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Endoscopic gastrostomy using a Foley catheter

To the Editor:

Percutaneous endoscopic gastrostomy (PEG) was introduced in 1981.[1] This technique does not require general anesthesia and is now an established method for performing gastrostomy. Although we performed 35 endoscopic gastrostomies in the last year without any complications, we have observed two major limitations of this technique. First, the size of mushroom catheter is limited, as a larger size mushroom catheter and bumper tend to get caught at the cricopharyngeal or gastroesophageal junction. Second, replacement of the gastrostomy tube requires a repeat endoscopy. In an attempt to address these problems we have modified the present technique for endoscopic gastrostomy.

The patient is placed on the endoscopy table in the supine position. The posterior pharynx is sprayed with topical anesthesia, and intravenous sedation is administered. The abdomen is prepared in a sterile fashion, and sterile drapes are applied. After panendoscopy a 75-cm silk suture is introduced into the stomach and brought out of the patient's mouth. A standard #24 Foley catheter is modified for the gastrostomy tube. The end opposite the balloon tip is cut off and a stitch is placed through this end. The stitch is then threaded through the plastic sheath of the Medicut catheter, and the Foley catheter is put on stretch, allowing it to slip into the Medicut catheter. The Foley catheter is pulled in retrograde fashion out of the abdominal wall. After about 4 inches of the catheter tip has emerged, the balloon of the Foley catheter is inflated with 10 cc of air through the opening (see *B* in Fig. 1), and a clamp is placed to prevent deflation of the balloon. Next, 2 ml of Crazy Glue mixed with 0.5 ml of water is injected via the opening, and the catheter is pulled under direct endoscopic vision to ensure that the balloon is firmly against the gastric wall. The clamp is then released. A bumper is prepared and is positioned on the catheter as it emerges from the abdominal wall. After about 2 weeks the balloon is deflated and the Foley catheter is pulled out. A standard Foley catheter is used as a replacement.

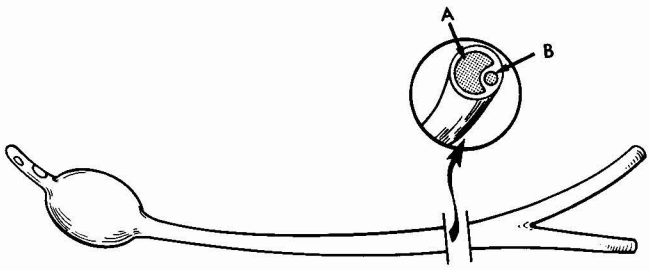

Figure 1. Foley catheter transected: *A* leads to the main lumen; *B* leads to the balloon.

Gastrostomy tube placement via the endoscopic technique has become the state of the art. This technique is safe, easy, and cost effective. This modification of PEG will not only make it easier to place larger size tubes and prevent problems of blockage but will also allow replacement of the tube without the complications and cost of repeat endoscopy.

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Chronic esophageal ulcers in an AIDS patient

To the Editor:

The acquired immunodeficiency syndrome (AIDS) is a disorder caused by the human T-cell lymphotropic virus type III/lymphadenopathy-associated virus (HTLV-III/LAV).[1] Specific gastrointestinal disorders found in AIDS patients have included diarrhea from *Entamoeba histolytica*, *Giardia lamblia*, and *Shigella*, *Salmonella*, and *Campylobacter* species, esophageal mucosal ulcerations from *Candida albicans* and *Herpes simplex*, and positive throat cultures for cytomegalovirus and Epstein-Barr virus.[2, 3] We report the case of an AIDS patient with idiopathic chronic esophageal ulcers that may be of interest to other endoscopists caring for these patients.

A 47-year-old homosexual Filipino male was in good health until he began developing fatigue, intermittent fever, mild diarrhea, odynophagia, and weight loss. He was found to have oral candidiasis, but other specific infections were not diagnosed. His odynophagia initially improved with oral nystatin, but soon he began deteriorating with a worsening of his original symptoms. He was admitted 3 months later for a complete evaluation.

Upon admission the patient appeared to be a poorly nourished, chronically ill male. Physical examination was significant for diffuse lymphadenopathy and mild epigastric tenderness. Laboratory tests revealed a hemoglobin of 8.7 with normal indices, a white blood cell count of 6.9 with 8% lymphocytes, and mild elevations of liver enzymes. T-lymphocyte studies revealed a helper to suppressor ratio of 0.08. (HTLV-III/LAV serology was not available at this time.) Multiple cultures of blood and stool showed no growth. CT scans of chest and abdomen were normal. Cytomegalovirus was isolated from urine and lung. Studies for toxoplasmosis and Epstein-Barr virus were negative.

