigation in order to confirm diagnosis.[1,8] Finally, as patients already admitted to hospital are at increased risk of mortality, concerns regarding blood loss raised by nursing or allied healthcare staff should be assessed without delay.

Initial management

Initial management of haemorrhage is common to any source and involves standard resuscitative measures. Assessment of the patient's airway and respiratory system is performed initially, with attention made to ensure adequacy of ventilation. Reduced conscious level and/or aspiration of either blood or gastric contents can result in airway obstruction. Oxygen saturation and respiratory rate are recorded and, in the event of inadequate ventilation, simple airway manoeuvres (head tilt, chin lift and jaw thrust) and adjuncts (oropharyngeal or nasopharyngeal airway) employed. The patient's heart rate and blood pressure are recorded and wide bore peripheral venous access obtained (at least two 16−18-gauge intravenous cannulae). In the event of compromised airway or inadequate ventilation and perfusion, and the inability to secure intravenous access or progress resuscitation attempts, seek immediate support via a priority or arrest team call. All hospitals in the UK have a major haemorrhage protocol which care-providers should be familiar with and able to activate when necessary. Failure to respond to initial resuscitation, as judged by prompt and ongoing observation using a system such as the National Early Warning Score (NEWS), should mandate referral to a higher level of care.[8]

Blood tests including haemoglobin (Hb), haematocrit, urea, creatinine, electrolytes, liver function, coagulation profile (international normalized ratio (INR), prothrombin time (PT), activated partial thromboplastin time (APTT) and fibrinogen) are performed and cross-matched blood obtained after discussion with the transfusion laboratory. When available, thromboelastometry (TEM, previously known as ROTEM) can provide rapid assessment of coagulation status.

An elevated serum urea may be observed in the case of upper GI haemorrhage as blood bound protein is metabolized to blood urea nitrogen (BUN) and then reabsorbed. Raised serum urea has traditionally been used to differentiate upper and lower GI blood loss.

Measurement of lactate from either arterial or venous blood gas allows for a prompt assessment of tissue perfusion and the patient's blood volume status and has been demonstrated to be a sensitive predictor of mortality.[9]

Previously healthy and young patients have robust compensatory mechanisms to ensure adequate circulatory pressure. Increases in myocardial contractility, heart rate and peripheral vasoconstriction maintain circulatory pressures at near normal levels and therefore a falling blood pressure is considered a late sign and should not be relied upon to guide assessment of severity of shock.

The ACS/ATLS guidelines on haemorrhage severity and class of hypovolaemic (haemorrhagic) shock is a useful tool in the estimation of blood loss in patients with significant gastrointestinal bleeding (Table 1).

A prompt fluid bolus of 500 ml crystalloid is recommended for initial volume replacement. In the event of major haemorrhage, judicious use of blood products will be required, but over-transfusion avoided due to the risk of circulatory overload and

Classification of Shock

Class of haemorrhagic shock

	I	II	III	IV
Blood loss (mL)	Up to 750	750–1500	1500–2000	>2000
Blood loss (% blood volume)	Up to 15	15–30	30–40	>40
Pulse rate (per minute)	<100	100–120	120–140	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mm Hg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate (per minute)	14–20	20–30	30–40	>35
Urine output (rnL/hour)	>30	20–30	5–15	Negligible
Central nervous system/mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

From 'ATLS – Advanced trauma life support. (2012). Chicago, Ill.: American College of Surgeons, Committee on Trauma'

Table 1

transfusion reaction. It may be prudent to adopt a policy of 'permissive hypotension' (maintaining blood pressure at a level required to maintain tissue perfusion and cognition) until definitive control of the source of bleeding can be established.

Hospitals will have a transfusion policy where, for stable patients, packed red blood cell transfusion is recommended below a threshold (typically Hb <70 g/L). However, in the event of major haemorrhage haemoglobin may not fall immediately as the patient loses both red cells and plasma volume – repeating the test within a few hours will demonstrate a delayed drop. When used, NICE guidelines recommend a transfusion target of Hb 80 g/L in patients with cardiovascular disease and 70 g/L in those without.[10] Meta-analysis shows restrictive transfusion is associated with improved outcomes in patients without cardiovascular disease but results are less clear and may be harmful in those with coronary artery disease, stroke or peripheral vascular disease.[11]

Activation of a major haemorrhage protocol triggers the rapid and continuous issue of red cells and other blood products from the blood bank in pre-agreed ratios – usually 1:1 packed red cells: fresh frozen plasma (FFP), with platelets and cryoprecipitate as directed by laboratory results. Key staff members in haematology, theatres and intensive care are therefore alerted when the protocol is activated. There is little published evidence to suggest specific platelet or FFP target levels, but several guidelines recommend patients who are actively bleeding and have a platelet count $<50 \times 10^9$ L are given platelets.[6,10] Use of FFP and cryoprecipitate (a more concentrated source of fibrinogen) will depend on the coagulation tests, and both products require liaison with a haematologist before use.

For all patients presenting with gastrointestinal bleeding, it must be remembered that a normal haemoglobin and systolic blood pressure does not exclude a life-threatening haemorrhage, that if left unrecognized, may manifest with sudden circulatory collapse.

Coagulopathy and clotting

Use of anticoagulants and antiplatelets has become commonplace and are often used for either primary or secondary thromboembolic prevention. Their widespread use has inevitably resulted in increased risk of bleeding and presents an extra challenge in the management of iatrogenic coagulopathy in the setting of acute haemorrhage. In the setting of acute GI bleeding anticoagulants and antiplatelets are often withheld whilst the primary haemorrhage is addressed, although there is some evidence of long-term cardiovascular harm resulting from this practice.[6] The following summary applies to both upper and lower GI bleeding, but exact management will vary between patients and even institutions.

