# Oesophageal emergencies

Richard H Hardwick

**Abstract**

Oesophageal emergencies are relatively rare but their management leaves little room for error. This article explains the aetiology, diagnosis and management of oesophageal bleeding, obstruction, perforation and caustic injury. The essential role of early flexible endoscopy and specialist multi-disciplinary care is emphasized.

**Keywords** Boerhaave's syndrome; dysphagia; oesophageal emergencies; varices

Oesophageal emergencies are relatively rare compared to other parts of the gastrointestinal tract. However, they are often life-threatening, especially if misdiagnosed or poorly managed. They fall into four broad groups: bleeding, obstruction, perforation and caustic injury. A detailed history should be obtained from the patient or a witness whenever possible as this will often suggest the likely cause of the emergency. For example, repeated vomiting and retching followed by a small haematemesis in a well patient is likely to be due to a Mallory–Weiss tear. In contrast, the shocked patient who has collapsed after a vomit may have an oesophageal rupture (Boerhaave's syndrome). Flexible endoscopy has revolutionized our ability to diagnose and treat many oesophageal conditions and plays a central role in the management of these patients.[1]

## Bleeding (Table 1)

The sight of a patient vomiting fresh blood faster than it can be infused intravenously can be dramatic. Luckily, however, this scenario is quite rare. More commonly, there is a history of vomiting blood, maybe a bowl of partially clotted blood beside the patient and evidence of variable degrees of hypovolaemia.

***Richard H Hardwick*** ***MD FRCS*** *is Consultant Upper GI surgeon at the Cambridge Oesophago-gastric Centre, Addenbrooke's Hospital, Cambridge, UK. He was an undergraduate at St Thomas' Hospital, London and trained in gastrointestinal surgery as lecturer in Bristol and then Senior Lecturer in Cardiff before moving to Cambridge. His research interests include surgery for carcinoma of the gastro-oesophageal junction, cell biology of Barrett's cancers and novel techniques for oesophageal surveillance. Competing interests: none declared.*

**Causes of oesophageal bleeding**

- Varices
- Ulcer/oesophagitis
- Mallory–Weiss tear
- Tumour

**Table 1**

### History and examination

A good history is vital. This should include whether the patient is known to have liver disease or portal hypertension, whether this has happened before, and if so where and how was it managed, and whether the patient is anti-coagulated.

Some basic observations are needed, including:

- oxygen saturation
- blood pressure
- pulse
- conscious level.

It is important to remember that young patients will maintain their blood pressure until significant intravascular volume loss has occurred and then suddenly decompensate. Elderly patients are less tolerant of hypovolaemia, their blood pressure will sag much earlier and their risk of death from bleeding is much higher than younger patients. The pulse is usually a good indicator of volume loss except in those who are taking β-blockers. Hourly urine output measurements are a useful secondary indicator of the state of hydration but trends over time of pulse rate are the most practically useful measurement. Measurements of central venous pressure (CVP) can also be useful but trends, again, are more helpful than absolute numbers (plus, the site of CVP lines must be taken into account).

### General management

As with all acutely ill patients, the 'ABC' takes priority over everything else. 100% oxygen by a re-breathing mask, protection of the airway and insertion of two large-bore intravenous cannulas, one in each arm, are essential. A sample of blood should be sent for urgent assessment of haemoglobin, clotting, kidney and liver function, and for cross-matching. Clearly, the patient who answers questions about what has happened rarely has an airway problem. A calm and reassuring bedside manner will help give the patient confidence during a frightening experience. Hartmann's solution or normal saline should then be given intravenously as a bolus (10–20 ml/kg). Rockall scoring (page 159, this issue) is useful for predicting the urgency of endoscopy and the likelihood of re-bleeding.[2] Some patients need scoping immediately, preferably in an operating theatre with anaesthetist, endoscopist and surgeon all in attendance. For others, an endoscopy will be needed early the next morning in the endoscopy department. Endoscoping a patient out of hours on the ward with inexperienced assistants is rarely successful and should be avoided.

