15.4.2 Gastrointestinal bleeding 2771

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15.4.2 Gastrointestinal bleeding 2771 15.4.2 Gastrointestinal bleeding Vanessa Brown and T.A. Rockall ESSENTIALS Gastrointestinal bleeding (GIB) is a common emergency, which can be subdivided into upper and lower, and acute or chronic, with acute upper GIB further subdivided into variceal (11%) and nonvariceal (89%) bleeding. Risk stratification in acute upper GIB can be performed using simple clinical and endoscopic criteria that can be used to estimate the risk of mortality, but there are no validated systems for use in acute lower GIB. The immediate management of the hypovolaemic patient is first directed towards resuscitation and then to identification of the site and cause of bleeding. Most patients will stop bleeding spontan- eously and should then be investigated with either upper gastro- intestinal endoscopy or colonoscopy as appropriate. Patients with acute ongoing upper GIB require urgent investiga- tion by oesophagogastroduodenoscopy with a view to applying endoscopic haemostatic therapy, which is efficacious in up to 95% of patients. High-dose proton pump inhibitor treatment should be given following successful endoscopic therapy to patients with major ulcer bleeding. If these techniques fail to arrest bleeding, then ei- ther selective mesenteric angiography with embolization or surgery is indicated. Patients who are unstable with acute lower GIB require early oesophagogastroduodenoscopy (to exclude an upper gastrointes- tinal cause) and then an interventional radiological procedure to embolize the bleeding vessel(s); surgery is generally a last resort. Introduction Gastrointestinal bleeding (GIB) is a common emergency, with approximately 85 000 cases per year in the United Kingdom. It is subdivided into upper and lower, and acute or chronic, with acute upper gastrointestinal haemorrhage further subdivided into variceal (11%) and nonvariceal (89%) bleeding. Despite significant advances in the management of GIB, particu- larly the introduction of proton pump inhibitors (PPIs) and advances in endoscopy, published mortality rates remain similar to the 1950s. This is because patients presenting with acute upper GIB are now older and have more comorbidities. The incidence of peptic ulcers in younger patients has fallen since the introduction of PPIs. Mortality therefore is often due to the complications associated with advanced age and multiple comorbidities as opposed to exsanguination. In- hospital mortality for patients admitted with acute upper GIB is 7%, but up to 15% of acute upper GIB occurs in established inpatients and is associated with a much higher mortality rate of approximately 30%. A

recent report has recommended that patients with any acute GIB should only be admitted to hospitals with 24-h on-site endoscopy, GIB surgery, critical care, and 24-h access to interventional radiology. Definition Acute gastrointestinal haemorrhage is classified by its origin, from either the upper or the lower gastrointestinal tract, anatomically de- marcated by the ligament of Treitz (at the junction of the duodenum and jejunum). Upper GIB is further subdivided into nonvariceal and variceal haemorrhage. Only when gastrointestinal bleeding is acute does it constitute an emergency. Chronic, low-volume blood loss is usually subclinical until such time as it presents with iron deficiency anaemia. Acute upper gastrointestinal bleeding Aetiology and pathogenesis Many gastrointestinal lesions may result in haemorrhage but peptic ulcer is the most frequent cause of acute UGIB in the United Kingdom (Table 15.4.2.1). Each diagnostic group has its own aetiological factors. Peptic ulcer disease Peptic ulcer disease accounts for 35% of cases of acute upper GIB. Helicobacter pylori infection and ingestion of aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) are important aetiological factors for peptic ulcer disease. Stress ulcers can de- velop in critically ill patients, for example, those in intensive care environments, and are caused by mucosal ischaemia. Ulceration may also occur at the site of surgical enterostomies (stomal ulcers) and in association with the Zollinger–Ellison syndrome. Peptic ul- ceration at specific sites is associated with major haemorrhage due to the anatomical relation of major arteries—the posterior wall of the first part of the duodenum (gastroduodenal artery), the lesser curve of the stomach (left gastric artery), and posterior wall of stomach (splenic artery). Varices Variceal bleeding is increasing in incidence. In Western countries, liver disease due to alcohol and hepatitis are the principal causes of portal hypertension, which leads to oesophageal varices; vari- ceal bleeding may rarely affect the stomach and the remaining Table 15.4.2.1 Diagnoses following acute upper gastrointestinal haemorrhage Diagnosis % Peptic ulcer 35–50 Erosive disease 10–15 Oesophagitis 10 Mallory–Weiss tear 5–10 Oesophageal varices 5–10 Upper gastrointestinal malignancy <5 Vascular malformations 5 Other/not established 5–15

 gastrointestinal tract. Bleeding from varices is often severe and com- plications associated with underlying liver disease mean this patient group are best managed by specialists in the care of patients with liver disease. Mallory–Weiss tears These are mucosal lesions at the oesophagogastric junction asso- ciated with profuse vomiting (most commonly from alcohol). The haematemesis that occurs follows a normal vomit, and is usually minor and nearly always self-limiting. Erosive disease Oesophagitis due to gastro-oesophageal reflux disease, gastritis, duodenitis, and gastroduodenal erosions are associated with the use of aspirin, NSAIDs, and H. pylori infection. Rare causes The most common malignant causes of upper gastrointestinal haem- orrhage are adenocarcinoma of the stomach and gastric lymphoma, but acute bleeding is an unusual presentation. Other causes include gastrointestinal stromal tumours, which may bleed when the mu- cosal surface ulcerates, angiodysplasia (and other vascular lesions), aortoduodenal fistula, haemobilia, and trauma. Epidemiology The incidence of acute upper GIB in the United Kingdom is approxi- mately 1 per 1000 adults/year, accounting for 50 000 to 70 000 acute hospital admissions per year. Fifteen per cent of cases occur in patients already in hospital. The male incidence is twice that of the female in all age groups except older people, where they are similar. The annual in- cidence increases dramatically with age, in a recent nationwide survey of patients requiring 4 or more units of blood, the mean age for vari- ceal upper GIB was 53 and for nonvariceal was 73 years old. Prevention All patients with a peptic ulcer should be tested for H. pylori infec- tion, and if positive, eradication therapy should be prescribed. This significantly reduces the risk of ulcer recurrence. Patients in hospital, particularly elderly people