In those taking warfarin and other vitamin K antagonists who experience life-threatening haemorrhage, warfarin is reversed with intravenous vitamin K and prothrombin complex concentrate (PCC) (Beriplex®). This contains human coagulation factors II, VII, IX, X and endogenous inhibitor proteins S and C – it is considered preferable to fresh frozen plasma (FFP) due to ease of administration, rapid normalization of INR and low risk of volume overload.[12] Guidance suggests restarting warfarin seven days after bleeding minimizes both rebleeding risk and thromboembolic events.[6]

In patients with high thrombotic risk (such as prosthetic mitral valve, atrial fibrillation with prosthetic heart valve or mitral stenosis, or venous thromboembolism within the last 3 months) low molecular weight heparin (LMWH) can be considered at 48 hours if the patient is stable with normalized coagulation. When a patient has a massive haemorrhage whilst prescribed low molecular weight heparin (LMWH) or unfractionated heparin (UFH), protamine is used as a selective antidote to its anticoagulant mechanism of action.

There is also lack of evidence for stopping antiplatelets to reduce risk of rebleeding. When used for secondary prevention only, Oakland et al. suggest continuing aspirin, but when used for primary prevention, aspirin could be stopped indefinitely. Liaison with cardiology will be required for patients taking dual antiplatelet therapy for coronary stents (commonly a P2Y12 antagonist such as clopidogrel, in addition to aspirin). If considered high risk for thromboembolic events (within 12 months of drug eluting coronary stents, or within one month of bare metal stents) dual-antiplatelet therapy or, at the least, aspirin alone may have to be continued. Restarting of drug therapy should be prompt but there is little evidence on timing. It must be remembered that discontinuing dual antiplatelet therapy in the at-risk period confers up to 40% risk of acute myocardial infarction or death.[13]

Direct oral anticoagulants have a short half-life unless there is concomitant renal failure. Dabigatran inhibits thrombin, whereas most others inhibit factor Xa. Life-threatening bleeding will

require liaison with haematology as reversal is complex and drug dependent. Idarucizumab (Praxbind®) is available for the reversal of dabigatran, and more recently andexanet alfa (Ondexxya®) has been licensed for the reversal of apixaban and rivaroxaban in patients with life-threatening bleeding. If the specific reversal agents are not available, PCC can also be used, but always after liaison with a haematologist.

Resuming anticoagulation or substituting for warfarin at seven days is recommended.[6]

Tranexamic acid has traditionally been given to patients presenting with gastrointestinal haemorrhage. However, the landmark HALT-IT trial found no difference between tranexamic acid infusion and placebo for mortality, blood transfusion and rebleeding. With only a very small increase in venous thromboembolism, most guidelines no longer recommend tranexamic acid for GI bleeding.[14]

Use of reversal agents and prothrombin complexes are used in liaison with haematology, and for patients being treated with anticoagulant or antiplatelet agents for a medical comorbidity, specialist cardiac or stroke advice is also essential, both when considering the stopping of the medications or upon resuming them.

Upper GI haemorrhage

Upper GI bleeding is the more common gastrointestinal haemorrhage, with an incidence of more than 100 per 100,000 population. A medical emergency, an overall mortality rate of 10% has persisted since the first high-quality studies of upper GI bleeding in the UK, by the senior author of this article, in 1995.[2] In the first audit mortality was 14% and has reduced slightly since then, remaining static at 10% since 2003.[15] Patients are typically admitted under the medical team that provide emergency upper GI oesophagogastroduodenoscopy (OGD, or more simply, endoscopy), as this is the diagnostic and treatment modality of choice, but as previously outlined, it is not recommended that overall leadership and oversight of care for patients with gastrointestinal bleeding is rigidly divided. Upper GI haemorrhage differs to lower GI bleeding in that prompt upper gastrointestinal endoscopy is essential and effective in more than 95% of cases.[1]

Pathology

Peptic ulcers: Peptic ulcer disease is the most common cause for upper GI bleeding and accounts for approximately 31%–67% of presentations.[16]

Ulceration beyond the mucosa into the submucosa results in inflammation, weakening and necrosis in arterial walls, leading to pseudoaneurysm formation, rupture and haemorrhage.

Up to 90% of duodenal ulcers and 70% of gastric ulcers are associated with infection of *Helicobacter pylori*. This Gram-negative bacterium causes disruption of the mucosal barrier resulting in inflammation and ulceration of the gastric and duodenal mucosa. Incidence of peptic ulcer disease has declined significantly since the identification of *H. pylori* and the widespread use of proton pump inhibitors (PPI).[17,18]

NSAIDs are also associated with peptic ulcer disease, by inhibiting cyclooxygenase and decreasing mucosal prostaglandin synthesis. This results in impaired mucosal defence, and a 40-fold increased risk of gastric ulceration and 8-fold increased risk of duodenal ulceration. Up to 20% of long-term NSAID users will have mucosal ulceration. For this reason, NICE recommends that all NSAIDs are stopped during acute haemorrhage. The decision to restart NSAIDs after GI haemorrhage should be considered carefully on a case-by-case basis. If the benefit of treatment appears to outweigh the potential risk of further bleeding, then a prophylactic PPI should be prescribed concurrently, which reduces the risk of new peptic ulcer formation by 50%–80%.[10,17]