**Varices:** as soon as bleeding varices are diagnosed or suspected, vasoactive therapy should be started (Table 2). A Cochrane meta-analysis in 2002 of trials including 1146 patients showed

### Management protocol for bleeding varices

- Resuscitate
- Vasoactive therapy; octreotide 50 μg i.v. bolus followed by 50 μg/hour i.v. for 3 days **or** terlipressin
- Intravenous antibiotics (ciprofloxacin 200 mg b.d.)
- Urgent endoscopy +/– band ligation or injection sclerotherapy (up to two attempts)
- Balloon tamponade or transjugular intrahepatic portosystemic shunt (TIPS) for uncontrolled bleeding

**Table 2**

that 83% of variceal bleeding will be controlled by vasoactive drugs alone.[3] However, there is some evidence that the combination of medical and endoscopic therapy, such as banding or sclerotherapy, may be even more successful,[4] although there is only a marginal positive impact of dual therapy on mortality.[5] The use of balloons to tamponade the varices should be reserved for patients who continue to bleed despite vasoactive drugs and endoscopic therapy as they often cause complications. Where available, insertion of a transjugular intrahepatic portosystemic shunt (TIPS) at a specialist centre may be necessary for patients who continue to bleed.[6]

**Ulcer/oesophagitis/Mallory–Weiss tear:** endoscopy may reveal severe oesophagitis with or without ulceration as the cause of the haematemesis. Occasionally, there may be therapeutic opportunities at endoscopy when, for instance, a visible vessel is seen which can be controlled by adrenaline injections with or without the application of a heater probe. Bleeding from a mechanically induced mucosal tear at the gastro-oesophageal junction (Mallory Weiss) can similarly be controlled with adrenaline injections or, if persistent, an endoscopic clip.[7] Although the evidence for their use is contradictory,[8] intravenous proton pump inhibitors (PPIs) should probably be given for 72 hours and the patient closely monitored for signs of on-going bleeding or re-bleeding. Oral free fluids are usually safe. After stabilization, high-dose oral PPIs should be given. For patients with an oesophageal ulcer, a repeat endoscopy should be arranged in two to four weeks for evaluation of healing and for biopsies to be taken to exclude malignancy.

## Obstruction

A food bolus obstruction is the commonest cause of sudden oesophageal occlusion and a history of the events leading up to presentation will usually raise this as the likely diagnosis. Americans refer to the 'steakhouse syndrome' when a patient develops sudden-onset complete dysphagia while eating red meat. There may be a preceding history of reflux symptoms or oesophagogastric surgery, either of which may have resulted in the development of a benign stricture. A less common cause of oesophageal obstruction is ingestion and subsequent impaction of foreign bodies, ranging from false teeth to drawing-pins.

### Investigation of sudden-onset complete dysphagia

If a foreign body impaction is suspected, the patient should have a chest X-ray. Food bolus obstruction may resolve spontaneously but, regardless of the likely aetiology, an urgent flexible endoscopy should be arranged for all patients. In the meantime, administer Buscopan (Boehringer Ingelheim; hyoscine-N-butylbromide) 20–40 mg intravenously (provided there are no cardiac contraindications); this may relax the oesophagus sufficiently to allow the bolus to pass. This can usually be done safely in the endoscopy department, but foreign body impaction is probably better tackled in an operating theatre where airway management can be more easily controlled. Food boluses can normally be pushed through into the stomach. Foreign bodies may require ingenious endoscopic solutions to aid their disimpaction, utilizing grasping instruments and snares.[9] An overtube should be employed for the safe removal of the offending item. Rigid oesophagoscopy is occasionally required under general anaesthesia to get a proper grip on the foreign body but this is rare. Close liaison between upper gastrointestinal surgeons, ENT, gastroenterology and anaesthetists is needed to deal with the more complex case. Only in the unlikely event of failure to remove the object should open surgery be considered by an experienced oesophageal surgeon. If all the relevant personnel are not available at the admitting hospital, the patient should be transferred to a specialist centre. Following successful removal of an oesophageal foreign body the patient should be kept nil by mouth until a contrast swallow has excluded perforation. Oesophageal strictures should be biopsied endoscopically as soon as possible to exclude malignancy and gastroesophageal reflux treated aggressively with PPIs.

