

# Acute upper airway obstruction due to displacement of a Sengstaken–Blakemore tube

doi: 10.1017/S0265021507002852

## EDITOR:

A 66-yr-old white male, with a known history of liver cirrhosis and associated portal hypertension, was admitted to our high-dependency unit (HDU) following oesophagogastroduodenoscopy for massive haemorrhage. The endoscopy had been technically difficult due to bleeding obscuring the visual field, however grade III lower oesophageal varices were seen but were not obviously bleeding. The bleeding appeared to be from an unidentified site below the gastro-oesophageal junction, possibly from fundal varices. Given the technical difficulties and the patient's condition, a Sengstaken–Blakemore tube (SBT) had been inserted nasally to gain control of the bleeding. The gastric balloon was inflated with 300 mL of air and 500 g of traction applied. A chest radiograph was performed on arrival in HDU and it showed the gastric balloon of the SBT correctly positioned below the diaphragm. The patient was started on terlipressin and given antibiotic prophylaxis according to local protocols.

Approximately 16 h after admission, the patient became agitated, complaining of retrosternal pain. On examination he was tachypnoeic with normal percussion but very quiet breath sounds bilaterally. His saturations had dropped to 80% despite high-flow oxygen. He was tachycardic but otherwise haemodynamically stable with no further evidence of haematemesis. Arterial blood gases showed a type I respiratory failure. In view of his poor oxygenation, a rapid sequence induction was undertaken and an 8.0 mm cuffed tracheal tube inserted. Although his oxygenation rapidly improved to 95%, his airway pressures were noted to be high with poor air entry bilaterally. An urgent chest radiograph was performed and demonstrated that the gastric balloon had migrated into the oesophagus and was now located between the levels of the carina and clavicular heads, causing extrinsic tracheal compression (Fig. 1). Deflation of the gastric balloon produced an immediate reduction in airway pressures and improvement in ventilation.

A repeat endoscopy was carried out the next day confirming distal oesophageal and fundal varices

(type 2 gastro-oesophageal varices). These were successfully banded. The patient spent a further 8 days in the ICU which were complicated by acute respiratory distress syndrome and another episode of massive haematemesis. Despite ongoing active treatment, the patient died on day 10 due to multi-organ failure.

The use of oesophageal tamponade to control bleeding varices was first described in 1930 by Westpal [1]. In 1950, Sengstaken and Blakemore developed a tube with an oesophageal and a gastric balloon enabling local pressure to be applied to both oesophageal and fundal varices [2]. In cases of massive haemorrhage, and when endoscopic therapy is technically difficult, balloon tamponade remains highly effective in controlling acute bleeding and can be life saving [3]. The SBT controls bleeding in around 90% of patients, with an incidence of rebleeding on removal of the tube being reported at around 50% [3]. Despite the efficacy of the SBT, complications associated with its use are high. The most common complications are aspiration and pressure necrosis of the mucosa, occurring in up to 15–20% of patients [4]. Rare major complications result from incorrect tube placement and from tube



**Figure 1.**

*Chest radiograph showing the displaced gastric balloon now positioned between the level of the carina and the clavicular heads. The endotracheal tube can be seen above the level of the gastric balloon. In this position, the SBT is causing extrinsic tracheal compression resulting in upper airway obstruction.*

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Accepted for publication 14 September 2007 EJA 4710  
First published online 22 October 2007

migration, often resulting in oesophageal rupture [5]. Extrinsic airway compression of the main bronchi has once been reported [6].

The correct placement of the SBT is vital in reducing the risk of major complications, and the use of a chest radiograph to confirm the position of the SBT prior to maximal inflation of the gastric balloon has been suggested [5,7]. The gastric balloon should be positioned retracted against the cardia. Although not performed prior to maximal inflation in our case, an early chest radiograph confirmed the correct position of the gastric balloon. Once correct positioning has been confirmed, the SBT is commonly placed on traction with 500–1000 g weight (500 mL of crystalloid in our case). The use of traction does place the SBT at risk of migration. In our case, the gastric balloon migrated into the oesophagus, lodging behind the trachea, possibly being held in place by the complete cricoid ring. Life-threatening extrinsic tracheal compression thus ensued. This was evidenced by the rapid improvement in tidal volume and reduction in airway pressures on removal of the SBT. To avoid the use of traction, and its associated risks of migration, many clinicians now prefer to simply tape the SBT in place once the correct position of the gastric balloon has been confirmed by chest radiograph. Whether the SBT is held under traction or is taped in position, it is important that the correct position of the SBT at the lips or nose is clearly marked on the tube. This simple, but commonly overlooked, step allows any future dis-

placement to be easily recognized. In our case, displacement of the SBT was not immediately recognized but would have been greatly facilitated had the correct position of the SBT been clearly marked at the nose.

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## The use of esmolol to treat systolic anterior motion of the mitral valve after mitral valve repair

doi: 10.1017/S0265021507002876

### EDITOR:

Systolic anterior motion of the mitral valve causing left ventricular outflow tract obstruction is common after mitral valve repair, but only rarely necessitates immediate additional surgical intervention. Although multiple surgical techniques have been proposed to prevent systolic anterior motion [1], it

remains a problem after mitral valve repair. The degree of systolic anterior motion extends along a continuous spectrum from minor chordal-only systolic anterior motion to its most severe form with permanent left ventricular outflow tract obstruction and moderate-to-severe mitral regurgitation.

The management of systolic anterior motion in the operative room remains controversial. Even if some authors advocate immediate surgical correction [2], most patients with systolic anterior motion can be successfully managed with medical treatment [3] (increasing systemic vascular resistance, augmenting intravascular volume and administering

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Accepted for publication 14 September 2007 EJA 4720  
First published online 22 October 2007