and those who have had surgery, are at higher risk of acute upper GIB. For this group when prescribing NSAIDs, it is prudent to consider coprescription of a PPI. Variceal haemorrhage can be prevented through programmes of variceal eradication by injection or banding and also by transjugular intrahepatic portosystemic shunt, or ultimately liver transplantation where indicated. Clinical features History The most common presentation in major acute upper GIB is haema- temesis or 'coffee-ground' vomiting, melaena (see Box 15.4.2.1), and a decrease in haemoglobin. Frank haematemesis indicates a severe bleed. It is not always a feature, but melaena will always follow a significant bleed. The absence of blood in the first vomit suggests a Mallory–Weiss tear. In rapid bleeding, symptoms of hypovolaemia may precede haematemesis or melaena. These include postural hypotension, syn- cope, shock, and even death. It is particularly important to pursue the past medical history for evidence of liver disease (alcohol, risk factors for hepatitis). With re- gard to drug history, the patient should be asked about ingestion of aspirin and other NSAIDs, and anticoagulants. The use of β-blockers may mask tachycardia associated with shock. Examination The first concern must be airway, breathing, circulation: the patient must be assessed rapidly for signs of hypovolaemic shock. Signs of liver disease may be present in patients with oesophageal varices, but this does not confirm the cause of blood loss since peptic ulcer is a common synchronous lesion. Rectal examination looking for fresh blood or evidence of melaena must not be neglected. Differential diagnosis In patients with rapid haemorrhage, usually accompanied by shock, fresh blood may be passed per rectum (haematochezia) and thus is difficult to distinguish from lower GIB. A large nasopharyngeal bleed, resulting in a significant volume of swallowed blood, can also present with haematemesis. Investigations Acute upper GIB is mainly a clinical diagnosis. Full blood count and coagulation tests In actively bleeding patients, the haemoglobin concentration will only fall after haemodilution has occurred, hence the initial haemo- globin estimation is not a useful indicator of the volume of blood loss. The haemoglobin level may be normal in a patient with a large, acute haemorrhage and cannot be relied upon to guide resuscita- tion. Equally, it may be low in a patient with iron deficiency anaemia resulting from chronic haemorrhage who presents with a small, acute bleed. The haemoglobin and haematocrit concentrations after volume resuscitation are more useful. Platelet count and coagula- tion studies are important to exclude a bleeding disorder and are of particular relevance in patients receiving therapeutic anticoagulants and in those with liver disease. Other blood tests Urea, creatinine, and electrolytes should be checked: the serum urea may rise disproportionately to the serum creatinine as the absorbed products of luminal blood are metabolized by the liver, and it is important to establish baseline renal function. Serum liver-related tests are required as a marker of liver disease. Group and save, or cross-match, may be required depending on clinical severity. Endoscopy Required for both diagnosis and treatment, as discussed later. Box 15.4.2.1 Melaena This is a clinical diagnosis made on the observation of black, tarry, offensive stool on rectal examination (or passed spontaneously). It occurs as the result of digestive enzymes and bacteria acting on haemoglobin. Although melaena is usually due to acute upper GIB, bleeding from the small bowel or right side of the colon may also pre- sent in this way.

 Management The management of acute upper GIB falls into four principal stages (Box 15.4.2.2). A pathway for management is shown in Fig. 15.4.2.1. Resuscitation, assessment, and monitoring Resuscitation Resuscitation is as for any hypovolaemic patient, with the immediate aim of rapidly restoring blood volume. Tachycardia, vasoconstric- tion, sweating, hypotension (including a postural drop), tachyp- noea, and a low central venous pressure all indicate hypovolaemia. Large-bore peripheral venous access, central venous access, and

Resuscitation

1. Adequate IV access - Large-bore peripheral venous access plus/minus central venous access
2. Urinary catheter
3. Balanced transfusion • Blood, platelets, and FFP as per local haematology guidelines • Activate massive transfusion protocol if necessary
4. Correct coagulation abnormalities
5. Monitoring - regular pulse, blood pressure, urine output and central venous pressure (if available). Transfer to high dependency setting if necessary
6. Drugs • Terlipressin if risk of variceal bleeding • Consider Tranexamic acid
7. Risk assessment
8. Early involvement of anaesthetists and surgeons if ongoing haemodynamic compromise
9. All patients should have a documented rebleed plan that should be updated after every intervention OGD Nonvariceal •Endoscopic treatment with dual therapy •Start PPI Variceal •Prophylactic anitbiotics •Oesophageal varices – band •Gastric varices – N-butyl-2-cyanoacrylate Second OGD Interventional Radiology? Mesenteric angiogram ± embolization Surgery TIPS Stable Unstable Rebleeding No Yes Rebleeding Fig. 15.4.2.1 Pathway for management of acute upper GIB. FFP, fresh frozen plasma; OGD, oesophagogastroduodenoscopy; PPI, proton pump inhibitor; TIPS, transjugular intrahepatic portosystemic shunt. Box 15.4.2.2 Management of acute upper GIB 1 Resuscitation, assessment, and monitoring 2 Diagnosis and haemostasis: — Endoscopy — Interventional radiology — Surgery — Drugs 3 Treatment of causative lesion: — Nonvariceal — Variceal 4 Prevention of recurrence