Benign peptic ulcers are best assessed endoscopically where they are typically described as having smooth, rounded edges. The Forrest classification (Table 2) categorizes ulcers into three classes, which helps guide management and risk-stratifies those patients at high risk of rebleeding and mortality. Any ulcer other than a 2c or 3 is considered high risk.[19]

Gastritis, duodenitis and oesophagitis: Stress gastritis is commonly seen in critically unwell inpatients and typically results from disruption to mucosal defences (ordinarily, maintained by mucus, bicarbonate and prostaglandins protecting the gastric mucosa from the acidic intra-luminal environment). NICE recommends routine use of PPI for prophylaxis in critically ill patients admitted to ITU to prevent gastrointestinal haemorrhage.[10,18]

Patients at risk of oesophagitis tend to have a history of gastro-oesophageal reflux disease and a hiatus hernia may be present. The stratified squamous epithelium that lines the oesophagus lacks many of the mucosal defences that protect against the caustic effects of gastric acid. Increased acid exposure

Forrest Classification

The Forrest classification

Class	Description	Endoscopic Intervention	Rebleeding Rate
1A	Active spurting	Yes	55%
1B	Active oozing	Yes	55%
2A	Nonbleeding visible vessel	Yes	43%
2B	Adherent clot	Consider	22%
2C	Flat pigmented spot	No	10%
3	Clean ulcer base	No	5%

Adapted from: Nelms, D. W. & Pelaez, C. The Acute Upper Gastrointestinal Bleed. Surg Clin. NA 98, 1047–1057 (2018)

Table 2

and reflux of gastric contents into the distal oesophagus results in inflammation and erosion which can result in haemorrhage.

Mallory-Weiss tears are longitudinal lacerations in the gastric cardia or at the gastro-oesophageal junction. They account for 4%−8% of upper GI bleeds and occur as a result of a sudden increase in intragastric pressure, for example, during hyperemesis of pregnancy, or after vomiting following alcohol intake or food poisoning.

Spontaneous resolution of bleeding is common, and intervention is only required in 10% of cases. Lesions not actively bleeding can be managed with PPI and anti-emetics alone; rebleeding from these tears is rare (7%).[7,18]

Tumours/malignancy: Tumours of the upper GI tract rarely present with acute haemorrhage and only form approximately 4%−8% of acute upper GI bleeds. Often tumours are asymptomatic until a late stage, therefore at presentation the disease is often advanced. Nevertheless, if ulcers and lesions appear suspicious (elevated, irregular borders with associated abnormal mucosal folds), biopsy should be undertaken, as approximately 6% of gastric ulcers contain underlying malignancy. It is also necessary to repeat the endoscopy after 6 weeks to ensure healing − a non-healing ulcer is suspicious for an underlying malignant process. Duodenal ulcers are rarely malignant and as such routine biopsies are not always recommended.

Dieulafoy lesions/vascular ectasia: Dieulafoy lesions are a rare cause of upper GI haemorrhage. These are large but histologically normal arterioles which protrude through the submucosa and mucosa and can occur anywhere in the GI tract, but typically are found on the lesser curve of the stomach and within 6 cm of the gastro-oesophageal junction. Exposure to the acidic intraluminal environment can result in necrosis and rupture of the affected arteriole resulting in sudden, brisk bleeding in a patient with no other significant symptoms or risk factors.[18,19]

Rare causes: Other rare causes of upper GI bleeding include aortoenteric fistulae and haemobilia. Aortoenteric fistulae occur following surgical intervention to the aorta or GI tract. Often there is a 'herald bleed' followed by massive exsanguinous haemorrhage. An urgent radiological investigation should be undertaken in patients with suspected aortoenteric fistula and immediate lifesaving reconstructive surgery is mandatory, with extra-anatomic bypass reconstruction, removal of any infected synthetic material (e.g. aortic graft) and closure of any enterotomy. Perioperative mortality is inevitably extremely high.[7] This rare diagnosis must be considered, especially for patients with negative endoscopic findings but prior history of vascular surgery.

Haemobilia can result from instrumentation of the biliary system (e.g. endoscopic retrograde cholangiopancreatography (ERCP)), trauma, or bleeding into the pancreatic ducts as a complication of pancreatitis (hemosuccus pancreaticus). Endoscopic control of bleeding from the biliary system is difficult to establish, therefore interventional angiography is the most appropriate management.[20]

Staple line bleeding following gastric surgery is a recognized complication of procedures such as laparoscopic sleeve gastrectomy for obesity. This may present with haematemesis or signs of intra-abdominal haemorrhage. Similarly, intragastric balloon placement may cause Mallory-Weiss like bleeding or ulceration requiring urgent endoscopic removal of the balloon. In such cases, resuscitation followed by discussion with a bariatric surgical service is essential. Once stable, the patient usually will require transfer to allow definitive treatment in an NHS centre that is able to provide appropriate anaesthetic and high dependency care for bariatric patients.

Variceal bleeding: Accounting for approximately 4%−20% of upper GI bleeds, varices are abnormally dilated veins which occur as a result of portal hypertension and development of portosystemic shunts, commonly found in the distal oesophagus and upper stomach. Most cases are secondary to cirrhosis, but rarely varices may be caused by non-cirrhotic portal hypertension (veno-occlusive disease) or portal vein thrombosis. Gastric varices are further subdivided into gastro-oesophageal varices and isolated gastric varices depending upon their anatomical location.[10,21]

The management of patients who have suspected variceal bleeding differs from those with non-variceal bleeding, reflecting the different pathology. There is a high risk of mortality; 20% within six weeks following first presentation of variceal bleeding.

