

Safe and efficacious placement of Sengstaken-Blakemore tubes

Notwithstanding the authors' eloquent description¹ of a Sengstaken-Blakemore-tube-related perforation, which was so successfully treated with a covered, removable metal stent, and the accompanying editorial comment describing the tubes as the "least preferred method of treatment," these tubes do have an ongoing role in the management of gastroesophageal varices. In our institution, we would still place about one each year or two, in cases where there is significant doubt about whether the endoscopic techniques have arrested the bleeding. In Australia, we do not subject the patients to wearing a football helmet as outlined by Dr. Brandt—our brand of football does not require such extreme measures to protect the cerebral tissue of the players. A cord is simply tied to a flange of the tube and then slung over a pulley with a liter bag of fluid attached to the other end of the cord.

To avoid the complication described, I have taken to placing these tubes under direct vision. With the patient anesthetized and intubated in the operating theater, a snare placed through the endoscope is used to grasp the end of the tube before the endoscope (with the tube now attached) is placed through the mouth and thence into the esophagus and stomach. I then release the tube by opening the snare (drag and drop technique) and, under direct vision, blow up the gastric balloon and pull it back, such that its superior edge is snug up against the cardia. This reduces the risk of inadvertently placing the tube through an esophageal diverticulum (as may have happened in the reported case) and also ensures that the balloon is correctly positioned. It also guards against the gastric balloon being blown up in a hiatus hernia, which can result in perforation.²

Andrew Thomson, FRACP
Gastroenterology and Hepatology Unit
The Canberra Hospital
The Australian National University
Canberra, Australia

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Response:

We thank Dr Thomson for his interest in our article and his comments regarding the safe insertion of Sengstaken-Blakemore tubes (SBT). We agree that balloon tamponade is a highly effective method of controlling variceal bleeding, and, indeed, in our institution, endoscopically assisted insertion is the preferred method.

However, in the United Kingdom, a recent audit of GI bleeding revealed that only 56% of hospitals have a formal on-call endoscopy rota.¹ This situation means that out-of-hours clinicians may not have access to endoscopy to assist in SBT insertion, and, because of the relative infrequency of variceal bleeding, tubes may be inserted by inexperienced staff, which increases the potential for complications. Since the publication of our article, we treated another patient referred to our center with an esophageal perforation from an SBT insertion. This patient was also treated with a DANIS stent insertion (ELLA-CS, Hradec-Kralove, Czech Republic) and a subsequent transjugular intrahepatic portosystemic shunting procedure, again with a successful outcome.

The DANIS stent was originally developed to treat bleeding esophageal varices and may have a role in the emergency treatment of variceal bleeding.² This stent can be inserted over an endoscopically placed guidewire or without endoscopy, because its design prevents incorrect placement. We reported our initial experience with DANIS stent insertion for variceal bleeding in 8 patients, including 2 patients in whom balloon tamponade had failed, and we believe that such stent insertion may be a viable and safe alternative to SBT insertion.³

As in Australia, footballers in the United Kingdom have no need for protective headgear; however, the cricket helmet (originally developed to protect British batsmen from Australian fast bowlers) may be a suitable Anglo-Australian solution to SBT fixation.

Wolf-Rudiger Matull, MD, MRCP
James O'Beirne, MD, MRCP
Department of Hepatology
Royal Free Hampstead NHS Trust
London, United Kingdom

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“White opaque substance” and “light blue crest” within gastric flat tumors or intestinal metaplasia: same or different signs?

To the Editor:

We read with interest the article by Yao et al¹ about a white opaque substance (WOS), a fine white reticular, maze-like, speckled pattern of the epithelial surface, within gastric flat tumors. With the use of magnifying endoscopy with narrow-band imaging (ME-NBI), the WOS was more frequently visualized in adenomas (78%) than in carcinomas (43%) and showed a regular distribution within adenomas (100%) but an irregular distribution within carcinomas (83%). Although they concluded that the morphologic analysis of the WOS could be a new useful optical sign, another similar optical sign, a light blue crest (LBC), had previously been described.²

Although endoscopic identification of gastric intestinal metaplasia has a high rate of interobserver variability and correlates poorly with the histologic findings, Uedo et al² described the LBC, a fine blue-white line on the crests of the epithelial surface, with the use of ME-NBI as a distinctive accurate sign for diagnosing gastric intestinal metaplasia with a high sensitivity (89%), specificity (93%), and accuracy (91%). Tahara et al³ also disclosed that the presence of the LBC, especially in the gastric corpus, was significantly associated with gastric atrophy and a high gastric cancer occurrence, to which Ueda agreed.⁴

Gastric intestinal metaplasia has the LBC sign² and is associated with a risk for the occurrence of gastric adenomas and carcinomas^{2,3,5,6}; these tumors have the WOS sign.¹ Although the WOS was speculated to be some intracellular component¹ and the LBC to be caused by dense reflection of short-wave-length light,^{2,4} we suspect that these signs are the same endoscopic findings based on color figures of the signs.^{1,2} We, therefore, would like to know whether gastric intestinal metaplasia has a WOS or not, whether gastric flat tumors have an LBC or not, and whether these characteristic signs are subsequently the same or different.

Mitsunobu Matsushita, MD
Shigeo Mori, MD
Kazushige Uchida, MD
Akiyoshi Nishio, MD
Kazuichi Okazaki, MD

*Third Department of Internal Medicine
 Kansai Medical University
 Osaka, Japan*

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Response:

We thank Matsushita et al for their interest in our article and for their germane questions. With regard to the difference between white opaque substance (WOS)^{1,2} and light blue crest (LBC),³ both of which are visualized by magnifying endoscopy (ME) with narrow-band imaging (NBI), we believe that they are different phenomena *and* different substances, as explained below.

From an optical point of view, we suggest that WOS and LBC are visualized because of different phenomena. LBC cannot be detected with white light imaging (WLI) alone; it can *only* be visualized with NBI, using light with short wavelengths of 400 to 430 nm.³ In contrast, WOS can be visualized with WLI alone and with NBI alone.¹ Furthermore, Uedo et al³ suggest that LBC is a phenomenon caused by the reflectance of light on the ciliated surface of the epithelium, whereas we speculate that WOS is a phenomenon caused by strong scattering of both WLI light and NBI light within the actual epithelium itself.¹ In reality, LBC can never obscure subepithelial microvessels, even when CD10 is strongly positive on the surface of the epithelium,³ whereas WOS always obscures them.^{1,2} Optically, reflectance and scattering are different phenomena.^{4,5}

From an anatomical point of view, the localization of WOS and LBC is quite different when visualized by ME with NBI; namely, LBC is always discerned on the surface of the epithelium, just at the edge of crypts, whereas WOS is invariably detected within the epithelium of the intervening part between the crypts.

We reviewed ME with NBI findings for 40 consecutive cases of early gastric epithelial neoplasia (Table 1). LBC was present within the neoplasia in 5 of them (12.5%) (Fig. 1) and WOS was evident in the background mucosa in 8 of them (20%), these 8 being previously confirmed