SECTION 15 Gastroenterological disorders 2774 placement of a urinary catheter will help in the resuscitation and monitoring of more severe cases and those with major cardiovas- cular and respiratory comorbidity. A haemoglobin concentration less than 7 g/dl is associated with compromised cardiac function and impaired tissue oxygenation. However, overtransfusion may be as damaging as undertransfusion, particularly in elderly patients with multiple comorbidities, and there has been much recent debate regarding indications for blood transfusion. Recent studies with varying levels of evidence have concluded that a restrictive policy and low trigger threshold for transfusion is beneficial, and that even if there is no absolute clin- ical advantage of a restrictive practice, then—as long as this is not detrimental—avoidance of blood transfusion both reduces transfu- sion risk and saves the health economy money. Decisions on trans- fusion should be patient specific, taking into account underlying comorbidities and their ability to tolerate an aggressive restrictive transfusion strategy. All patients with massive bleeding should have replacement of blood, platelets, and clotting factors in order to restore volume and maintain clotting, which should be administered in line with local major transfusion protocols (Box 15.4.2.3). The National Institute for Health and Care Excellence (NICE) recommends that (1) plate- lets should be given to actively bleeding patients with platelet concentrations of less than 50 × 109 (but not to those that are haemodynamically stable and not actively bleeding), and (2) fresh frozen plasma should be given if the concentration of fibrinogen is less than 1 g/litre (with cryoprecipitate offered if fibrinogen levels remain <1.5 g/litre despite fresh frozen plasma) or if INR or acti- vated partial thromboplastin time are greater than 1.5 times the normal value. Assessment of risk There are several scoring systems to predict the need for interven- tion, risk of rebleeding, and risk of death.

The simplest and most widely used scoring system is the Rockall score, which was devel- oped to stratify patients for the risk of death. The full Rockall score can only be calculated after endoscopy. The Blatchford score can be used prior to endoscopy and is a better predictor of rebleeding and the need for intervention. Both the Rockall score and the Blatchford score have been extensively externally validated. Recent NICE guidelines recommended that all patients with acute upper GIB should have a formal risk assessment with the Blatchford score at first assessment and the full Rockall score after endoscopy. Early discharge should be considered in patients with a Blatchford score of 0. Independent risk factors that accur- ately predict mortality have been identified (see 'Prognosis' and Tables 15.4.2.2 and 15.4.2.3). These include increasing age, comorbidity, shock, and endoscopic findings. Monitoring Once circulating blood volume has been restored, management should be aimed at monitoring the patient for continued or recur- rent bleeding, replacing blood, making a diagnosis, and instituting therapy. Regular pulse, blood pressure, central venous pressure, and urine output will give a good guide. High-risk patients should be monitored in a high-dependency unit or intensive therapy unit. Ten to fifteen per cent of patients with an acute upper GIB will have a rebleed, and all patients should have a documented rebleed plan that should be updated after every intervention. While further fresh haematemesis obviously indicates another acute haemorrhage, the passage of further melaena has to be interpreted in the light of the cardiovascular signs and repeated estimations of blood haemo- globin concentration.

Table 15.4.2.2 The Rockall score: acute upper gastrointestinal haemorrhage scoring system

Variable	Score 0	1	2	3
Age (years)	<60	60–79	≥80	
Shock	'No shock'	'Tachycardia'	'Hypotension'	
Systolic BP (mmHg)	≥100	≥100	<100	
Pulse (beats/min)	<100	≥100		
Comorbidity	No major comorbidity	Cardiac failure Renal failure Ischaemic heart disease	Liver failure Any major comorbidity Disseminated malignancy	
Diagnosis	Mallory–Weiss tear All other diagnoses	Malignancy of upper gastrointestinal tract		
No lesion identified and no stigmata of recent haemorrhage		Major stigmata of recent haemorrhage None or dark spot only	Blood in upper gastrointestinal tract Adherent clot	Visible or spurting vessel

Box 15.4.2.3 Definition of major haemorrhage—when to activate the major transfusion protocol Loss of more than • 1 × blood volume in 24 h • 50% of blood volume in less than 3 h • Greater than 150 ml/min

15.4.2 Gastrointestinal bleeding 2775 Diagnosis and haemostasis Haemostasis occurs spontaneously in most cases. When bleeding continues, haemostasis can be achieved by endoscopic, radiological, or surgical means. Endoscopy Endoscopy is the diagnostic and therapeutic investigation of choice in all patients with upper GIB (Box 15.4.2.4) but should only occur after optimal resuscitation. NICE (2013) recommends oesophagogastroduodenoscopy is performed within 2 h of op- timal resuscitation in those patients with haemodynamic com- promise, and within 24 h of all those with a presentation of an upper GIB. Patients in whom haemodynamic stability cannot be achieved despite adequate resuscitation should have concomitant oesophagogastroduodenoscopy and resuscitation. These patients should be discussed with anaesthetists early as they are at increased risk of aspiration and often require postprocedural critical care. Endoscopic haemostatic therapy may be given in the form of the following: • Injection of adrenaline—this is performed in quadrants around the bleeding point, and then into the bleeding vessel, using a total of 4 to 16 ml of a 1:10 000 adrenaline solution in normal saline. Haemostasis is achieved in 95% of cases, although bleeding can recur in 15 to 20%. Adrenaline injection alone is inferior to dual- modality treatments or mechanical clipping. • Mechanical—clips can be applied to bleeding points and are par- ticularly useful for actively bleeding large vessels (Fig. 15.4.2.2). •