On presentation, patients with suspected variceal bleeding should be commenced on a splanchnic vasoconstrictor which is continued until definitive haemostasis is achieved or until after 5 days following presentation. NICE guidelines currently recommend terlipressin, however some authors recommend the somatastatin analogue octreotide which is licenced in North America.[16,22]

Patients with variceal bleeds are at high risk of bacterial infection and antibiotics have been shown to reduce rebleeding, infection and mortality, therefore prophylactic antibiotics are mandatory. Broad-spectrum antibiotics (such as a quinolone, cephalosporin or piperacillin-tazobactam) are most appropriate, although local guidelines should be consulted.[21]

Pre-endoscopic care

Timing of endoscopy: Patients who are haemodynamically unstable and with evidence of active bleeding should undergo immediate endoscopy after initial resuscitative measures. All patients requiring admission should receive endoscopy within 24 hours. UK hospitals should have access to 24-hour endoscopy services and an on-call endoscopy team.[1]

There is an association between endoscopy performed more than 24 hours after admission and increased risk of mortality, evidencing the need for early intervention. Even after a period of stabilization, if the patient further deteriorates, immediate repeat intervention is necessary. Correction of abnormal coagulation should not delay endoscopy if bleeding is life threatening.[5,8,17]

While most endoscopy is performed in a dedicated department, emergency or out-of-hours endoscopy is commonly performed either in the emergency operating theatre or even bedside in intensive care. The management plan formulated by the endoscopist needs communicating to the clinical team responsible for continuity of care and written or verbal communication must be promptly reviewed upon the patient's return to the bed space.

Pre-endoscopic nasogastric drainage tube insertion is no longer considered to offer any benefit and should be avoided.[8]

Risk stratification: Given the associated morbidity and mortality associated with acute upper GI haemorrhage and reported rebleed rate of approximately 5%–20%, even after successful endoscopic intervention, it is essential to risk stratify patients to identify those at high risk of serious adverse events. This allows prediction of further endoscopic intervention and aids triage of patients.

Current NICE guidelines advocate a two-step risk assessment for the assessment of acute upper GI haemorrhage.[10]

Prior to endoscopy and within 24 hours of admission, the Glasgow-Blatchford score (Table 3) is used to risk-stratify patients. Low risk patients (Blatchford score 0) may be appropriately discharged with suitable further outpatient investigation. The higher the Blatchford score, the higher the risk of adverse clinical outcome and the more urgently endoscopy should be considered.

After endoscopy, NICE recommends use of the full Rockall scoring system (Table 4) to predict risk of rebleeding or death following endoscopic intervention; a score of more than 2 indicates increased risk.

All patients with GI haemorrhage should have an agreed rebleed plan which should be based on individual risk and pathology. This second stage risk stratification helps clinical teams anticipate further treatment and intervention.

Proton Pump Inhibitors (PPIs) act by irreversibly blocking that H^+/K^+ gastric proton pump in gastric parietal cells, preventing the luminal secretion of H^+ ions, and reducing up to 99% of gastric acid production.

The use of PPI in acute upper GI haemorrhage prior to endoscopy remains a controversial topic. Most guidelines agree that following an endoscopic diagnosis of ulcerative disease with high-risk features, high dose PPI is recommended. The European Society of Gastrointestinal Endoscopy (ESGE) recommends intravenous high dose proton pump inhibitor (omeprazole 80 mg) be given as a bolus on presentation followed by continuous infusion (omeprazole 8 mg/hr) for all patients requiring admission.

This recommendation is refuted by NICE who advise against offering PPI prior to emergency endoscopy, citing lack of evidence that PPIs reduce rebleeding rate or mortality, may downgrade underlying pathology and delay definitive endoscopic management.

A recent NCEPOD audit found that 73% of patients with acute bleeding received PPI contradictory from guidance, indicating a clear disparity between national standards and common practice. Following endoscopy NICE do recommend commencing PPI in those with stigmata of recent haemorrhage.[8,10,18]

Prokinetics: ESGE recommends intravenous erythromycin (250 mg) 30–120 minutes prior to gastroscopy to improve mucosal visualization, by accelerating gastric emptying. There is no evidence to support the use of metoclopramide.[17,19]

Endoscopic management

Endoscopic Therapy – Non variceal UGIH: in the case of non-variceal upper GI bleeding, NICE recommend one of the following methods to achieve haemostasis:

- **Mechanical treatment** – Direct compression of a bleeding vessel using a designed haemostasis device such as the endoclip or haemoclip.
- **Thermal coagulation (with or without adrenaline injection)** – May be achieved using either contact thermal

Glasgow-Blatchford Criteria

Criteria (on admission)		Score
Hb – Male (g/L)	**Hb – Female (g/L)**	
120–130	100–120	1
100–120		3
<100	<100	6
Urea (mmol/L)		
6.5–8		2
8–10		3
10–25		4
≥25		6
Systolic blood pressure (mmHg)		
100–109		1
90–99		2
<90		3
Others		
Pulse ≥100		1
Melaena		1
Syncope		2
Hepatic disease		2
Cardiac failure		2

From: Blatchford O, Murray W, Blatchford M. A risk score to predict need for treatment for upper gastrointestinal haemorrhage. Lancet 2000; 356: 1318–21