## Oesophageal perforation (Table 3)

### Diagnosis

This may be immediately obvious at the time of an oesophageal dilatation, or not become apparent for days while the patient is on a ventilator in intensive care. The usual reason for perforation being missed is that it has not been considered as a possible diagnosis. Prompt and correct treatment will usually result in a live patient. Oesophageal perforation should be suspected after endoscopy if the patient complains of severe retrosternal pain which may radiate into the neck. Surgical emphysema is a give-away but may not be present. An urgent water-soluble contrast swallow should be performed on all patients with suspected oesophageal perforation. Alternatively, an unconscious patient may require a CT scan to look for mediastinal air. A Senior Gastrointestinal Radiologist should review all images where there is significant clinical concern above a perforation even if the study appears normal. Small leaks are easily missed.

### Causes of oesophageal perforation

**Iatrogenic**
- Stricture/tumour dilation
- Achalsia balloon dilation
- Scope into a diverticulum
- Post surgical (anastomotic leakage)

**Spontaneous (Boerhaave)**

**Benign ulcer**

**Foreign body**

**Table 3**

### Management of oesophageal perforation

Oesophageal perforations are best managed by a specialist multi-disciplinary team consisting of an oesophageal surgeon, a gastrointestinal interventional radiologist, a gastroenterologist, a dietitian, a microbiologist and an intensivist.

### Perforations following endoscopy

Here, the size of the defect, the cause of the injury and the co-morbidities of the patient will dictate management. Balloon dilation of a benign stricture or for achalasia resulting in a small leak confined to the mediastinum can usually be managed conservatively with nil by mouth, intravenous broad-spectrum antibiotics, anti-fungal agents, and parenteral nutrition, although enteral nutrition via a naso-jejunal finebore feeding tube should be considered whenever technically feasible. The same is true for a perforated oesophageal tumour; although emergency oesophagectomy may be feasible (Figure 1), it is rarely curative and non-operative management is preferable.[10] If the patient remains well, a repeat swallow is done at one week and if the leak has sealed, oral intake slowly restarted. The development of clinical sepsis should prompt a computed tomography (CT) scan to look for mediastinal sepsis and, if found, it should be drained percutaneously under CT guidance. Persistent leakage after 10 days should now be managed by the insertion of a removable plastic stent as there is good evidence they work.[11]

A large oesophageal tear resulting in pleural contamination normally requires immediate surgery (Figure 2). If diagnosed early, it may be possible to perform a primary repair of the oesophagus. If not, the perforation should be closed over a T-tube to create a controlled fistula, large pleural and mediastinal drains placed, a venting gastrostomy placed, and a feeding jejunostomy inserted for enteral feeding.[12] Perforation of a cervical oesophageal diverticulum can usually be managed conservatively although failure to manage resulting cervical sepsis adequately can be fatal.

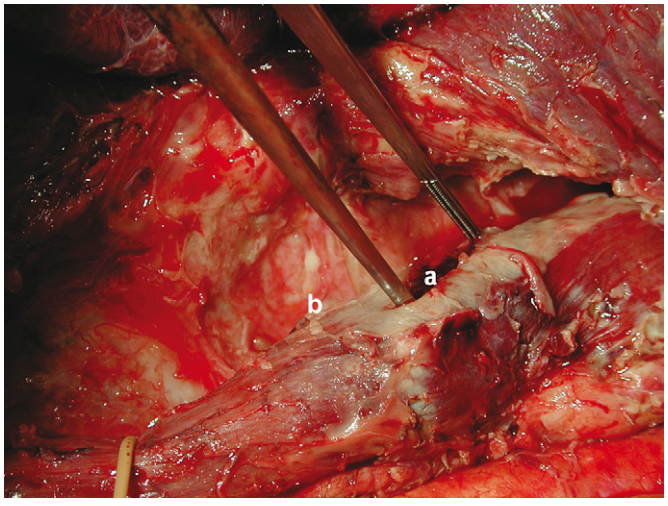


**Figure 1** This picture shows a perforated oesophageal tumour **(a)** with associated mediastinal abscess **(b)**. The patient presented with retrosternal pain and a swinging fever. Resection and reconstruction was performed in two stages, three months apart but the patient died of recurrence within 6 months of the initial procedure.

