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OPINION

Sengstaken–Blakemore tube in critical upper gastrointestinal bleeding: Implications for aeromedical retrieval

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Abstract

Sengstaken–Blakemore tubes (SBTs) are rarely used in Australia, because of improved access to endoscopy and interventional radiology, as well as overall lower rate of variceal haemorrhage from improvements in primary prophylaxis. SBT's use is associated with significant rate of serious complications, such as oesophageal perforation, mucosal necrosis, aspiration pneumonia and respiratory compromise secondary to external compression of the trachea. As such, SBT is currently only recommended for use in life-threatening variceal haemorrhage, where endoscopic, embolization and pharmacologic therapy have been unsuccessful or are unavailable. No data exist for its use in Australasia but one area that it could be indicated is for hemodynamically unstable patients in remote setting, where long transfer times often means delayed access to endoscopy. We present a case of SBT insertion in retrieval medicine and discuss placement in the management of an unstable upper gastrointestinal bleed, complicating factors such as lack of radiology to confirm balloon position, the impact of flight altitude on balloon pressures, the maintenance of traction in flight and logistics of long flight times across the state of Queensland. This is the first case report of SBT use in the Australian aeromedical environment. It is also the first one where SBT has been used for duodenal bleeding, although the source of bleeding was unknown prior to insertion.

Key words: *minnesota tube, Sengstaken-Blakemore, upper gastrointestinal bleed, variceal bleed.*

Case report

A LifeFlight Retrieval Medicine (LRM) team was tasked by Queensland Health to manage a 69-year-old man with upper gastrointestinal bleeding located in a rural facility, 250 km from a major tertiary referral centre. The patient presented with hypovolaemic shock following a 24 h history of melena. There were no risk factors for liver disease and he was not on antiplatelet or anticoagulant agents. The local team had administered 80 mg i.v. pantoprazole, 500 mL Hartmann's and commenced packed red blood cell (PRBC) transfusion (2 units given). On LRM examination, he was pale with a heart rate of 140/min, a BP of 70/40 mmHg, respiratory rate of 35/min and oxygen saturation of 94% on 2 L/min nasal prongs. Palpation of the abdomen revealed epigastric tenderness and melena was present on stool inspection. A venous blood gas showed metabolic acidosis with a lactate of 5.4 mmol/L and haemoglobin of 69 g/L.

Blood transfusion was continued, 1 g of tranexamic acid and 1 g of calcium chloride were administered to optimise coagulation. During assessment, the patient suffered a massive hematemesis, followed by aspiration and hypoxemia, which necessitated intubation. Hematemesis continued following intubation and haemorrhagic shock progressed to a state of PEA. Bedside US showed good cardiac contractility. CPR was commenced whereas maintaining the priority to be blood transfusion.

At that stage, 11 of the 12 available PRBC units had been used. No coagulation profile testing, viscoelastic haemostatic assays, fresh frozen plasma, cryoprecipitate, fibrinogen concentrate or endoscopic services were available. Flight time to the nearest hospital with gastroenterology service was 50 min. The LRM team inserted a Sengstaken–Blakemore tube (SBT) which was sourced from the rural hospital, and return of spontaneous circulation (ROSC) was achieved. Traction to the SBT was applied and maintained with adhesive taped to the patient's head and vacuum mattress.

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In flight, the patient received the last available unit of PRBC and arrived at the tertiary centre with a systolic BP of 90 mmHg. Rotational thromboelastometry showed no significant coagulopathy.

A CT revealed evidence of a large amount of blood clot around the gastric balloon and the proximal duodenum. Endoscopy revealed a Forrest 1a 30 mm ulcer in the duodenal bulb (D1). Bleeding continued despite adrenaline injection, cauterisation and clipping. Haemostasis was achieved through radiological embolization of the proximal gastroduodenal artery. The patient was discharged a week later without any significant morbidity.

Discussion

This is the first case report of SBT use in the Australian retrieval setting. Resource limitations such as limited blood products and lack of endoscopy led us to the insertion of an SBT in a patient in cardiac arrest from upper gastrointestinal haemorrhage. Challenges to overcome were the lack of radiology, the impact of altitude on balloon pressures and maintaining SBT traction in flight. There are a few aeromedical ramifications we employed to navigate around these challenges.