Table 3

Post Endoscopy Rockall Score

Criteria (on admission)	Score
Age*	
<60	0
60–79	1
≥80	2
Shock*	
Pulse >100	1
Systolic BP <100 mmHg	2
Comorbidity*	
Cardiac, other major	2
Renal/liver failure, cancer	3
Endoscopic Diagnosis	
Normal, Mallory-Weiss	0
Ulcer, erosion, oesophagitis	1
Cancer	2
Endoscopic SRH	
Clean base ulcer, flat pigmented spot	0
Active bleeding, clot, vessel, blood	2

*Denotes components of pre-endoscopy Rockall Score. From: Rockall T, Logan R, Devlin H et al. Risk assessment after acute upper gastrointestinal haemorrhage. Gut 1996; 38: 316–21

Table 4

haemostasis (monopolar diathermy) or through non-contact thermal haemostasis (such as argon plasma coagulation; especially useful in the management of angiodysplasia)

- **Fibrin or thrombin treatment (with or without adrenaline injection)** – Specifically designed compounds which may be applied over a large area. These substances mechanically adhere to bleeding points and activate coagulation factors. They are useful when managing large areas of oozing such as in gastritis, malignancy, or portal hypertensive gastropathy.

Injection of adrenaline alone has been demonstrated to be inferior to the above methods; however, it can be utilized as an adjunct.[5,8]

Several novel modalities have been proposed as either an adjunct or a therapeutic alternative to treat non-variceal GI bleeding. Products such as Hemospray (a haemostatic powder spray) have been advocated, particularly in areas difficult to access endoscopically using traditional techniques (such as the lesser curve of the stomach, posterior bulb of the duodenum and gastric cardia). However, there are few randomized, prospective studies.

Endoscopic Therapy – Variceal UGIH – NICE and the British Society of Gastroenterology recommend variceal band ligation (VBL) for oesophageal varices. This involves deployment of a small rubber band around the varices to induce strangulation and thrombosis of the vessel. Following the procedure some patients may develop ulceration at the site of deployment, but this can be improved with PPI use.

Gastric varices should be offered N-butyl-2cyanoacrylate injection as first line therapy. Commonly referred to as glue, this strongly adhesive substance is injected into bleeding varices resulting in haemostasis and has been found to be superior to VBL in achieving haemostasis and reducing re-bleed rates in the sub-cohort of patients. Thrombin injection may also be used for this purpose and has a reported haemostasis rate of 94% with a re-bleed rate of 18%.

When these methods fail to adequately achieve haemostasis, it may be necessary to consider a second-line endoscopic technique to control haemorrhage. Balloon tamponade (e.g. Sengstaken-Blakemore tube) may be undertaken in most cases of oesophageal and junctional variceal haemorrhage. These are successful in controlling haemorrhage in 91% of cases but re-bleed rates are high (approximately 50%), which means they are often utilized in a temporizing manner and should be removed after 2 days. The tubes are often poorly tolerated and may result in pressure necrosis, aspiration pneumonia and, rarely, oesophageal perforation. Patients should be intubated and monitored in an intensive care setting. Self-expanding metal stents have been used for the same purpose and can remain in place for 14 days.[8,10,23]

Use of Hemospray and other novel haemostatic techniques have been trialled in the management of variceal bleeding and while early results are encouraging more studies ae required before such techniques are implemented routinely.

Management - Interventional Radiology – according to NICE guidance interventional radiology should be considered for an unstable patient who has re-bled after endoscopic treatment. However, if there is high risk of re-bleeding or doubt about haemostasis at initial endoscopy, or evidence of rebleeding after initial control, further endoscopy should be planned in the first instance.[10] North American guidelines suggest that computed tomography (CT) angiography should be considered in specific circumstances – arterial bleeding that cannot be controlled endoscopically, no clear source of bleeding, or negative endoscopic findings.[24] Similarly, in unique circumstances such as the early postoperative period following upper gastrointestinal or bariatric surgery, or after trauma, it may be preferred to proceed to radiological management over endoscopy. In the UK such cases will require discussion with senior endoscopy and interventional radiology staff before progressing to radiological treatment. It must be remembered that interventional radiology services are not available out of hours in every UK hospital, whereas following the NCEPOD and upper GI bleed audit findings, every hospital should be able to provide emergency endoscopy.[1]

CT angiography has a sensitivity of 86% and specificity of 95% for obscure GI blood loss and may be used to help identify vascular malformations, neoplasms, and can exclude small and large bowel sources of bleeding. However, in order to accurately identify the source of blood loss, patients must be bleeding at a rate of 0.5 mL/min.[5,6,24]

If a bleeding source is identified, then the interventional radiologist may attempt selective angiography of the mesenteric vessels and radiological embolization where appropriate. The most common source of bleeding and target for embolization is the gastroduodenal artery. Haemostasis is achieved using coils, however several other products including polyvinyl alcohol particles and gelfoam are available.

Embolization of vessels may result in abdominal pain, ischaemia, arterial injury, and contrast induced nephropathy, but is considerably much less morbid than traditional surgical salvage.

In the case of bleeding varices, when endoscopic control has failed, a trans-jugular intrahepatic portosystemic shunt (TIPSS) procedure can be undertaken. This procedure involves radiologically guided deployment of a stent bridging the portal and hepatic veins, creating a portosystemic shunt across the liver parenchyma, resulting in rapid reduction in portal pressure. These procedures are performed only in specialist centres and there are several contraindications and considerations that should be addressed prior to treatment. Early liaison with the specialist liver centre and interventional radiology service is essential when standard endoscopic management has failed.[8,10]

Management – Surgery – once considered to be the default option for patients with uncontrolled upper GI haemorrhage, surgery is now considered the treatment modality of last resort, when all other means to control haemorrhage have failed. Improvements in endoscopic management and increased availability of interventional radiology have seen a fall in surgery for all GI bleeding of 50% over 10 years. The 2007 BSG audit reported that only 2.3% of patients underwent surgical management of uncontrolled haemorrhage.[1,15]

Surgery depends upon the origin and underlying pathology resulting in haemorrhage (Table 5). The most common surgical procedure is under-running or over-sewing of bleeding duodenal or gastric ulcers.