Thermal energy—the application of heat energy in the form of a heater probe or diathermy is effective. The heater probe is useful because it includes a powerful water jet which aids clot removal. Laser therapy is no longer used. • Fibrin glue or thrombin works by encouraging clot formation and has been shown to be effective. • Injection of other agents such as sclerosants (e.g. polidocanol) or alcohol does not confer additional advantage, but does increase the risk of perforation There is good trial evidence that endoscopic therapy reduces the rate of rebleeding and mortality. The reported rate of rebleeding after endoscopic therapy is between 5 and 20%. A peptic ulcer greater than 2 cm in size and hypotension are both risk factors for rebleeding. Endoscopic therapy can be repeated if a patient rebleeds and cur- rent evidence supports two attempts at endoscopic control in most cases. Repeat endoscopy confirms that bleeding has recurred and can allow further endoscopic treatment. It can also enable plan- ning as metal clips placed at the site of bleeding can be used to guide subsequent radiological management. The benefits of further endoscopic therapy need to be balanced against the risk of delaying definitive treatment should further bleeding occur. Patients who rebleed after endoscopic treatment and are unstable should have interventional radiology or urgent surgery if interven- tional radiology is not immediately available. There is no evidence from randomized controlled trials that planned, repeated endo- scopic therapy further reduces rebleeding in peptic ulcer. Repeated injection or banding has been shown to reduce rebleeding and mor- tality from oesophageal varices. A second attempt at endoscopic therapy should be made (espe- cially in young patients) before resorting to surgery, although there is rarely any place for a third attempt. Radiological studies and interventions Nuclear scintigraphy Nuclear scintigraphy, discussed further in the section on acute lower GIM, can be used to detect active haem- orrhage but cannot determine the underlying cause (Fig. 15.4.2.3). Table 15.4.2.3 Observed rebleeding and mortality by risk score Score 0 1 2 3 4 5 6 7 8+ Rebleed (%) 4.9 3.4 5.3 11.2 14.1 24.1 32.9 43.8 41.8 Deaths no rebleed (%) 0 0 0.3 2.0 3.5 8.1 9.5 14.9 28.1 Deaths with rebleed (%) 0 0 0 10.0 15.8 22.9 33.3 43.4 52.5 Deaths total (%) 0 0 0.2 2.9 5.3 10.8 17.3 27.0 41.1 Box 15.4.2.4 The aim of endoscopy is: 1 To confirm the diagnosis and aetiology 2 To apply haemostatic therapy where appropriate 3 To assess the risk of further haemorrhage based upon the site, size, and nature of the lesion (including stigmata of recent haemorrhage) 4 To inform the radiologist or surgeon as to the site of the lesion in cases requiring urgent surgery due to rapid, ongoing blood loss, and to exclude varices in these cases Fig. 15.4.2.2 Clipped bleeding in Mallory–Weiss tear. From Marks D, Harbord M (2013). Emergencies in gastroenterology and hepatology. By permission of Oxford University Press.

 In cases where maximal endoscopic therapy has failed, inter- ventional radiology and embolization of the bleeding vessel is the second-line treatment. In those patients with a duodenal ulcer, this is often the gastroduodenal artery. Recent guidance stated ‘No pa- tient should undergo surgery for nonvariceal upper GI bleeding without first undergoing endoscopic treatment, and if this fails or is inappropriate, interventional radiology’ (Fig. 15.4.2.4). CT angiography can detect bleeding rates higher than 0.3 to 0.5 ml/min. It allows assessment of the entire gastrointestinal tract and can delineate between upper and lower GIB when the diagnosis is in doubt. It has an accuracy of up to 89%, can be used to localize the site of bleeding, and can guide radiological or surgical inter- vention. Mesenteric catheter angiography allows identification and embolization of any bleeding points, but requires specialized skill to selectively cannulate often second- or third-order arterial branches. As with endoscopy, these patients should be discussed with an- aesthetists early as they often require concomitant resuscitation and postprocedural critical care. Surgery Due to advances in endoscopic and

radiological treatments, emer- gency surgery for acute upper GIB is now rarely undertaken and is generally considered a last resort. It is indicated in massive, acute bleeding not amenable to endoscopic therapy, or where endoscopic therapy fails to control active bleeding, with operative mortality being of the order of 30%. Elective or semielective surgery is indicated when malignancy is suspected or in lesions considered at high risk of perforation, and there is some evidence that early surgical intervention in those older than 60 is appropriate. It is important to inform an experienced surgeon about the possible need for surgery at an early stage. All patients should have a formal assessment of preoperative risk to inform postoperative care and treatment decisions, and a docu- mented rebleed plan should bleeding recur after surgery. Drugs Acid suppression Patients in critical care should be given acid suppression with PPIs or H2 receptor antagonists to prevent upper GIB. This medication should be reviewed on discharge. Tranexamic acid Tranexamic acid is recommended in patients with ongoing haemodynamic compromise or a delay in intervention. The HALT-IT trial—a randomized, double blind, placebo-controlled trial which intends to recruit 12 000 patients—is evaluating whether Fig. 15.4.2.3 Tagged red blood cell scan showing radiotracer activity in the right upper quadrant (black arrows) in a 48-year-old woman with a history of a roux-en-Y gastric bypass who presented with melaena. From Covey AM, Pua BP, Aguado A, Madoff DC (2014). Interventional radiology cases. By permission of Oxford University Press. (a) (b)
Fig. 15.4.2.4 (a) Superselective arteriogram of a branch of the pancreaticoduodenal artery shows contrast extravasation into the duodenum (arrow) in the patient whose tagged red cell scan is shown in Fig. 15.4.2.3. (b) Following coil embolization to exclude the bleeding branch. From Covey AM, Pua BP, Aguado A, Madoff DC (2014). Interventional radiology cases. By permission of Oxford University Press.