The indication for SBT placement is gastroesophageal bleeding that is not controlled by medical or endoscopic therapy. The tube has two balloons, one designed for the stomach and one for the oesophagus. There are three ports – one for each of the balloons and one to allow gastric aspiration. The patient is normally intubated prior to placement, the tube placed to a measured depth with X-ray confirmation. The gastric balloon is then inflated with air in 50 mL aliquots up to 300 mL. Gentle traction is then applied to pull back against the gastric fundus and secured to maintain traction. The oesophageal balloon is only inflated if haemorrhage continues at this point.[1,2]

Air inflation of SBT balloons allows pressure to be monitored during inflation and in flight. This provides an indirect warning of oesophageal placement of gastric balloon, limiting the risk of perforation, especially upon ascent when the balloon will enlarge as atmospheric pressure falls. Upon descent, the balloon may decrease in size, causing loss of tamponade and migration. If re-inflated, injury may result if the gastric balloon has migrated to the oesophagus. Water being incompressible, mitigates the effect of atmospheric pressure but does not allow pressure to be monitored during insertion or flight.[3] In our case, we used air to inflate the balloons because no chest X-ray was available to confirm whether the gastric balloon was in the stomach prior to inflation. We used pressure monitoring as a surrogate marker of stomach placement. About 50 mL increment of air was injected into the gastric balloon and the pressure measured at each volume, up to a total of 300 mL. Each post inflation balloon pressure was <15 mmHg, thus ensuring that we avoided oesophageal inflation of the gastric balloon. The gastric lumen was suctioned, and pH tested to further verify stomach placement prior to balloon inflation. Despite inflation of the gastric balloon and traction until firm resistance was felt, there was ongoing haematemesis. We therefore inflated the oesophageal balloon to a pressure of 40 mmHg.

No data exists for a safe flight height for a patient with SBT in situ. EMS helicopters are unpressurised and therefore have an operating altitude limitation in Australia of 10 000 ft;[4] the maximum expected pressure change between the scene and the cruise height for a retrieval is approximately 234 mmHg (313 HPA).[5] In our case, with additional considerations relating to terrain clearance, fuel and weather planning, the pilot was able to fly the helicopter at low altitude to minimise pressure changes. The rate of pressure decrease with increasing altitude is not linear and the expected average pressure change for our journey was about 4000 ft (approximately 100 mmHg)[6] which is significant for SBT balloon overinflation. The oesophageal balloon, which was initially at 35 mmHg, was deflated prior to take off because overinflation of the oesophageal balloon as pressure changes could cause oesophageal rupture. The pressure of the gastric balloon was regularly monitored in flight and no further hematemesis was encountered. Fixed-wing retrievals can provide a pressurised cabin that provides a safer milieu by limiting the risk of over-inflation of the balloons.[6]

Maintaining traction on SBT is challenging in a rotary-wing aircraft. Many techniques are described in the literature, suggesting a lack of standardised method.[7] Attaching objects as traction devices, such as 1 L bag of saline, has aeromedical implications; gravitational forces and turbulence have significant effects on unsecure objects and as such, traction devices can become projectiles during flight. We maintained traction by using a vacuum mattress to mould the patient's head and cervical spine into a fixed position, then used Hypafix tape to secure the SBT to both the patient's head and vacuum mattress. Whereas transferring the patient from our vacuum mattress onto the receiving hospital bed, traction was lost; this is a disadvantage of our method. Other techniques which do not involve unsecured objects include securing the SBT to the rim of a helmet and the use of wooden tongue depressors with padding.[7]

Although rarely used in Australia, SBT has an established role as salvage treatment in uncontrolled variceal haemorrhage. Isolated case reports exist of balloon tamponade being used in the management of Mallory-Weiss tears, surgical repair of aorto-oesophageal fistulae and non-variceal distal oesophageal bleed.[8] Because of the position of the gastric balloon against the cardia and its maximum inflation pressures of 45 mmHg, SBT is generally not useful in controlling non-variceal gastro-duodenal haemorrhage and is not recommended in that setting.[8] Our decision to use SBT as a salvage therapy was based on several factors: cardiac arrest resulting from ongoing massive hematemesis, limited availability of blood products, no endoscopic service available onsite, long flight time and uncertain origin of bleeding.

On CT, the SBT did not have a direct tamponade effect on the D1 arterial bleed but did show a large amount of gastric blood clot around the balloon. It is unclear if the gastric balloon

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may have contributed to haemostasis by promoting clot formation in the stomach and subsequently the duodenum. However, bleeding appeared to have slowed after insertion of the SBT as evidenced by improved haemodynamic stability. Transfusion had been titrated to a systolic BP of 80–90 mmHg. BP on arrival of the retrieval team was 78 mmHg and until patient's arrival at the destination hospital 5 h later, it did not exceed 90 mmHg. No procoagulant blood products or drugs were given apart from 1 g of tranexamic acid, because of lack of availability. ROTEM on arrival at the tertiary hospital showed no correctable coagulopathy. Therefore, we suggest that SBT placement was a contributing factor to the survival of our patient. Indirect mechanical compression allowing clot formation in non-variceal upper gastrointestinal bleed is described as a therapeutic physiological mechanism in the literature.[8] Whereas we were successful in achieving ROSC with SBT insertion, it should not necessarily be used as a therapy for non-variceal bleeding.

Conclusion

In this case of haemorrhagic shock resulting in hypovolaemic cardiac arrest, placement of an SBT appeared to contribute to haemostasis and ability to gain ROSC. Without control of haemorrhage, PRBC would have been depleted prior to arriving at the tertiary hospital. This article highlights the aeromedical implications of SBT and how the retrieval team managed these.

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Competing interests

None declared.

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