Mortality following surgery is high (29%) and has remained static.[18,19]

Follow-up/Ongoing Care: Intravenous PPI is recommended for 72 hours after successful haemostasis or where there are stigmata of recent haemorrhage with no active bleeding observed.[10]

Long-term primary and secondary PPI prophylaxis can be used; however, side effects include increased risk of hip fracture, *Clostridium difficile* infection, pneumonia, and other symptoms such as diarrhoea. For this reason, one must consider carefully the risks and benefits, with as low a dose as possible prescribed.

It is essential during endoscopy that a Campylobacter-like organism (CLO) test is performed and, if positive, *H. pylori* eradication treatment commenced (PPI for 4 weeks and dual antibiotic therapy (amoxicillin or clarithromycin with metronidazole) for 7 days). *H. pylori* eradication is linked to improved outcomes, reduced rates of re-bleed and if commenced immediately improved compliance is observed. Patients should be re-tested for *H. pylori* at least 6 weeks after initial positive test, and 2 weeks after completing the 4-week course of PPI. Testing for the presence of *H. pylori* whilst taking PPI treatment may result in a false negative result. Retesting can be performed via urease breath test or increasingly via a stool antigen test.

Following variceal bleeding repeat endoscopy is recommended at 2–4 week intervals, as recurrent varices may warrant further band ligation. A non-selective β-blocker such as propranolol or carvedilol may be used to reduce portal pressure by splanchnic vasoconstriction and reduced cardiac output. These have no role in prevention of varices development but may be useful in prevention of bleeding in patients with established cirrhosis and varices.[21]

All patients with portal hypertension should be referred to a hepatology service for ongoing management; screening for hepatocellular carcinoma and surveillance will need to be established.

Surgical options for Upper GI Bleeding

Disease Process	Surgical Options
Peptic ulcer	Oversew
	3-point ligation of gastroduodenal artery
	Vagotomy and pyloroplasty
	Vagotomy and antrectomy
	Highly selective vagotomy
Mallory-Weiss tear	Oversew
Dieulafoy lesion	Oversew
	Wedge resection
Varices	Portacaval shunt
	Mesocaval shunt
	Distal splenorenal shunt
Gastric cancer	Distal gastrectomy
	Total gastrectomy
	D2 lymphadenectomy
Hemobilia	Selective ligation
	Resection of aneurysm
	Nonselective ligation
	Liver resection
Aortoduodenal fistula	Angiography and stent (if hemodynamically stable)
	Open repair
	Extra-anatomic bypass

From Feinman, M. & Haut, E. R. Upper gastrointestinal Bleeding. Surg. Clin. NA 94, 43–53 (2014)

Table 5

Lower GI haemorrhage

Lower GI haemorrhage is less common, with an incidence of 33–77 per 100,000 population. This group forms approximately 3% of all acute surgical admissions.[6] Small volume lower GI bleeding is much more common in the population and likely to be under-reported or assessed by general practitioners.

Unlike upper GI haemorrhage, 80% of lower GI bleeding stops spontaneously after initial resuscitation and correction of coagulopathy with only a small proportion of patients requiring intervention. In most UK hospitals, lower GI haemorrhage is referred to emergency general surgery; however, when the endoscopy suite is required, liaison with gastroenterology will usually be needed. For most patients, ward-based care alone is sufficient.

Overall mortality from lower GI bleeding is 3.4%, and mortality is generally related to comorbidity rather than gross exsanguination.[6] It is essential therefore to exclude an upper GI source of blood loss, and where there is any doubt about origin of haemorrhage, particularly in the haemodynamically compromised patient, rapid assessment for an upper GI bleeding source and preparation for endoscopy should be undertaken. The consequence of missing a true upper gastrointestinal haemorrhage can be catastrophic for the patient.

Pathology

Diverticular bleeding is the most common cause for lower GI bleeding, accounting for approximately 30%–65% of cases in western populations. Diverticular disease is increasingly common with advancing age, but patients in their early thirties may present. The cause is uncertain, but traditionally was thought to be related to lack of fibre and lifestyle factors such as smoking and obesity in western countries. Increasingly, is thought of as part of the inflammatory bowel disease spectrum and may involve some heritability.

Bleeding occurs as a complication when small vessels in the wall of a diverticulum are eroded, most prevalent in the sigmoid and descending colon. Incidence of rebleeding after a single diverticular bleed is low (approximately 15%) but is much higher after subsequent bleeding (approximately 50% of patients with two episodes will have a further bleed).[25,26]

Diverticula of the small bowel, commonly jejunum, may occasionally manifest with rectal haemorrhage and diagnosis usually requires radiological imaging; both upper and lower GI endoscopy will be negative. Such cases, although rare, support the need for progressing quickly to urgent CT imaging in patients who have no diagnosis made at endoscopy.

Vascular abnormalities: Angiodysplasia is responsible for approximately 5%–10% of acute lower GI bleeding. Angiodysplastic lesions mainly affect the caecum and ascending colon but may affect the small bowel in 15% of patients. These result from abnormally dilated mucosal capillaries communicating with tortuous and dilated submucosal veins, and have a typical 2–5 mm flat, red, regular bordered appearance at endoscopy. Incidence increases with age.