15.4.2 Gastrointestinal bleeding 2777 the early administration of tranexamic acid to patients with acute GIB reduces mortality. It is due to finish recruitment in May 2019. Anticoagulants and antiplatelet agents In life-threatening haem- orrhage warfarin should be stopped and the INR corrected with vitamin K and prothrombin complex concentrate in line with local haematology protocols. In the acute bleeding phase, NSAIDs (including cyclooxygenase-2 inhibitors) should be stopped until bleeding is controlled. Low-dose aspirin for secondary prevention can be continued once haemostasis has been achieved. For patients on clopidogrel or dipyridamole, the risk and benefits of continuing should be discussed with the patient and the appropriate specialist. Treatment of causative lesion Treatment of the causative lesion should be started as soon as possible after diagnosis. Non variceal upper GIB Maintaining gastric pH higher than 6.5 enhances platelet aggrega- tion and stabilizes clot formation, but there is no evidence that the use of PPIs prior to endoscopy changes clinical outcomes; hence, in patients with major ulcer bleeding—following successful endo- scopic therapy—treatment with a high-dose PPI is recommended in accordance with local hospital guidelines (e.g. omeprazole 80 mg immediately followed by an infusion of 8 mg hourly for 72 h). PPIs are effective in healing peptic ulcers and have been shown to reduce the risk of rebleeding in patients with a nonbleeding visible vessel, although studies have not demonstrated a significant reduction in overall mortality. Elective surgery may be indicated where the causative lesion is a tumour (benign or malignant). Angiodysplasia can be treated with laser or argon beam therapy. Specific treatments may be required for rarer causes such as Crohn’s disease or tuberculosis. Variceal upper GIB A recent report has shown that patients with alcoholic liver disease who present with an upper GIB are equally likely to have a variceal as a nonvariceal bleed. The oesophagus is the site of varices in 80% of patients with cirrhosis. Current guidelines recommend that patients with suspected or confirmed variceal bleeding should have

terlipressin and prophy- lactic antibiotics at presentation. Terlipressin is a long-acting ana- logue of vasopressin and works by reducing portal pressure by constricting splanchnic arterioles. When used in conjunction with endoscopic therapy it has been shown to improve mortality and de- crease rebleeding rates. It should be stopped after definitive haemo- stasis or after 5 days. Prophylactic antibiotics have been shown to reduce the incidence of bacteraemia and spontaneous bacterial peritonitis. Uncontrolled variceal haemorrhage may be controlled with a Sengstaken–Blakemore tube as a temporary measure before more definitive treatment (Fig. 15.4.2.5). Endoscopic band ligation should be the first-line therapy in all patents with oesophageal variceal upper GIB and has been shown to have a significant mortality benefit. Patients with bleeding gastric varices should be offered endoscopic injection of N-butyl-2-cyanoacrylate. Transjugular intrahepatic portosystemic shunt is a minimally in- vasive method of creating a portosystemic shunt, thereby reducing portal pressure. It is the recommended therapy for bleeding oe- sophageal and gastric varices that are not responsive to endoscopic management (Fig. 15.4.2.6). Outcomes for patients with variceal upper GIB are affected by the severity of the underlying liver disease and the degree of bleeding; hence, patients with variceal upper GIB should have a formal assess- ment of the severity of cirrhosis by a Childs–Pugh score and ideally be managed by a specialist hepatologist. Oesophageal transection is now rarely undertaken, but is occa- sionally life-saving where all other attempts at haemostasis have failed, although is associated with a very high mortality. Fig. 15.4.2.5 Sengstaken–Blakemore tube. From Marks D, Harbord M (2013). Emergencies in gastroenterology and hepatology. By permission of Oxford University Press. Fig. 15.4.2.6 Transjugular intrahepatic portosystemic shunt following deployment. The white arrows point to the ends of the shunt. From Marks D, Harbord M (2013). Emergencies in gastroenterology and hepatology. By permission of Oxford University Press.

 Prevention of recurrence Recurrent episodes of bleeding from peptic ulcers can be prevented by eradicating H. pylori infection and through the avoidance of ulcerogenic drugs. Persistent ulceration despite these measures may require long- term acid suppressive therapy. Zollinger–Ellison syn- drome should be excluded. Prognosis Prognosis depends on many factors including the severity of the bleed, the age of the patient, the associated comorbidity of the patient, the diagnostic category, the endoscopic features (stigmata of recent haem- orrhage), and whether continued or recurrent bleeding is a feature. Overall, the crude mortality for patients presenting to emergency departments with acute upper gastrointestinal haemorrhage is about 10%, but is significantly higher (approximately 30%) among in- patients who develop GIB while hospitalized for other reasons. Most deaths occur in older people and those with severe comorbidity. Death in those under the age of 60 with no comorbidity is very low (0.1%) regardless of the severity of the haemorrhage. The factors that contribute to mortality have been combined in a prognostic risk score which is represented in Table 15.4.2.2. The mortality associ- ated with each risk score is represented in Table 15.4.2.3. Acute lower gastrointestinal bleeding Epidemiology Lower GIB accounts for approximately 1% of acute hospital admis- sions. It is three times less common than upper GIB and has a signifi- cantly lower mortality (2.2%). Lower GIB occurs more commonly in the elderly, with a mean age of 74 years at presentation in a recent national study in the United Kingdom. In contrast to upper GIB, pa- tients present with a higher haemoglobin concentration, less shock, and are less likely to require a blood transfusion. Approximately 15% of patients with acute severe rectal bleeding/haematochezia will have an upper gastrointestinal source of bleeding. Most (80–85%) acute lower GIBs will stop spontaneously, al- though 35% will require blood transfusion, 25% of those with severe lower GIB