An upper endoscopy was done to evaluate the patient's odynophagia. This revealed two discreet midesophageal ulcers measuring 1 and 3 cm, respectively, with little intervening inflammation. There was no evidence of Candida infection, and the distal esophagus appeared normal. Biopsies

from the edges of the ulcers revealed both acute and chronic inflammation with no evidence of viral inclusions, neoplasia, or columnar epithelium. Brushings were negative for cytology, culture, fungal studies, and viral isolation.

The patient was treated with ranitidine, sucralfate, and antacids with good symptomatic control of his odynophagia. A repeat endoscopy was done 1 month later and revealed no change in the endoscopic appearances of the esophageal ulcers. Repeat biopsies and brushings were negative except for inflammation. The patient was discharged home on ranitidine, sucralfate, and antacids.

A diagnosis of Kaposi's sarcoma was made from a skin lesion 3 months later. A final endoscopy revealed that the esophageal ulcers were unchanged despite reasonable symptomatic control of the odynophagia.

Esophageal ulcers have been found in a variety of clinical settings including gastroesophageal reflux, viral infections, and pill-induced damage, especially by tetracycline.[4-6] Although one case of idiopathic esophageal ulcers has been reported in a patient with X-linked immunodeficiency and neutropenia,[7] idiopathic esophageal ulcers have not been previously reported in an AIDS patient.

The mechanism for our patient's esophageal ulcers is unclear. Although our patient had cytomegalovirus infection, no viral inclusions could be seen on multiple biopsies from different endoscopies. Other potential causes of esophageal ulcers including infection, gastroesophageal reflux, neoplasm, and columnar epithelium seem unlikely based on the endoscopic and microscopic findings. It may be postulated that the ulcers were of viral etiology without inclusion bodies. Future cases may benefit from electron microscopy for identification of specific viruses in biopsy specimens.

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Nifedipine treatment of bolus esophageal obstruction

To the Editor:

The esophagus is the most common site in the gastrointestinal tract for food and foreign body impaction.[1] Once an object is impacted in the esophagus, spontaneous passage is unlikely, and some type of intervention is usually required.[1] Food impactions have been treated in a variety of ways including enzymatic dissolution, muscle relaxants, and endoscopic extraction.[2-4]

Nifedipine, a calcium channel blocking agent (Procardia, Pfizer), has been used extensively in the treatment of ischemic heart disease[5] because of its ability to relax coronary artery smooth muscle.[6] This ability to relax smooth muscle is not restricted to the coronary arteries but extends to smooth muscle in other organs including the body of the esophagus.[7] Thus, nifedipine has been used experimentally in the treatment of achalasia and "vigorous" achalasia.[8,9] Your readers may be interested in this report of the use of sublingual nifedipine in the successful treatment of esophageal food impaction.

A 57-year-old man who was previously in good health was seen in the emergency room with a sensation of food lodged in his chest. While eating a roast beef dinner 4 hours before admission he felt "as if a piece of meat had caught in his throat and wouldn't pass into his stomach." Despite attempts to dislodge the food bolus with fluids and self-induced vomiting, the sensation persisted, leading the patient to seek medical advice. The patient denied a prior history of dysphagia, weight loss, or dyspepsia, and he did not wear dentures. He had no history of cardiac disease, exercise intolerance, or hypertension. Physical examination was unremarkable except for the presence of markedly increased salivation. Barium swallow disclosed a bolus impaction in the midesophagus with complete luminal obstruction. No bone was seen within the food bolus.

The patient was administered 10 mg of nifedipine sublingually by puncturing a 10-mg capsule with an 18-g needle and squirting the contents under the patient's tongue. Within a few minutes, the patient noted a sensation of retrosternal "relaxation," with relief of his symptoms. Subsequent barium swallow revealed clearance of the food bolus from the esophagus and unobstructed flow of barium. A barium meal performed electively at a later date was entirely normal. Although upper endoscopy was recommended, the patient declined further work-up.

Although endoscopic extraction has become the treatment of choice for acute bolus impaction of the esophagus, this method of treatment carries a low but finite risk of esophageal perforation or aspiration. As spontaneous bolus obstructions are often associated with an area of localized muscular spasm rather than a true stricture, use of smooth muscle relaxants would appear to offer a logical and noninvasive approach to this problem. Unfortunately, the use of glucagon and anticholinergics has yielded variable results.[2,3] Or interest in the use of nifedipine for this problem was prompted by reports that calcium-channel blockers relax esophageal smooth muscle and the recent use of nifedipine in the treatment of achalasia.[8,9] A major advantage to its use in