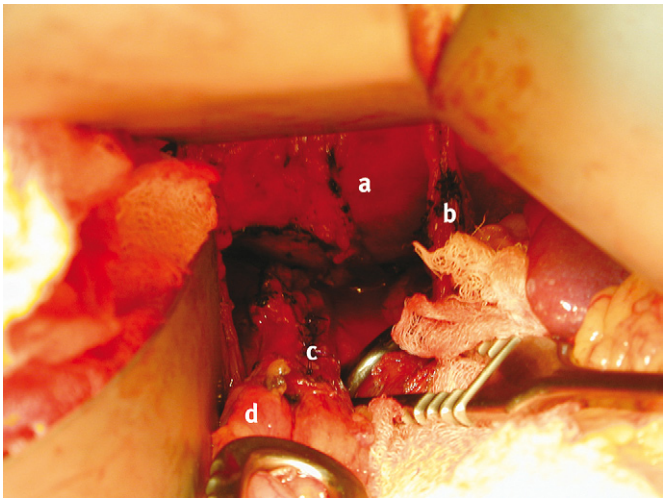


**Figure 2** View of the distal oesophagus **(d)** showing a trans-hiatal primary repair of an oesophageal rupture following balloon dilatation for achalasia. The interrupted sutures closing the mucosal tear are just visible **(c)**. The hiatus **(b)** has been widely opened and the heart **(a)** retracted anteriorly to gain access to the rupture.

### Spontaneous perforations

Boerhaave's syndrome occurs when a patient vomits violently against a closed lower oesophageal sphincter.[13] The high intra-luminal pressure developed bursts the lower part of the oesophagus, commonly on its left side, spilling food debris into the mediastinum and chest cavity. Sepsis syndrome sets in soon afterwards. The patient may remember vomiting prior to becoming unwell but not always, particularly if they have been drinking alcohol. Shortness of breath and hypoxia result from pleural effusion and sepsis, causing metabolic acidosis. Chest and epigastric pain is common. A chest X-ray may show an effusion and free mediastinal air or be completely normal. Contrast radiology is essential.

Unlike iatrogenic perforations where the stomach is empty, conservative management of Boerhaave's syndrome is rarely successful due to the degree of mediastinal contamination. The best chance of survival for most patients is early diagnosis and surgery in a specialist centre.[12] Primary surgical repair may be possible but closure of the defect around a T-tube to create a controlled fistula is more likely to be appropriate. Occasionally with late presentation, the injured oesophagus takes on the characteristic of wet blotting paper, whereupon it is necessary to perform emergency oesophagectomy with proximal diverting oesophagostomy and insertion of a feeding gastrostomy.[14] Reconstruction is performed months later if the patient survives.

Oesophageal ulcers rarely perforate and, if they do, chronic mediastinal inflammation around the ulcer usually prevents mediastinitis by helping to wall off the perforation. Non-steroidal inflammatory drugs (NSAIDs) have been implicated in some cases of oesophageal ulcer, particularly if there is structuring and the tablets lodge in the oesophagus.

Anastomotic leakage post oesophageal surgery is one of the most feared complications of upper GI surgery.[15] As with other causes of leakage from the oesophagus, early diagnosis, the size of the defect and the underlying cause are all important in deciding

management. A small anastomotic leak on postoperative day seven in a well patient will be managed conservatively whereas a large leak on day three will almost certainly need further surgery or a temporary plastic stent to seal the defect. Management of these complications is highly complex and should be performed in a specialist centre, preferably where the original surgery was undertaken.

Perforations resulting from the removal of foreign bodies from the oesophagus can nearly always be managed conservatively.

## Caustic injury

Rarely seen in the UK now, this is still a very common cause of oesophageal emergencies in the developing world. Some are due to inadvertent ingestion of chemicals stored in old drink bottles, others are attempted suicides. After resuscitation, early endoscopy is important to grade the degree of oesophageal and gastric injury as this correlates well with the final outcome. Early surgery for the resulting strictures seems to give a better outcome than the traditional 'watch and see' policy.[16] ◆


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