Although spontaneous resolution is high (90%), there is a relatively high re-bleed rate with 26% of patients having rebled after 1 year and 45% after 3 years.

Other vascular abnormalities include varices (commonly rectal), and Dieulafoy lesions occurring in the lower GI tract; however, the incidence is rare (<3%).[25,26]

Neoplasms and polyps: Colorectal cancer is the fourth most common cancer in the UK with 42,000 new diagnoses per year. Rectal bleeding is a high-risk symptom that warrants urgent investigation. Colorectal tumours can present with bleeding, usually slow in nature, although it is important to note that benign polyps can also present with haemorrhage.

Fresh red rectal bleeding is mostly associated with left-sided tumours (accounting for >60% of cancers). Overall, neoplasms and polyps are responsible for 2%−15% of acute lower GI bleeding.

Ischaemic colitis and other colitides: Ischaemic colitis accounts for approximately 5%−20% of presentations. It may affect any part of the colon but typically affects the vascular watershed area of the splenic flexure. This area of colon is supplied by the marginal artery of Drummond, which bridges the middle colic (from the superior mesenteric) and left colic (from the inferior mesenteric) arteries. The condition is thought to be caused by inadequate blood supply to the affected colonic wall leading to erosive lesions and secondary bleeding.[25]

There are several risk factors for the development of ischaemic colitis (which may result from either arterial or venous hypoxia), however the condition is broadly divided into occlusive and non-occlusive disease and may be thrombotic or embolic in origin. Management is dependent on the degree of ischaemia with full-thickness necrosis and gangrene indicating the need for surgical resection; however, in most cases the condition will be transient, and resolution of symptoms observed with appropriate non-operative management.

Less commonly, inflammatory colitis (encompassing inflammatory bowel disease, mainly ulcerative colitis) and infective colitis may also result in catastrophic GI haemorrhage.

When pelvic organs have been previously irradiated (e.g. for prostate cancer) one should suspect radiation proctitis or colitis; this can occur months or years after completing treatment.

Meckel's diverticulum: Often referred to by the rule of 2's (under two inches in length, within two feet from the ileocaecal valve, affecting 2% of the population, typically presenting under the age of two and containing two types of heterotrophic mucosa), a Meckel's diverticulum is the most common congenital malformation in the GI tract. They may present with lower GI haemorrhage originating from the distal small bowel as a result of acidic secretions from ectopic gastric mucosa causing ulceration.[26]

Anal lesions and post-procedure bleeding: The management of anal lesions (most commonly haemorrhoids; 5%−20%), post-polypectomy (2%−7%) or postoperative bleeding may be thought of as different from other sources of lower GI haemorrhage as the source of blood loss can be identified from an adequate history and examination.[25]

As with other causes of lower GI bleeding, lesions within the anal canal will often spontaneously stop haemorrhaging; however, where active bleeding is observed during examination (including with proctoscope or rigid sigmoidoscope), attempts can be made at haemostasis through direct application of pressure, cautery or through application of sutures. This may need to be done under general anaesthesia; blind suturing of the anal canal should be avoided; appropriate input from a coloproctologist will be required.

Where control of bleeding in this manner fails or where another source of bleeding is suspected, patients should be managed in the same manner as a more proximal cause.

In the patient who has had a recent polypectomy, there is no need for radiological investigation before proceeding to colonoscopy, which is the diagnostic and therapeutic modality of choice in BSG guidelines. Haemoclips and endoclips with or without adrenaline are recommended to control bleeding. Heater probe and bipolar diathermy anywhere other than in the rectum (below the peritoneal reflection) should be used with caution as the bowel wall is thinner and at increased risk of perforation following polypectomy.[6]

Bleeding following right hemicolectomy with stapled ileocolonic anastomosis is common and can manifest with significant amounts of brisk lower GI blood loss originating from the staple line. This is usually in the immediate postoperative period, but occasionally patients may be readmitted after discharge. Supportive treatment via resuscitation, with blood products, and correction of coagulopathy is recommended as first line treatment, but if haemorrhage is not controlled, re-operation may be necessary.

In the patient presenting with bleeding per-stoma, digital examination, and thorough assessment of the stomal orifice should be undertaken. Similarly, patients with a defunctioning stoma can present with rectal bleeding originating from the distal rectum or anal canal, especially if radiotherapy has been used. If a local cause is not found, then the patient should be managed the same as all others, with note made of the type of previous surgery and presence or not of rectum and anal canal. Such background should be communicated clearly to the endoscopist or radiologist when undertaking further investigations.

Risk stratification: British Society of Gastroenterology (BSG) guidelines recommend a two-staged approach to the assessment of the patient with lower GI bleeding. Initially the patient should be assessed for signs of haemodynamic instability and shock (calculated using the shock index (SI = heart rate/systolic blood pressure)) and if the patient is haemodynamically unstable or has SI>1, emergent investigation with or without definitive management is required.

Haemodynamically stable patients or those with an SI of ≤1, should be assessed using the Oakland score (Table 6).