will have a rebleed, and 5 to 10% will require urgent sur- gical intervention. Aetiology As in upper gastrointestinal haemorrhage, several pathological causes are responsible. Most causative lesions are colonic or ano- rectal, with 0.7 to 9% originating in the small bowel (Table 15.4.2.4). In developed countries, diverticular disease represents the largest proportion of cases. Bleeding is not uncommonly associated with coagulopathy, but studies have shown the distribution of causative lesions in these cases to be the same, although in severe cases there may be generalized mucosal bleeding. Diverticular disease Acute colonic diverticular bleeding is common. The estimated risk of bleeding with this disease is about 15%. After a single bleed, the risk of recurrence is 25%, and after two bleeds it is 50%. Eighty per cent of all bleeds stop spontaneously and no therapy is indicated. Operative intervention should be considered after two major bleeds because the risk of further recurrence is high. However, many of these patients are frail and elderly, and continuation of conservative treatment for multiple, self-limiting episodes may be appropriate. Inflammatory bowel disease This often manifests itself as bloody diarrhoea, but more rarely may present with profuse haemorrhage. This is more common in Crohn’s disease than in ulcerative colitis because the inflammation involves the whole thickness of the bowel wall, and up to 6% of patients with this disease may sustain a major haemorrhage. About 50% stop bleeding spontaneously, but of these 35% will rebleed. For this reason, urgent surgery is usually indicated for patients with a life-threatening haemorrhage as a result of inflammatory colitis. The operation usually required is a subtotal colectomy, with the rectum usually being pre- served at this stage unless this is the site of major haemorrhage. Ischaemic colitis rarely causes severe haemorrhage. Bloody diar- rhoea is more usual and may be accompanied by pain. Colonic tumours Benign and malignant colonic tumours may present as profuse bleeding, although occult blood loss and minor fresh bleeding is more common. A history of change in bowel habit, weight loss, and pain are suggestive of colorectal cancer. Rarely is urgent surgical intervention required. Angiodysplasia Vascular anomalies occur with increasing frequency with age. They may originate from chronic, partial venous obstruction of sub- mucosal veins due to incompetence of the precapillary sphincters and arteriovenous malformations. These lesions are usually mul- tiple and are most frequent in the caecum and ascending colon. Bleeding is usually slow, intermittent, and recurrent, although it is occasionally massive (2–15%). Most (90%) stop spontaneously, but 25 to 85% will recur. The treatment of choice is endoscopic coagu- lation if the lesions can be identified (Fig. 15.4.2.7). Colectomy is reserved for those with repeated major haemorrhage. Benign anorectal disease Benign anorectal disease does present as lower gastrointestinal haemorrhage and a careful examination of the anorectum (with proctosigmoidoscopy) is imperative before initiating more invasive Table 15.4.2.4 Source of lower gastrointestinal haemorrhage Diagnosis % Diverticulosis 35 Colonic polyp or cancer 15 Benign anorectal conditions (including haemorrhoids) 10 Inflammatory bowel disease (including ulcerative colitis, Crohn’s, infective colitis) 15 Ischaemic colitis 5 Angiodysplasia (including angiomas and arteriovenous malfunctions) 10 Small bowel (including Meckel’s diverticulum) 1–2 Others (including rectal ulcer, postpolypectomy, radiation colitis, rectal varices) 10

 examinations. However, anorectal lesions are common and com- plete colonic evaluation is usually required even after identifying an anorectal source such as haemorrhoids. Iatrogenic haemorrhage The risk of haemorrhage after polypectomy is estimated to be be- tween 0.2 and 3%. Haemorrhage is usually immediate but may be delayed by up to 2 weeks. When identified, endoscopic haemostatic techniques are usually successful (injection of adrenaline, resnaring, recoagulating, placement of a ligature or clip). Clinical features History A

good history from the patient may give clues as to the cause of colo- rectal haemorrhage. Important points to explore include any prior history of bleeding; the exact nature of the bleeding, specifically the colour of the blood (bright red or altered), whether the blood is mixed with or separate from the stool, duration, and relationship to defeca- tion; and any associated change in bowel habit or mucus discharge. Bright red blood separate from the stool suggests an anorectal cause. Diarrhoea and mucous associated with darker blood mixed in with the stool suggests colitis or neoplasm. Bleeding from the small bowel or right colon can present with melaena. Other risk fac- tors which should be identified include a previous history of pelvic radiation which may suggest radiation colitis and personal history of liver cirrhosis with associated coagulopathy and varices. None of these clinical features, however, is absolutely diagnostic. It is also very important to explore comorbidities, which may signifi- cantly affect management, and drug usage, particularly of agents that affect coagulation or platelet function (aspirin, NSAIDs, and warfarin). Examination Aside from checking for evidence of hypovolaemia and shock, a full clinical examination should be performed, with particular focus on examination of the abdomen and rectum. Proctosigmoidoscopy is a simple bedside test and is mandatory to confirm or exclude ano- rectal causes of bleeding. Management A pathway for management of acute lower GIB is shown in Fig. 15.4.2.8. Resuscitation Immediate resuscitation is as for bleeding from the upper gastro- intestinal tract. In a patient with significant bleeding, it is essential that good intravenous access is established, even in the stable pa- tient. The patient should be catheterized, closely monitored, and if necessary transferred to a high-dependency setting. Further management Since most lower GIBs stop spontaneously, initial management should be conservative with transfusion and correction of clotting abnormalities. Once haemorrhage has ceased, bowel preparation and colonoscopy can be undertaken in a stable patient and with a much higher chance of detecting the pathological lesion (>90%). In contrast to upper GIB, there are no validated scoring systems for lower GIB, but the following risk factors are associated with se- vere/uncontrolled bleeding and/or death: • Haemodynamic compromise including heart rate >100 bpm, systolic blood pressure <115 mm Hg, syncope • Age • Multiple comorbidities—two or more doubles the risk of a severe bleed • Drugs that affect coagulation or platelet function (e.g. warfarin, NSAIDs, aspirin) • Second PR bleed within first 4 hours of admission • Inpatient lower GIB (mortality 23%) In the few patients in whom active colonic bleeding continues, in- vestigation to localize the source of the haemorrhage is indicated so that directed treatment can be administered in the form of endo- scopic therapy, interventional radiology, or surgery. Localization of bleeding Oesophagogastroduodenoscopy In a patient with significant lower GIB, it may be necessary to exclude an upper gastrointestinal source prior to evaluation of the colon. This is because massive upper GIB can masquerade as lower GIB, with up to 15% of patients presenting with haematochezia having an upper gastrointestinal source. An oesophagogastroduodenoscopy will ex- clude an upper gastrointestinal cause, although passing a nasogastric tube and checking the aspirate for blood was used historically. CT angiography CT angiography is now often the first line investigation for unstable LGIB with a sensitivity of 91–92%. This drops to 45% if the bleeding is intermittent. As in acute upper GIB, CT angiography can detect bleeding rates higher than 0.3 to 0.5 ml/min. It can be used to differentiate be- tween upper and lower GIB, and can determine the underlying cause. The main use of CT angiography is to provide anatomical information about the site of bleeding and variance in vascular anatomy, as well as confirming active bleeding prior to invasive mesenteric angiography. It is a diagnostic tool only and is not therapeutic. Whilst it is re- peatable, CT angiography involves the use of iv contrast which can Fig. 15.4.2.7 Angiodysplasia on the ileocaecal valve. From Marks D, Harbord M (2013). Emergencies in gastroenterology and hepatology. By permission of Oxford University Press.

