Oesophago-aortic fistula

A. C. V. MONTGOMERY*  
F.R.C.S.

A. S. CHILVERS†  
F.R.C.S.

* Breast Unit, Royal Marsden Hospital, Fulham Road, London, S.W.3, and † St Helier Hospital, Carshalton, Surrey

Summary
This report of oesophageal peptic ulceration communicating with the thoracic aorta, successfully treated, illustrates the importance of prompt recognition of major bleeding and early repair.

Case report
A 19-year-old boy was transferred from a long-stay hospital for the handicapped, after having passed melaena stools, vomited bright blood and having become shocked. In the past 6 months he had been noted to be anaemic and barium examination had been reported as showing a hiatus hernia and a duodenal ulcer.

On admission, the patient was vomiting bright red blood profusely and passing melaena stools. He was shocked with a pulse of 120/min and a BP of 60 mmHg systolic. Despite being transfused 6 units of blood, his condition improved only temporarily, his hypotension and tachycardia recurring with copious quantities of fresh blood being vomited.

The patient was transferred direct to the operating theatre. At laparotomy, the stomach was distended with more than 2 litres of clotted blood. Gastrotomy and digital examination through the cardia revealed an ulcer approximately 10 cm above the hiatus. The aortic wall was palpable through the ulcer. Haemorrhage was controlled by digital pressure whilst the left chest was opened.

Dense inflammatory adhesions were found between the descending thoracic aorta and the lower third of the oesophagus. The aorta was freed and clamped, permitting repair of the aorta with 3/0 silk. The oesophagus was closed with catgut and wrapped posteriorly with Dacron mesh.

The patient recovered well, with only a superficial wound infection.

At follow-up 3 years later he remained asymptomatic and his blood count was normal. Barium swallow examination at that time was clear.

Comment
Peptic ulceration commonly occurs in the oesophagus and is well recognized as perforating into the pleural cavity (Stillwell, 1978; Hancock and Barnett, 1974) and pericardium (Monro et al., 1974). However, severe bleeding from such ulcers is a rarity and direct communication with the normal aorta has not been described previously.

Ulceration communicating with the aorta at vascular suture lines has been documented (Curry and Anderson, 1974), the stomach usually being protected by the retroperitoneal tissues from direct communication.

Primary aortitis, such as tuberculosis (Bigge and Rothnie, 1974), has also been described as causing aorto-oesophageal communication and similar massive haematemesis. Foreign bodies may also give rise to haematemesis by damaging the oesophagus (Laforet, 1973).

Aorto-intestinal fistulae are recognized by the copious quantities of bright blood vomited and the inability to maintain the circulation adequately by transfusion. Treatment consists of early surgery to control blood loss, when the circulatory volume can be maintained and repair and recovery effected.

Acknowledgments
Our thanks to B. W. Wells, F.R.C.S., Consultant Surgeon, St Helier Hospital, for permission to report this case.

References