This system allows identification of patients who are unlikely to suffer a serious adverse event (Oakland score ≤8). Without other indication for admission, this recommends safe discharge for expedited outpatient investigation. The Oakland score is validated and specific to lower GI bleeding in the UK and is superior to other risk assessment tools in predicting safe discharge, transfusion requirements and re-bleed, but is inferior to other scoring systems in predicting mortality.[6]

Management − CT angiography and interventional radiology: When there is haemodynamic instability or where active bleeding is suspected, BSG guidelines suggest that CT angiography should be performed. When a bleeding source is identified through extravasation of contrast appearing as a blush, if local

expertise is available, patients may then undergo targeted intervention through embolization. If a patient is unstable, this should take place within an hour. When interventional radiology services are unavailable, such findings give an indication to the endoscopist of the origin of bleeding and will help guide endoscopic intervention. Again, it must be reiterated that if CT angiography is negative, an upper GI bleeding source must be considered, and such patients should proceed to endoscopy.

There are no high-quality studies comparing embolization and endoscopic intervention in the management of lower GI bleeding; however, targeted embolization is reported to be 93%–100% successful. Empirical arterial embolization may also be beneficial, even where no active bleeding is seen, and this may be particularly useful when dealing with bleeding from a tumour, with a reported clinical success rate of 68%.

Oakland Score

Predictor	Score component value
Age	
<40	0
40–69	1
≥70	2
Gender	
Female	0
Male	1
Previous LGIB admission	
No	0
Yes	1
ORE findings	
No blood	0
Blood	1
Heart rate	
<70	0
70–69	1
90–109	2
≥110	3
Systolic blood pressure	
<90	5
90–119	4
120–129	3
130–159	2
≥160	0
Haemoglobin (g/L)	
<70	22
70–89	17
90–109	13
110–129	8
130–159	4
≥160	0

Patients scoring ≤8, with no other indications for hospital admission are suitable for immediate discharge from Accident and Emergency and outpatient investigation. ORE, digital rectal examination; LGIB, lower gastrointestinal bleeding.
From Oakland, K. et al. Diagnosis and management of acute lower gastrointestinal bleeding: guidelines from the British Society of Gastroenterology. Gut; 0: 1–14 (2019)

Table 6

There are risks associated with embolization including bowel ischaemia (7%–24%) and rebleeding (10%–50%). Empirical embolization is associated with a higher 30-day mortality (31%) compared to targeted treatment (9%). BSG guidelines therefore conclude that the decision to proceed to embolization or primary therapeutic colonoscopy following CTA should be based on local expertise and patient factors.[6]

Management – flexible sigmoidoscopy/colonoscopy: Identification of a bleeding point and ability to achieve haemostasis is often more challenging due to limitation of view (presence of faecal matter and blood) in the unprepared bowel. It may be possible to give the patient bowel preparation or an enema to improve visualization, but views may still be unsatisfactory, with reports of diagnostic yield ranging widely from 48% to 100%.[6,26]

The timing of endoscopic intervention remains controversial with many conflicting studies; however, BSG guidelines suggest that for patients with evidence of major bleeding, this should take place on the next available list and within 24 hours after admission to hospital.[6]

The endoscopic management of lower GI bleeding includes using the same haemostatic techniques as used in acute upper GI haemorrhage, namely mechanical treatment, use of thermal coagulation and fibrin or thrombin products. No one technique appears superior to another, however BSG recommend the use of mechanical clips as first line modality in diverticular bleeding due to low risk, widespread availability, and ease of use. Angiodysplastic lesions may, on the other hand, be more amenable to thermal coagulation.[6,26]

CO_2 and gas exchange should be used, and diathermy and argon plasma coagulation limited in this setting due to the risk of gas explosion. Sub-mucosal infiltration of adrenaline may be useful in obtaining initial haemostasis, but its use should be limited in the rectum and anal canal due to the risk of migration into the systemic circulation.[6]

Management – other options: In approximately 10% of patients with lower GI bleed the source of the bleeding is never identified. Where patients remain haemodynamically stable there is the option to repeat investigations or to progress to more specialized tests including Tc99m scintigraphy, CT or MR enterography, video capsule endoscopy, push enteroscopy or double balloon enteroscopy. These investigations are of value when obscure small bowel bleeding is suspected, but availability is limited and may require specialist referral.[6,26]

Management – surgery: In extremely limited circumstances, e.g., an aorto-enteric fistula, surgery would be the treatment of choice; otherwise, it should be considered a salvage option, only to be used when all other means to control haemorrhage have failed.

Where there is an identified colonic bleeding source, but haemorrhage control has failed, a segmental colectomy may be performed. Where no identifiable bleeding source is identified a subtotal colectomy may be undertaken. However, both procedures are associated with significant morbidity and mortality.[6,26]

Recent guidelines suggest that immediately prior to skin incision, a further attempt should be made using on-table colonoscopy to identify a bleeding source and where possible control haemorrhage. It is suggested that only a specialist colorectal

surgeon should undertake such a procedure due to the associated risk.[6]

Summary

Massive GI haemorrhage is a relatively common emergency presentation to hospital and is associated with significant risk of mortality. Outcomes can be improved by rapid assessment and resuscitation, correction of coagulopathy, and early diagnosis and intervention.

Interventional endoscopy and radiology are now the investigative and therapeutic modalities of choice, with surgery only considered as a last resort when other treatment strategies have failed. ◆

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Practice points

- Upper and lower GI haemorrhage are common emergency presentations associated with significant risk of mortality.
- Validated risk scores for both upper and lower GI haemorrhage are now available to aid selection and timing of investigation.
- Prompt endoscopy is the investigation and treatment modality of choice for upper GI haemorrhage.
- For the unstable patient with lower GI haemorrhage, management via CT angiography is recommended. For stable patients, early colonoscopy is preferred.
- Surgery remains the last treatment option once it is clear that other modalities have failed, or there has been rebleeding even after repeat attempts to control the source of haemorrhage.