SECTION 15 Gastroenterological disorders 2780 have a detrimental effect on renal function and is associated with a risk of allergic reactions and exposure to ionizing radiation. Colonoscopy Colonoscopy can be both diagnostic and therapeutic and has a reported 90% sensitivity and 87% positive predictive value in identifying lower gastrointestinal sources of bleeding. However it is often technically challenging and its use in acute LGIB is there- fore often limited. The use of bowel preparation is still debated. In studies, some using bowel preparation and some without, the causative lesion was identified in approximately three-quarters of patients. In a recent randomized controlled trial comparing urgent versus elective colonoscopy, there was little difference in outcomes. Treatment of bleeding in the right colon is associated with a higher perforation rate of 2.5%. Colonoscopy should be abandoned if massive haemorrhage obscures the diagnosis or severe mucosal or ischaemic colitis is encountered, as the risk of perforation in these cases is high. Control of active bleeding is an important therapeutic indi- cation for colonoscopy, with haemostasis achievable in many cases such as diverticular haemorrhage, angiodysplasia, and post polypectomy bleeding. In patients with postpolypectomy bleed ing where the approximate site of bleeding is known and can be endoscopically treated, colonoscopy should be considered as a first-line investigation as it has a reported technical success rate of 90 to 100%. Early OGD Interventional Radiology? Surgery Colonoscopy/ Flexible sigmoidoscopy Rigid sigmoidoscopy ± proctoscopy Mesenteric angiogram ± embolization Resuscitation

1. Adequate IV access - Large-bore peripheral venous access plus/minus central venous access
2. Urinary catheter
3. Balanced transfusion • Blood, platelets, and FFP as per local haematology guidelines • Activate massive transfusion protocol if necessary
4. Correct coagulation abnormalities
5. Monitoring - regular pulse, blood pressure, urine output and central venous pressure (if available). Transfer to high dependency setting if necessary
6. Drugs • Consider tranexamic acid
7. Early involvement of anaesthetists and surgeons if ongoing haemodynamic compromise
8. All patients should have a documented rebleed plan that should be updated after every intervention Stable Unstable Rebleeding Yes No Rebleeding Severe bleeding/ cannot be stabilized Rebleeding Fig. 15.4.2.8 Pathway for management of acute lower GIB. FFP, fresh frozen plasma; OGD, oesophagogastroduodenoscopy.

15.4.2 Gastrointestinal bleeding 2781 Control of bleeding can be accomplished with monopolar or bi- polar coagulation, heater probe, injection of various agents (such as adrenaline), use of Nd:YAG laser, and clipping. Endoscopic treatment of focal bleeding lesions in the colon is highly effective and safe, diminishing the need for surgical interven- tion. However, rebleeding rates have been reported of between 13 and 53%, and many patients require further treatments. Once LGIB has settled, urgent outpatient colonoscopy has an established role on determining the cause of the LGIB. Mesenteric angiography If colonoscopy fails to stop bleeding in acute lower GIB, selective mesenteric angiography is the second-line treatment. About 10 to 15% of cases of acute lower GIB will eventually require endovascular intervention. Mesenteric angiography can detect a rate of bleeding of 0.5 to 1.0 ml/min. The sensitivity of angiography reported in various studies ranges from 40 to 86%, although this may be increased by the use of so-called provocative measures, such as vasodilators, heparin, or thrombolytic agents. Once the site of haemorrhage is identified,

