References

1. Baisi A, Cioffi V, Nosotti M, DeSimone MD, Rosso L, Santambrogio L. Intrapericardial left pneumonectomy after induction chemotherapy: The risk of cardiac herniation. *J Thorac Cardiovasc Surg.* 2002;123:1206-7.
2. Buniva P, Aluffi A, Rescigno G, Rademacher J, Nazari S. Cardiac herniation and torsion after partial pericardiectomy during right pneumonectomy. *Tex Heart Inst J.* 2001;28:73.
3. Nazari S. Letter. *Tex Heart Inst J.* 2002;29:72.
4. Walker WE. Hazards of chest tubes after pneumonectomy (letter). *Tex Heart Inst J.* 2002;29:72.
5. Cooley DA. Editorial commentary (letter). *Tex Heart Inst J.* 2002;29:73.
doi:10.1067/mtc.2003.268

Reply to the Editor:

Kirschner's comment focuses on two points: the use of a chest tube after pneumonectomy and the similarity of our case to one recently reported in another journal.¹

Regarding the use of a chest tube after pneumonectomy, it is worthwhile to repeat the commentaries of Nazari and Cooley cited in Kirschner's letter.^{2,3} It is a personal choice of the surgeon, and no evidence exists supporting one option or the other. However, we would like to remind Kirschner that after pneumonectomy, the chest tube is not attached to continued suction and does not "court disaster produced by excessive mediastinal shift," as he says, but is connected to an appropriately balanced pneumonectomy drain set.⁴ This is completely different from the one-way valve used after partial lung tissue resection. It is designed to achieve both positive and negative pressure control avoiding any abnormal mediastinal shift.

We reply to the second point of Kirschner's comment stressing the role played by induction chemotherapy in our case, as emphasized both in the title and in the discussion of our article. In the past 5 years we performed 208 pneumonectomies, 47 with intrapericardial vessel ligation. The pericardial defect was closed directly by stitches in 35 cases and by a pericardial patch in 12 cases in which the defect was too large and a direct suture would have been under tension. The reported case of cardiac herniation was the first in our experience. We are sure that the closure we performed in the first operation was not under any abnormal tension, that the pericardium was fibrotic because of the previ-

ous chemotherapy, and that this played a major role in its disruption.

The message we would like to give is not that cardiac herniation may happen after intrapericardial pneumonectomy—this is widely known—but that it is more likely if the patient has undergone previous induction chemotherapy. In these cases, the use of pericardium to close small defects can also be recommended.

Alessandro Baisi, MD

Ugo Cioffi, MD

Luigi Santambrogio, MD

University of Milan

Department of Surgery Ospedale

Maggiore Policlinico IRCCS

Milan, Italy

References

1. Buniva P, Aluffi A, Rescigno G, Rademacher J, Nazari S. Cardiac herniation and torsion after partial pericardiectomy during right pneumonectomy. *Tex Heart Inst J.* 2001;28:73.
2. Nazari S. Letter. *Tex Heart Inst J.* 2002;29:72.
3. Cooley DA. Editorial commentary (letter). *Tex Heart Inst J.* 2002;29:73.
4. Waters PF. Pneumonectomy. In: Pearson FG, Deslauriers J, Ginsberg RJ, Hiebert CA, McKneally MF, Urschel HC, editors. *Thoracic surgery*. New York: Churchill Livingstone; 1995. p. 844-8.
doi:10.1067/mtc.2003.269

Intussusception and spontaneous amputation of the esophagus

To the Editor:

I read with interest the case of a patient with acute leukemia and segmental esophageal necrosis reported by Salo and associates.¹ The authors imply that intussusception led to ischemia, and circumferential full-thickness loss of the esophagus was repaired "without interruption." Both assumptions may be challenged. The patient had no risk factor for gastroesophageal intussusception,² in which stomach is the intussusceptum, not esophagus, and perforation, rather than necrosis, a reported complication. Fungal infection of the esophagus in leukemia, however, is known to result in full-thickness necrosis and esophageal expulsion.³ Their final comment relating to *Aspergillus* infection of the esophageal wall points to the likely cause of necrosis.

A complete loss of all esophageal layers, no matter how short, is expected to result in cicatricial stricture that may have

been delayed in this case by cytotoxic therapy. Indeed, dysphagia later developed, necessitating further intervention. The mediastinal soft tissues may resemble the esophageal lumen so that perforation is not obvious. However, bare of epithelium and exposed to esophageal contents, the mediastinum provides an entry to systemic infection, particularly in an immunosuppressed state. Esophageal resection with immediate or delayed reconstruction should be considered even in this patient population to prevent mediastinitis and restore swallowing.

Henning A. Gaissert, MD

Massachusetts General Hospital

Boston, MA 02114

References

1. Salo JA, Ristimäki A, Salminen JT, Siren J. Intussusception and spontaneous amputation of the esophagus. *J Thorac Cardiovasc Surg.* 2002;124:205-6.
2. Gowen GF, Stoldt HS, Rosato FE. Five risk factors identify patients with gastroesophageal intussusception. *Arch Surg.* 1999;134:1394-7.
3. Abildgaard N, Haugaard L, Bendix K. Non-fatal total expulsion of the distal oesophagus due to invasive candida oesophagitis. *Scand J Infect Dis.* 1993;25:153-6.
doi:10.1067/mtc.2003.261

Reply to the Editor

We thank Gaissert for his very valuable comments.

Spontaneous amputation of the esophagus is a serious and rare event, and its exact pathophysiology is not well understood. There were several reasons that led us to believe that intussusception, followed by ischemic necrosis, may be an etiologic factor. Our patient had a hiatal hernia, as previously stated. Hiatal hernia is a widely known risk factor for intussusception, as well as dysphagia, a condition that developed after severe vomiting.¹ On careful inspection of Figure 1, one can see that the inner circular muscle layer of the esophagus is located superficially as a result of intussusception.

Abildgaard and associates² have described total expulsion of the distal esophagus in a severely immunocompromised patient with *Candida* esophagitis. Multifactorial pathogenesis, including intussusception, is presumable inasmuch as their patient had also had regurgitation, vomit-