If you want to evaluate your teaching style and delivery, peers can be a useful source of feedback:

- Ask a colleague to observe part or all of a lecture and provide feedback afterwards. It is important to inform the observer what aspects of the lecturing process you want evaluated—for example, clarity, logical flow, effectiveness of the media used
- Videotape the lecture for private viewing, and arrange a joint viewing with a colleague later.

Lectures are still a common teaching method in both undergraduate and postgraduate medical education. Their continued popularity is due to the fact that they represent an effective and efficient means of teaching new concepts and knowledge. This article has emphasised the importance of good lecture planning and of the inclusion of student interaction to ensure effective learning.

Please rate the lecturer on the following items

	Strongly agree	Slightly agree	Slightly disagree	Strongly disagree
Was enthusiastic				
Was clearly audible				
Seemed confident				
Gave clear explanations				
Encouraged participation				

Example of an evaluation form focusing on the lecturer rather than the lecture. Adapted from Brown et al, 2001 (see "Recommended reading" box)

Recommended reading

- Newble DI, Cannon R. *A handbook for medical teachers.* 4th ed. Dordrecht, Netherlands: Kluwer Academic, 2001.
- Gibbs G, Habeshaw T. *Preparing to teach.* Bristol: Technical and Educational Services, 1989.
- Bligh DA. *What's the use of lectures?* San Francisco: Jossey-Bass, 2000.
- Brown G, Manogue M. AMEE medical education guide No 22: refreshing lecturing: a guide for lecturers. *Medical Teacher* 2001;23:231-44.

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Lesson of the week

Epistaxis: an overlooked cause of massive haematemesis in cirrhosis

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Epistaxis should be considered as a cause of massive haematemesis in patients with cirrhosis

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The national audit of acute upper gastrointestinal haemorrhage reported an overall incidence of acute upper gastrointestinal haemorrhage in the United Kingdom of 103 cases per 100 000 adults a year. Varices have been identified as the source of blood loss in 8% of patients aged less than 60 years, and mortality among these patients is four times the overall mortality for the age group in patients with haematemesis.[1]

The most dramatic presentations often occur in patients with chronic liver disease. Variceal bleeding is a life threatening complication of cirrhosis, and survival is closely related to failure to control haemorrhage or early rebleeding, which occurs in as many as 50% of patients.[2] In cases of suspected variceal bleeding, immediate treatment with agents such as terlipressin or octreotide is recommended, followed within 12 hours by upper gastrointestinal endoscopy, which is essential for accurate diagnosis and allows variceal sclerotherapy or band ligation.[3] Endoscopic diagnosis can be difficult when views are obscured by blood. Nevertheless, a diagnosis of variceal haemorrhage is acceptable when a venous spurt is seen or there is fresh blood in the lower oesophagus in the presence of varices. In about half of cases there is no active bleeding; variceal haemorrhage is indicated by the presence of a "white nipple sign" (a plug of platelet fibrin on a varix) or when varices are the only lesion identified.[4,5]

We describe two patients with alcoholic liver disease and haematemesis whose bleeding was not controlled by endoscopic treatment. Delayed diagnosis of severe epistaxis led to prolonged haemodynamic instability and further decompensation.

Case reports

Case 1

A 45 year old woman with alcohol induced cirrhosis (Child's-Pugh class C) and idiopathic thrombocytopenic purpura presented with shock after fresh haematemesis. On admission she had a haemoglobin concentration of 24 g/l, platelets 10×10^9/l, and prothrombin time 16.0 s (control 10.0 s). She was resuscitated with transfusion of whole blood, fresh frozen plasma, and platelets. Variceal bleeding was suspected, and she was given an infusion of octreotide.

Gastroscopy showed a large volume of fresh blood restricting the view of the oesophagus and stomach. No source of bleeding was identified. The patient's history indicated that variceal bleeding was the most likely cause of blood loss, and a Sengstaken-Blakemore tube was inserted.

Blood loss continued for several hours, and the patient remained haemodynamically unstable. Repeated suction of the oral cavity was necessary, prompting examination of the nasopharynx, which showed an arterial bleeding point in the left nasal cavity. Bilateral nasal packs were inserted to achieve haemostasis. The patient's condition deteriorated as a consequence of aspiration pneumonia and renal failure, and she died.

Case 2

A 51 year old man with alcohol induced cirrhosis (Child's-Pugh class B) presented with large volume haematemesis. On admission the patient had a haemoglobin concentration of 88 g/l, platelets 116 × 10^9/l, and prothrombin time 15.6 s. He was resuscitated with transfusion of whole blood and fresh frozen plasma. Gastroscopy within six hours of admission showed medium sized oesophageal varices, with fresh blood along the full length of the oesophagus, which was aspirated. Variceal band ligation was undertaken, as no other source of blood loss had been identified.

When the endoscope was withdrawn, fresh blood was seen in the oropharynx, and temporary nasal packs were inserted. Formal examination by an ear, nose, and throat specialist confirmed an active bleeding point in the right posterior nasal cavity, and fresh packs were placed. These were removed after 72 hours, and bleeding did not recur. Alcohol withdrawal symptoms and a left basal pneumonia complicated his recovery. He was discharged home after seven days and had subsequent follow up with an ear, nose, and throat specialist for further treatment of the nasopharyngeal bleeding point.

Discussion

In both patients the identification of the source of haemorrhage was delayed, leading to life threatening blood loss and further decompensation. Published case series suggest that at endoscopy 26-56% of cases of upper gastrointestinal bleeding in patients with cirrhosis will have a non-variceal source, such as peptic ulceration or portal hypertensive gastropathy.[6] Furthermore, even with emergency endoscopy, active variceal bleeding (blood spurting from a ruptured varix) is seen in about 20-30% of patients with a final diagnosis of variceal bleeding. Epistaxis can be added to this list, as these cases and one earlier report illustrate.[7] Haematemesis has been reported after nasal trauma or nosebleed in otherwise well patients.[8] We believe that the bleeding originated from abnormal blood vessels within the nasopharynx (with a similar aetiology to spider naevi), because haemodynamic stability was achieved only on nasal packing and not after variceal treatment. The blood loss was exacerbated by coexisting coagulopathy and thrombocytopenia in both patients.

If a patient is haemorrhaging and varices are suspected, the passage of a Sengstaken-Blakemore tube is recommended.[9] When bleeding is not controlled by this manoeuvre, the usual cause is fundal bleeding, and angiography is performed. We suggest that a careful examination of the mouth and nasopharynx may show a bleeding point that has hitherto gone unrecognised by the endoscopist and unreported by the patient. Such bleeding is easily treated. The continued aspiration of blood from the oesophageal port of a Sengstaken-Blakemore tube, despite inflation of the oesophageal balloon, should alert the clinician to the possibility of epistaxis.

The most consistently reported risk factors associated with a fatal outcome in patients with cirrhosis who are admitted to hospital are a Child's-Pugh classification or its components, impaired renal function, older age, and active alcohol abuse.[10 11] Many of these factors were evident in the cases described here. Since large volume blood loss is poorly tolerated in these patients, if control of bleeding is not achieved by standard treatment the source of blood loss should be rapidly re-evaluated.

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Competing interests: None declared.

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Endpiece

Every knock

One evil in old age is that, as your time is come, you think every little illness is the beginning of the end. When a man expects to be arrested, every knock at the door is an alarm.

Sydney Smith (1771-1845), English clergyman, essayist, and editor. In a letter to Sir Wilmot-Horton, 8 February 1836

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