there is the therapeutic possibility of arterial selective embolization. Selective embolization using coil springs, polyvinyl alcohol foam, or gel foam into the most distal vessel results in high initial rates of haemostasis, and the rate of intestinal infarction is low. It is an especially good technique for patients with very high predicted operative mortality. Vasopressin infusion (via a catheter positioned in the artery sup- plying the site of bleeding)—now less frequently used—has considerable side effects including mesenteric thrombosis, intestinal infarction, myocardial ischaemia, hypertension, arrhythmias, and death. Nitroglycerine may be infused simultaneously to counteract the systemic effects of the drug. It can be used to treat bleeding from diffuse lesions or when superselective catheterization is not possible. Vasopressin infusion is successful in stopping bleeding in about 59 to 90% of patients, but is associated with a high rebleeding rate (36–43%). Nuclear scintigraphy Nuclear scintigraphy can be used to detect active haemorrhage but cannot determine the underlying cause. It is very sensitive and can detect bleeding rates of 0.1 ml/min and is therefore useful for inter- mittent bleeding. Sensitivity for scintigraphy is over 90%, although specificity is lower (76–95%), and overall accuracy reported in the literature is between 41 and 94%. Technetium-99m (99mTc)-labelled sulphur colloid can be used, which has the advantage of not requiring preparation but the dis- advantage of a very short half-life. If there is no active bleeding at the moment it is given, the test may be nondiagnostic, and its rapid enhancement of the liver and spleen can also obscure the diagnosis. A better method is the use of 99mTc-labelled red cells. Unfortunately, although sensitive, this is also very nonspecific and localizes lesions very poorly. Its half-life of up to 12 to 24 h means that, even if the bleeding is intermittent, the labelled blood cells can accumulate at the site of bleeding up to 24 h after injection and the patient can therefore be scanned multiple times within 24 hours. There is still controversy over the role of nuclear scintigraphy, and many surgeons are reluctant to proceed to a colectomy based solely on its results. It may be useful immediately before angiog- raphy (as scintigraphy can detect lower bleeding rates) to con- firm active haemorrhage before undertaking the more invasive procedure. Whenever there is massive, active haemorrhage, how- ever, this is unnecessary, and the patient should proceed directly to visceral angiography. Other aspects Surgery Surgery is a last resort in patients with LGIB as it is associated with a high morbidity and mortality. An emergency operation for lower gastrointestinal haemorrhage is required in 10 to 25% of patients. Indications for surgery include haemodynamic instability, clinical deterioration, transfusion requirements of greater than 6 units, and persistent or recurrent haemorrhage. In patients where the site of bleeding has been localized a directed segmental colectomy can be performed with an associated rebleed ing rate of 6% and a mortality of 4%. In patients where the bleeding site has not been localized a blind segmental colectomy is associated with much higher morbidity and a rebleeding rate of 75% and mor- tality of 50%. In these patients a subtotal colectomy could be con- sidered if deemed fit enough. Obscure bleeding In about 5% of cases the source of bleeding remains obscure, des- pite multiple attempts at localization. In many of these cases, the bleeding source is eventually found to be in the small bowel, with the commonest causes being angiodysplasia (accounting for 75% of episodes), Meckel’s diverticula, neoplasia, and Crohn’s disease. Additional investigations may include capsule endoscopy, small- bowel enteroscopy, or laparotomy with on-table enteroscopy. Capsule endoscopy Wireless capsule endoscopy has been shown to be useful in the in- vestigation of recurrent obscure gastrointestinal bleeds. It is the most sensitive modality for identifying lesions in the small bowel. The videocapsule is a self-contained unit, approximately 1 × 3 cm in size, and contains a miniature image-capturing system, battery, light source, and transmitter. Likely future developments Rebleeding still occurs in 20% of cases of upper GIB. New thera- peutic endoscopic techniques such as endoscopic suturing devices may reduce the need for salvage surgery. There

have been several promising case series on the use of high-dose barium enema for the treatment of ongoing diverticular bleeding, and this may prove to be useful, particularly in patients with multiple comorbidities and high anaesthetic risk. A small ran- domized controlled trial from Japan has shown a reduction in recur- rence of bleeding. FURTHER READING British Society of Gastroenterology Endoscopy Committee (2005). Non-variceal upper gastrointestinal haemorrhage: guidelines. Gut, 51 Suppl IV, iv1–6. Calvet X, et al. (2004). Addition of a second endoscopic treatment following epinephrine injection improves outcome in high-risk bleeding ulcers. Gastroenterology, 126, 41–50.

 Hoedema RE, Luchtefeld MA (2005). The management of lower gastrointestinal hemorrhage. Dis Colon Rectum, 48, 2010–24. Jensen DM, Machicado GA (2005). Endoscopic hemostasis of ulcer hemorrhage with injection, thermal, and combination methods. Tech Gastroint Endosc, 7, 124–31. Jensen DM, et al. (1999). Prospective effectiveness study of an urgent endoscopic approach to diagnosis and treatment of patients hospi- talized with recurrent hematochezia. Gastrointest Endosc, 49, 331. Kahi CJ, et al. (2005). Endoscopic therapy versus medical therapy for bleeding peptic ulcer with adherent clot: a meta- analysis. Gastroenterology, 129, 855–62. Keeling D, et al. (2011). Guidelines on oral anticoagulation with warfarin—fourth edition Br J Hameatol, 154, 311–24. Khorrani GJP, et al. (2004). H. pylori eradication therapy vs. antisecretory non-eradication therapy (with or without long-term maintenance antisecretory therapy) for the prevention of recurrent bleeding from peptic ulcer. Cochrane Database Syst Rev, 2, CD004062. Martins NB, Wassef W (2006). Upper gastrointestinal bleeding. Curr Opin Gastroenterol, 22, 612–19. National Confidential Enquiry into Patient Outcome and Death (NCEPOD) (2015). Time to get control? A review of the care received by patients who had a severe gastrointestinal haemorrhage. London: NCEPOD. National Institute for Health and Care Excellence (2012). Acute upper gastrointestinal bleeding in over 16s: management. Clinical guideline. (Last updated: August 2016.) https://www.nice.org.uk/guidance/ cg141 Palmer KR, Church NI (1999). Therapeutic endoscopy for upper gastrointestinal bleeding. Cont Med Ed J Gastroenterol Hepatol Nutr, 2, 75–8. Ramaswamy RS, et al. (2014). Role of interventional radiology I the management of acture gastrointestinal bleeding. World J Radiol, 6, 82–92. Rockall TA, et al. (1995). Incidence of and mortality from acute upper gastrointestinal haemorrhage in the United Kingdom. BMJ, 311, 222–6. Rockall TA, et al. (1996). Risk assessment following acute upper gastrointestinal haemorrhage. Gut, 38, 316–21. Scottish Intercollegiate Guidelines Network (SIGN) (2008). 105: Management of acute upper and lower gastrointestinal bleeding. Edinburgh: SIGN. Vernava AM, et al. (1997). Lower gastrointestinal bleeding. Dis Colon Rectum, 40, 846–58. Williams SG, Westaby D (1994). Management of variceal haemor- rhage. BMJ, 308, 1213–17.

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